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LOCAL DISEASES OF THE MOUTH.

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LOCAL DISEASES OF THE MOUTH.

DISEASES OF THE TONGUE.

We have already discussed in the first part of this article* a number of diseases which involve the tongue, such as the diffuse acute glossitis of various forms of stomatitis (stomatitis mercurialis), the sclerosing glossitis of tertiary syphilis, gummata of the tongue, leucoplakia lingualis, tuberculosis and actinomycosis of the tongue, etc.

In the present section we shall study the diseases which are confined to the tongue. Some of these affect the surface of the tongue only, others involve the entire organ.

Glossitis Superficialis Simplex.

A series of acute or chronic inflammatory diseases, as well as some peculiar processes which are analogous to certain dermatoses and as to the connection of which with inflammatory disease there is still uncertainty, are met with on various parts of the tongue.

Superficial catarrh of the lingual mucous membrane occurs very frequently in all kinds of febrile diseases and in disturbances of the alimentary canal in the form of the "coated tongue." This familiar symptom of disease, which is well known even to the laity, was briefly noticed in connection with those affections of the mouth which are associated with general diseases and needs here no detailed description. This condition is often due not so much to an increased desquamation of the superficial horny epithelial layers of the surface of the tongue as to the insufficient removal of these cast-off masses, the regular automatic cleansing of the tongue involved in the processes of mastication and deglutition having ceased. While on the one hand there may be a very transient coating of the tongue, as in acute gastritis and in infective diseases of brief duration, on the other hand such coatings may be very obstinate, persisting even for years.

* Unavoidable delay in the preparation of the manuscript necessitated a division of the article on Diseases of the Mouth. The first portion, treating of the buccal manifestations of the systemic diseases, will be found in Vol. VIII.—Ed.

This depends essentially upon the length of the time during which the ingestion of food is diminished.

A frequent phenomenon in acute as well as in chronic superficial glossitis is a swelling or hypertrophy of the lingual papillæ, which causes, for example, the so-called "strawberry tongue" of scarlet fever (swelling and reddening of the papillæ fungiformes), also the peculiar horny prolongations of the papillæ filiformes in the different forms of the "hairy tongue" or "cat's tongue." These deserve a separate section, but first certain diseases which are, it would seem, peculiar to the tongue and which are analogous to the dermatoses, must be described.

Geographical Tongue.

(Lingua geographica; Annulus migrans.)

Peculiar eruptions occur upon the tongue, especially of youthful individuals, characterized by their transitoriness and their changeable form. These have as yet been observed in only a few cases. While Parrot[1] and Kaposi[2] suppose that the "migratory exfoliations" of the tongue which characterize this affection are of syphilitic origin, Caspary, Unna, and indeed almost all other writers who have treated of this disease, positively deny any connection of these "transitory benign plaques of the lingual mucous membrane" (Caspary) with syphilis. We share the opinion of the latter, but admit that exceptionally similar desquamations with curving edges ("desquamations à contours festonnés" of Gautier[3]) occur in the secondary stage of syphilis. But such syphilides of the mucous membrane persist much more obstinately upon the affected area than the non-specific eruptions.

The statement of Butlin that the lingua geographica is hardly ever observed in adults or even in children more than six or seven years of age is erroneous, as appears, among others, from the history of a case depicted in Mikulicz' Atlas, Plate XXIX., Fig. 1.

Unna has called attention to the fact, the correctness of which we can confirm from our own observations, that in many nursing infants recurring circular exfoliations of the mucous membrane of the tongue occur as the result of dentition. But the second dentition—in this connection reference may again be made to the history of the case mentioned above—also sometimes causes the lingua geographica. We also occasionally see cases in which the affection is directly connected with a preceding stomatitis aphthosa, so that it appears as if a general stomatitis caused by other conditions and the consequent greater vulnerability of the mucous membrane of the tongue predispose to the development of such migratory patches. The course

of the affection speaks in favor of a parasitic origin, but nothing further is known with respect to the etiology.

The appearance and the course of the disease are very characteristic. Bright red plaques appear which vary in form and size but are generally round. They are slightly elevated and within their limits the papillæ are considerably swollen. The red spots are often sharply circumscribed by a gray marginal zone which consists of crowded dots. The marginal zone may even form a double contour about the whole or a part of the central plaque. The gray dots correspond to the papillæ filiformes, which are broader than the normal and the epithelium of which appears thickened. This change of the papillæ is to be detected also upon the remaining mucous membrane which is free from the plaques already described and may give it the appearance of shagreen. The papillæ fungiformes are also thickened and enlarged, but are more conspicuous on account of their vivid redness.

Extraordinary variability is characteristic of these plaques. In a few days they may greatly change their form and size; they may become confluent; they may disappear entirely and develop elsewhere. The forms change, to use Caspary's comparison, like the figures in a kaleidoscope. The discomfort resulting from this affection is trifling. The patients are conscious only of the frequently accompanying diffuse stomatitis, but this rarely is marked.

The disease could hardly be confounded with any other except possibly with Möller's glossitis and with mucous patches. From the first the diagnosis is made by the marked painfulness and the extremely chronic course of Möller's glossitis, and from the second by the long duration of condylomata without material change in their appearance or extent.

Treatment is unnecessary. The affection disappears in a short time without any medication.

Glossitis Papulosa Acuta.

Of this disease, so far as is known, only one case, that of Michelson[4] (depicted in Mikulicz' Atlas, Plate XXX., Fig. 1) has been published. A quite analogous case, however, was observed at almost the same time in the surgical clinic at Königsberg.

In both cases the patients were laborers' wives, one thirty-four, the other thirty-two years of age, who had had no previous disease of any importance. The affection began in one case with fever and chills, depression, loss of appetite, headache, and at the same time burning pains in the tongue, upon which there appeared several whitish spots

about the size of a pea. In the other case there is no account of these febrile symptoms. The disease developed without pain and only one such whitish ulcer appeared, but this attained a more considerable size (that of a five-cent [fünfpfennig] piece). All signs of syphilis were absent in both cases. The affection was cured under rather indifferent treatment, erosions ½ to 1½ mm. deep, with reddened, somewhat jagged edges and with a floor covered with greenish pus, developing from the white papules, and subsequently cicatrizing. The whole disease lasted about three weeks. It was accompanied by a well-marked but otherwise not especially characteristic diffuse catarrhal stomatitis. The etiology of the process is quite obscure. In the fragment which was examined microscopically were found superficial gaps in the epithelium, which were filled with fibrinous masses. The epithelium was lifted up by these. Under it was a not well-developed zone of infiltration with small cells, which presented no peculiarities. The papillæ were enlarged, especially thickened. The superficial layers of the epithelium and the gaps in it were infiltrated with numerous bacteria, but specific pathogenic organisms were not found. It is quite possible that the disease is connected with foot-and-mouth disease, but further investigations are necessary in order to determine this.

Chronic Superficial Glossitis.

(Glossodynia exfoliativa; Möller's Glossitis.)

This peculiar disease of the surface of the tongue, described by Möller in 1851 and rescued from oblivion by Michelson[4] in 1890, occurs without known cause, as a rule, in hypochondriacs, especially, it would seem, in women.

Accompanied by severe burning pains in the tongue, which may increase during the mastication of solid food or after protracted speaking until they become unendurable, there appear upon the dorsum of the tongue generally irregularly scattered bright-red spots and streaks, which are contrasted with the surrounding absolutely normal surface, sometimes but slightly, sometimes strikingly, by their color. The spots are caused by the thinning, in places even the entire absence, of the epithelium. The superficial layers of the tunica propria are infiltrated with small cells. In one case small whitish nodules were observed in the midst of the reddened tissues, which consisted of crowded masses of round cells. Over these the epithelium was completely absent. Within the reddened portions the papillæ are often strongly hypertrophied. These foci are localized chiefly upon the dorsum and the tip, sometimes upon the lateral edges of the tongue,

very rarely upon isolated portions of the buccal and labial mucous membrane. The remaining portions of the tongue present an absolutely normal appearance.

The subjective symptoms do not at all correspond to the trifling anatomical changes. Severe burning pains in the tongue, especially during mastication of solid or highly seasoned food, may so annoy the patients that eating becomes torture and is limited to a minimum. Since the patients are generally poorly nourished and anæmic and the disease is of long duration, this fact is of no small importance.

The affection has generally lasted for months before the patient consults the physician, so that hardly anything is known about its beginnings. Its course is exceedingly chronic. Exacerbations generally occur from time to time; in the intervals between these the condition of the patient may be endurable for weeks or months, although the sufferers never recover entirely. The disease may be protracted for years without leading, so far as is known, to any more serious affection.

The treatment has as yet proved of little efficacy in any of the cases. Applications of silver nitrate, lactic acid, and the like have indeed produced a limitation of the inflammatory process and a temporary mitigation of the pains, but in all the reported cases the latter soon returned and the anatomical lesions remained unchanged.

Hairy Tongue.

(Lingua nigra.)

On that portion of the dorsum of the tongue which is situated immediately in front of the circumvallate papillæ there is sometimes seen a diseased condition which presents a most singular appearance. Of this the number of recorded cases is not very large, but they have been reported with increasing frequency of late years. The first observations date from the middle of the century (Rayer, Eulenburg), but it is only within the last ten years that the literature of the subject has become abundant. Compilations of it are to be found especially in the works of Brosin;[6] a series of the more recent cases has also been collected by Sendziak.[7]

The disease consists in a peculiar coloration of the region designated, the intensity and also the color of which vary greatly. Cases have been reported of a yellow and brown (Dinkler), a black (the majority of writers), and recently even a green (Mourek) coloration. This colored surface forms a patch of an irregularly oval, elliptical, or triangular shape. The surperficies of this part of the tongue is very rarely smooth; generally there is a marked swelling, especially

a prolongation, of the filiform papillæ. This swelling may attain such a degree that the papillæ resemble well-developed, somewhat bristly hairs. A patient of Sendziak was even accustomed to shave them off by means of an instrument constructed by himself for that purpose. They, however, promptly grew again.* These hair-like and peculiarly colored papillæ give the dorsum of the tongue an extraordinarily strange appearance, so that the patients themselves generally notice it.

The affection may pursue an acute or a chronic course, sometimes lasting only a few days; in other cases it may persist even for years unchanged or with greater or less variations in intensity.

Anatomically these hair-like formations are the result, as is generally admitted, of a prolongation and thickening of the papillæ filiformes; their epithelium is especially thickened in thorn-like prolongations.

The disputed point is the cause of the peculiar coloring. It was ascribed by some even of the less recent authors (see Brosin's references) to an accumulation of bacteria or yeast-like organisms. But a really positive demonstration of these was first made by Ciaglinski and Hewelke.* They found a mould assigned by them to the family Mucor, the conidia of which were studded with black spores. Sendziak discovered quite similar growths. Both writers cultivated the fungi. Analogous observations have not been made by others.

Mourek, the most recent investigator of the subject as far as we know, found on the prolongations of horny epithelium which lengthen the papillæ filiformes, a peculiar coloration due to their horny nature. This was noticed also by earlier authors. On the other hand characteristic fungi were entirely absent in his case, although he sought carefully for them.

Earlier authors were inclined to ascribe the coloring to thei ngesta or even to an intentional dyeing. The latter may occur, though certainly not in all of a series of cases, but, on the other hand, staining from the ingesta cannot be so easily dismissed. Microscopical technique has shown how firmly certain dyes (picric acid) are held by horny, flat epithelium. It is therefore quite possible that in these pathological states certain stains play a part.

But that question is still unsettled. It does not, however, seem very probable that the conidia of the Mucor niger, which are distinctly visible macroscopically as dots, could cause such a uniform and diffused coloring of the papillæ. At all events we know, with respect to the analogous mycoses of the external auditory meatus, either that here thick masses of fungus are formed in which the individual

* Amatus Lusitanus in 1557 found hairs upon the tongue of a man, "hairs which when pulled out grew again" (Arnold: Virchow's Archiv, vol. cxi., 1888).

black dots are not to be differentiated, or that on close inspection the conidia can be distinctly recognized as colored dots.

Further investigations are certainly necessary, and it is to be regretted that Sendziak has not reported the findings of the autopsy of his second case in greater detail.

The clinical symptoms of the hairy tongue are not characteristic. The patients complain often of gastric disturbances and of various conditions which result in a diminution of the amount of food taken. We know that when food is sparingly ingested, morbid conditions of the surface of the tongue are frequent because its automatic cleansing is deficient. A peculiar "pasty" feeling and a certain insipid taste appear to be the only symptoms caused directly by the disease of the tongue.

The duration of the affection is quite various, as has already been mentioned. When it appears worth the while to treat the disease, a thorough scraping of the diseased portions of the tongue, followed by the application of strong antiseptics, and a careful toilet of the mouth are indicated.

Phlegmonous Processes in the Tongue.

(Abscess of the tongue; Glossitis diffusa acuta.)

The surface as well as the substance of the tongue has, as is well known, an enormous power of resistance to pathogenic bacteria. Even in pre-antiseptic days its wounds healed almost without exception by first intention. Notwithstanding, phlegmonous processes of the tongue are not extremely rare. They manifest themselves in the form of superficial or deep abscess or of a diffuse inflammation of the substance of the tongue.

Superficial abscesses of the tongue occur, as a rule, upon the dorsum, generally near the base. They are formed in a way analogous to a certain extent to the furuncles of the skin. As in the latter the hair follicles and the sebaceous glands are points of entrance for micro-organisms, so upon the tongue the mucous glands present depressions of the surface of the mucous membrane which afford a favorable breeding and dwelling place for the pyogenic bacteria, which are undoubtedly taken in with the food in large numbers. Perhaps small foreign bodies which gain an entrance during eating are the cause of the infection. As a rule, however, the history gives no information as to the mode of infection.

The inflammatory process is rarely so violent in these abscesses as is the case in furuncles. In most of the histories of cases it is reported that the affection began gradually. From the beginning

there are moderate, rarely severe pains and at the same time the rather rapid and considerable increase in the volume of the tongue greatly impedes speaking and swallowing. The abscesses may reach a considerable size before the patients seek medical advice. They then generally extend widely immediately beneath the surface of the mucous membrane, are tense, and fluctuate distinctly upon palpation; here and there the yellow color of their purulent contents is visible through the overlying tissues.

Spontaneous rupture appears to be long delayed. As a rule it is necessary to open the abscesses—an operation which presents no difficulties. Healing then takes place uneventfully, sometimes with surprising rapidity.

The diagnosis is generally not difficult. The painfulness of the affection prevents confusion with cysts of the tongue. The superficial abscess is as a rule distinguished from a softened gumma (which is generally situated more deeply within the substance of the tongue) by its painfulness, its relatively rapid development, and by the distinct fluctuation.

Deep phlegmons of the tongue (glossitis diffusa acuta) are very rare, but are sometimes caused by severe stomatitis (especially mercurial stomatitis) or by the penetration of foreign bodies (fish bones, husks, and the like). They may also occur as a complication of various infectious diseases, especially typhoid fever, erysipelas, and small-pox. Foot-and-mouth disease has already been mentioned as a cause. Diffuse glossitis also occurs in anthrax. Stings of wasps and bees and other poisoned wounds may sometimes cause enormous phlegmon-like swellings of the tongue. We may also here refer to the exacerbations of glossitis diffusa, to be described farther on, which occur in the course of diffuse lymphangioma of the tongue.

The chief symptoms of the disease are a considerable swelling of the organ, which often very quickly becomes too large to be contained within the mouth, and extremely severe pain. The surface of the mucous membrane is of a violet or bluish-red color. It often becomes quite transparent from œdema, like dropsical skin. Of course speech and deglutition are greatly impeded. There is a tendency to the development of inflammatory œdema of the ostium laryngis, of the pharyngeal wall, and of the arches of the palate, and this œdema may become directly dangerous to life.

Besides this danger there is that of a general infection, as in all phlegmonous processes which are near the centre of the circulation. Finally, the prognosis is rendered worse by the presence of the original disease, especially if it is anthrax or foot-and-mouth disease.

If the disease does not lead directly to death there may be either

absorption of the exudation or the formation of an abscess. The former termination is not rare. It is constant in infection from foot-and-mouth disease. The abscess forms generally deep within the organ, and hence the presence of pus is often not easy to determine, the detection of fluctuation being frequently especially difficult. It is therefore quite possible that the abscesses may be confounded with gummata, especially if the history gives no positive evidence of the mode of origin. As a rule the fact that pain has been present during at least a part of the course of the disease will prevent this mistake, yet often enough the diagnosis may remain doubtful. In this case the administration of potassium iodide is to be recommended.

According to the statements of most authors (König, Albert, and others), abscesses may slowly develop deep within the tongue without fever or well-marked inflammatory manifestations—chronic lingual abscesses. Their cause is unknown. They are certainly very rare. The differential diagnosis between these and gummata must present insuperable difficulties.

The treatment of the deep phlegmons of the tongue must be energetic, unless anthrax is established as the cause, so that recovery appears impossible. Painting with iodine, parenchymatous injections of carbolic acid, and the like, which are still advised in the text-books, promise little benefit and may become dangerous. If the use of ice and of cooling antiseptic mouth washes is not sufficient, the most benefit is to be expected from not too superficial scarifications in the long axis of the tongue. The hemorrhage from these is often considerable, but soon ceases. Iodoform should then be rubbed into the wounds and iodoform gauze applied. Sometimes tracheotomy may be necessary. Deep glossitis is often confined entirely or for the main part to one-half of the tongue (hemiglossitis).

Traumatic (Decubital) Ulcer and Induration.

Of greater diagnostic than clinical importance are circumscribed affections of the tongue which arise from injuries due to frequent and long-continued friction of the organ against a diseased tooth.

The teeth are generally carious molars with sharp or pointed edges, which pierce the tongue. These injuries being constantly repeated for long periods, the affected portion of the lingual mucous membrane is first deprived of its epithelium, then a chronic inflammatory induration is developed in the subjacent tissues. A nodule is formed which at first is small and quite superficial but which gradually penetrates more deeply into the tissues of the tongue. This is painful during deglutition and especially during mastication. The

attention of the patients is as a rule attracted by the pains and they discover the nodule by palpation with the finger. If the size of the induration increases quite rapidly, as is generally the case (the infiltration may reach the size of a hazelnut), driven by fear of cancer they generally betake themselves to the physician a few weeks after they have discovered the disease. The pains are of no great intensity and are annoying only during mastication and deglutition. Other symptoms, except the stiffness of the tongue, are rare. It is especially

FIG. 1.—Decubital Ulcer of the Tongue. (From Mikulicz' Atlas, Plate XXXII., Fig. 1.)

The case from which this illustration was taken, was that of a man sixty-three years of age. Three or four weeks previously the patient felt pain while eating. At the painful spot was a nodule of the size of the head of a pin, which grew steadily and in two weeks became raw. Especial pain from contact with the decayed tooth had only recently been observed. On the posterior part of the right edge of the tongue there is a nodule, the size of a bean, about half of the vertical diameter of which projects above the level of the tongue. On the anterior part of this nodule is a crateriform ulcer of the size of a lentil, the posterior edge of which is elevated and is of a darker red than the surrounding tissues. The anterior edge is flat and is surrounded by a soft scar. When the tongue is protruded the ulcer corresponds exactly with the sharp edge of the carious wisdom tooth. After extraction of the tooth the ulcer healed permanently without further treatment.

noteworthy that even when the infiltration is of considerable extent the lymphatic glands in the vicinity are very rarely swollen. The generally small ulcer often heals without treatment if the offending edge of the tooth becomes somewhat rounded. But the infiltration does not disappear so rapidly and new ulcerations are easily developed at other portions of it. Ulcerations and recent scars are then found present at the same time.

In other cases new infections and abscesses may occur within the infiltrated area; these seldom attain any considerable size but may greatly aggravate the annoyances from the malady.

The diagnosis of this not uncommon and otherwise rather unim-

portant disease is of the greatest moment because it is readily confounded with carcinoma. It is also probable that the latter may develop occasionally from a neglected decubital ulcer. Characteristic signs by which the two affections can be distinguished clinically can hardly be said to exist. Only one thing distinguishes them from each other with absolute sharpness: the decubital ulcer or infiltration has the strongest tendency towards recovery; the infiltration, as a rule, disappears in eight to ten days after the carious tooth has been extracted or rendered harmless. The superficial ulcerating cancroid may also heal in places, when this has been done, but never in its entire extent; the induration caused by it is scarcely changed.

It should therefore be made a rule in every case of suspicious induration upon the edge of the tongue to look for sharp edges upon the teeth. If the situation of the suspected tooth agrees with that of the ulcer when the tongue is at rest, the tooth should be extracted. After eight to ten days the diagnosis can be made easily between carcinoma and decubital ulcer.

There may on superficial examination be some confusion with syphilitic ulcers, but a careful examination of the tongue will usually make the differentiation easy.

DISEASES OF THE FLOOR OF THE MOUTH.

Except tumors, diseases of the floor of the mouth are rare. We have to consider only diseases of the sublingual gland, of its duct and of that of the submaxillary gland, and diseases of the loose connective tissue between the lower jaw and the genioglossus and mylohyoid muscles.

Pyorrhœa of Wharton's Duct; Salivary Calculus.

Small foreign bodies (husks, grains of wheat, small fruit seeds, such as of raspberries and currants) penetrate not very infrequently into the ducts of the salivary glands, especially into the duct of Wharton, which opens anteriorly on the floor of the mouth beneath the tongue. These, being generally firmly wedged in the duct and frequently, probably from the movements of the tongue, being driven farther towards the gland, excite an inflammatory irritation which leads to a purulent catarrh. The salivary gland also quickly becomes diseased when its duct is closed. These small foreign bodies may lie for a long time, often for years, in different portions of the duct without causing serious annoyance. The patient generally first becomes aware of the condition when concretions are formed about

the foreign bodies, which gradually increase in size and become salivary calculi. These consist for the most part of calcium phosphate and are chiefly found at two points, the first being in the duct of Wharton 10 to 20 mm. from the salivary caruncle. They have here, as a rule, a long oval form, more rarely a spherical shape. Their size varies between that of an oat grain and that of a date stone. Thorowgood observed the largest stone on record; it was one and one-eighth inches long. Salivary calculi are also found within the substance of the gland, having probably originated in the proximal portion of the duct. Here, as a rule, they do not occur in the form of a single large concretion, but there are found in the centre of the gland, which is much enlarged and indurated from fibrous degeneration, numerous concretions of the size of a hemp-seed to that of a pea and of the consistence of soft mortar; the stones of Wharton's duct, as a rule, are harder.

Salivary calculi do harm in two ways: First, they narrow or at times completely close the duct of the gland, which in consequence becomes swollen, sometimes acutely, generally, however, very insidiously; secondly, the concretion sooner or later causes ulcerations in the wall of the duct, which lead to purulent catarrh. This catarrh then extends backwards to involve the glandular substance. The damming back of the secretion, combined with the inflammation, leads to a chronic adenitis which often results finally in the complete destruction of the parenchyma of the gland. Thus in time the submaxillary gland, which normally is scarcely the size of a chestnut, becomes transformed into a hard tumor larger than a walnut. Many patients first become aware of the existence of the disease and consult the physician when this tumor appears. One is then easily misled into suspecting the tumor to be malignant, especially if in consequence of the inflammation the gland has become adherent. Other patients are disquieted by the purulent discharge from Wharton's duct (pyorrhœa salivalis). The secretion is generally scanty, and only a few drops of pus may be discharged in a day. Occasionally the wall of Wharton's duct may be completely pierced by the calculus. Infection of the surrounding connective tissue and the formation of a phlegmon in the floor of the mouth result. The phlegmon develops always in the posterior part of the floor of the mouth beside the root of the tongue, and readily attacks the arches of the palate. It may be confounded on the one hand with angina phlegmonosa and on the other with Ludwig's angina, which is to be described later.

The diagnosis of salivary calculi is based upon the phenomena described above. The patient, as a rule, complains of darting pains, which from time to time are subject to exacerbations, along the floor of

the mouth and in the region of the gland itself. The affection, as a rule, exists for years. The objective symptoms which are almost always present are the pyorrhœa of Wharton's duct and a moderately painful swelling of the submaxillary gland, with which may be associated the above-described phlegmons of the floor of the mouth with symptoms of acute inflammation. Frequently the stone can be directly demonstrated in Wharton's duct either by sounding the duct, or, in the case of the larger stones, by bimanual examination, palpation of the supposed stone being tried by placing one finger within the mouth and another behind the angle of the jaw.

The aim of the *treatment* is above all the removal of the stone. This can almost always be accomplished through the mouth. Lay open Wharton's duct upon a director as far back as possible, push up the floor of the mouth strongly from without, and the concretion can be removed with the dressing forceps or the sharp spoon. If the calculus has caused a phlegmonous abscess, it is not infrequently found in the abscess cavity. If the enlarged and degenerated salivary gland is not filled with calculi it can be left undisturbed; otherwise it is better to extirpate it.

The other salivary glands and their ducts may be similarly affected, but much more rarely than are the duct of Wharton and the submaxillary gland. Here, too, the swellings may readily be mistaken for tumors. The sublingual gland is rarely the seat of other diseases. Its implication in syphilis has been already mentioned but is of slight importance.

Phlegmonous Processes of the Floor of the Mouth.

Phlegmonous processes of the floor of the mouth which are known under the name of "Ludwig's angina" (so named after Ludwig, of Stuttgart, who first described them in detail[9]) may become highly dangerous.

The disease is developed in the rather thick layer of very loose connective tissue which fills the space between the lower jaw and the genioglossus and mylohyoid muscles. This connective tissue is rich in vessels, especially lymphatics, and contains the sublingual gland and the duct of Wharton. Infection of this tissue may result from ulcerations or from operative or other wounds of the floor of the mouth, from carious teeth, and from infectious diseases of the remainder of the mouth and of the pharynx.

The symptoms of the disease are often of great severity from the outset. Often only a few hours after the patient has noticed the first manifestations of the disease a hard swelling of considerable size may be found to involve the region between the arch of the lower jaw and

the hyoid bone. This swelling increases rapidly and is accompanied by a wide-spreading, thick, œdematous swelling of the skin of the head and neck. The respiration of the patient is soon impeded by the involvement of the deep connective tissue of the neck, as well as that of the ostium laryngis and the mediastinum, in the inflammatory œdema. He generally holds his head as stiffly as possible, the chin being raised high or carried forwards. This posture and the deep cyanosis render the appearance of the patient very striking.

Inspection is very difficult on account of the inability of the patient to open his mouth properly, and it is usually necessary to depend upon the sense of touch. Then it is found that the tip of the tongue is much elevated and often firmly pressed against the hard palate by an indurated swelling of the soft parts situated between the tip of the tongue and the lower jaw. This is continuous with the swelling of the submental region and is frequently, especially at the beginning of the disease, either wholly or for the greater part confined to the sublingual tissues of one side of the mouth. The difficulty in swallowing resulting from this condition may be easily imagined. This together with the disturbances of respiration render the state of the patient one of extreme suffering. To these symptoms are generally soon quickly added those of a general septic infection. Fever ushered in by chills has generally been present from the beginning and now rapidly increases. Icterus, albuminuria, and cardiac disturbances make their appearance and in a great number of cases the patient succumbs to a general sepsis.

The prognosis is rather bad. G. Leterrier [16] has collected thirty-one typical cases of the disease, of which only thirteen recovered; eighteen died, the majority in a few days (two or three) after the beginning of the disease. It is true, however, that some of the cases are from the older literature, and of late the results have been on the whole better; but even to-day there are cases enough in which the exceedingly rapid march of the disease and the early development of symptoms of general sepsis render surgical interference hopeless. But that this may be efficacious is shown from the fact that almost all the cured cases of the above-mentioned collection were treated by free incisions.

Spontaneous cure may also result, as a rule, from the formation of an abscess which breaks through the thin mucous membrane of the floor of the mouth. But this is a rarity and therefore it is necessary to make an incision as soon as possible, at the risk of not finding pus. This incision must be made beneath the chin through the mylohyoid muscle. Between this muscle and the mucous membrane of the mouth is the seat of the disease, and a focus of suppuration will be

most easily discovered after the division of the muscle. It is well to make the incision in the median line, and after division of the muscle to penetrate into the connective tissue on each side of the frenulum, for it is rarely possible when the disease is advanced to determine upon which side the process was originally situated. It is generally necessary to dispense with anæsthesia on account of the impeded respiration.

Sometimes tracheotomy is indicated. The œdema of the glottis may develop acutely in an extraordinarily short space of time, even in the course of half an hour. One should therefore be ready for a tracheotomy in every case of angina Ludovici.

If on account of marked general infection operation seems hopeless, morphine comes into play. Local applications of ice may also alleviate the patient's sufferings. As in every septic disease, alcohol must be administered in large doses. If drinking is very much impeded, fluids must be given by means of a Nélaton's catheter passed through the nose or by the rectum.

Bacteriologically little is known of the disease. Streptococci and staphylococci have been found. In those cases sometimes seen which run a fatal course with incredible rapidity one is led to suppose the presence of especially virulent septic organisms, but with regard to this as yet nothing is known with certainty.

DISEASES OF THE GUMS.

In a number of forms of stomatitis, especially in those which proceed from bad teeth, the gums are the principal, sometimes the sole part of the mucous membrane of the mouth which is affected. These diseases have already been noticed in the sections on stomatitis simplex, stomatitis ulcerosa, tuberculosis, syphilis, and actinomycosis in the article on general diseases of the mouth in Volume VIII.

Pyorrhœa Alveolaris.

This is a form of inflammation peculiar to the gums and requires a special description. The affection occurs, as a rule, in patients who are past their thirtieth year and is generally the result of neglect of the teeth, but sometimes is met with when the teeth are properly cleaned, if tartar accumulates readily upon them. The incisors and the anterior molars are generally first and most seriously affected. The upper teeth are more frequently attacked than the lower.

The commencement of the disease is insidious. As a rule, the patients have noticed for months or years a loosening and a slight

painfulness of the gum and a certain tendency to hemorrhage. A sensation of slight tension being now experienced, the characteristic symptom develops—the formation of small foci of suppuration between the gum and the neck of the tooth. These are emptied easily by pressure on the gum, a small greenish-yellow, generally not fetid, drop of pus appearing upon the edge of the gum. A fine probe inserted at the point of exit of such a drop of pus penetrates far along the neck of the tooth without encountering any resistance; but the periosteum of the jaw, at first at least, is intact. At the beginning of the affection the tooth, which is very often itself quite healthy, is still firmly fixed in its alveolus, but in the further course of the disease it becomes more and more loosened and finally whole rows of teeth may become loose and may even fall out without showing a trace of caries.

This happens the more easily because the disease is often quite overlooked or misunderstood by the patient and unfortunately often by the physician also, so that many a tooth, itself sound, falls a victim to this neglect; and that, too, although with a little attention an error of diagnosis or failure to recognize the presence of the disease is hardly possible.

The *treatment* must be vigorous. Astringent and antiseptic mouth washes, which as a rule are prescribed, are of little or no use. On the other hand, the removal of an otherwise sound tooth, which is frequently advised, is decidedly to be reprobated. The proper treatment is to divide the gum over the whole extent of the neck of the tooth. This operation, which must be performed conscientiously upon all the affected teeth, often demands the exercise of some patience. The procedure is made easier for the patient by the application of cocaine to the gums. After the little abscesses are laid open in this way the cavities thus produced must be thoroughly rubbed with iodoform and afterwards if necessary packed with small strips of iodoform gauze.

Under this simple treatment the disease is completely cured in a few weeks at the longest, but the operation requires frequently to be repeated after two or three weeks and even in especially obstinate cases to be done a third time. Often teeth which are already much loosened become firmly fixed in their sockets again and are preserved indefinitely by the patient. A knowledge of and attention to this, in its symptoms, trivial affection is of great importance on account of the ease with which good results in the treatment are obtained.

DISEASES OF THE LIPS AND CHEEKS.

The mucous membrane of the lips and cheeks, being situated outside of the teeth, is especially exposed to mechanical injuries. These have already been considered briefly elsewhere (see Vol. VIII., p. 11). The lip is also frequently the seat of all possible infections. It is one of the favorite sites for the syphilitic primary lesion as well as for the secondary plaques. Tuberculous ulcerations which occur here and the implication of the upper lip in scleroma have already been mentioned in the chapters upon these affections in the preceding volume. The numerous diseases to which the cutaneous side of the lips is exposed do not belong here.

It should be stated here, however, that diseases of the epidermis which covers the lips may be propagated to the deeper layers of tissue, resulting in cheilitis.

Cheilitis.

Acute cheilitis is due chiefly to traumatisms of the most various kinds. Such infections are rare because of the tendency to rapid healing of the wounds of the lips and cheeks. Still, occasionally, enormous swellings of the lip are met with, due to œdematous infiltration of the loose subcutaneous tissues.

This is still more markedly the case if a furuncle or carbuncle develops upon the lip (this happens more frequently upon the upper lip). Then the lip attains an enormous size and is pushed forwards like a snout, giving the patient a strange and hideous appearance. The resulting suffering is naturally considerable; speaking and chewing are especially impeded. There is also, as is well known, no inconsiderable danger from such infections of the lips.

Similar acute inflammatory swellings may be connected with an infection of the glands of the mucous membranes, but are here much rarer. The glands of the lips, less abundant in the middle and at the corners of the mouth, form an almost continuous layer lying directly upon the muscular tissues. Infection generally occurs in single glands and leads to a disease analogous to furuncle, labial abscess. On account of the depth at which the glands are situated, and the firm texture of the mucosa, considerable inflammatory swelling may develop before the abscess presents distinctly beneath the mucous membrane. Pain and slight fever may be present, but as a rule the general disturbances are slight. The diagnosis is not difficult, and treatment by means of an incision is simple. In fact after a few days an abscess generally breaks spontaneously.

Subacute and Chronic Cheilitis.—In contrast with this acute cheilitis are other forms which pursue a more chronic course. We will first mention the *cheilitis glandularis* described by Baelz, Volkmann, and Unna.

This disease consists in a gradually increasing and not very painful swelling of the lower lip, which finally becomes hard and immovable and may attain very considerable dimensions. In this swelling the mucous glands are especially affected. They enlarge and become kernels of the size of a millet seed to that of a pea. Their excretory ducts are much dilated and they secrete a tough, viscid, or sometimes a purulent fluid. In the further course of the disease they tend to form abscesses, and those furuncle-like abscesses, already mentioned, develop in one gland after another. These abscesses rupture generally into the mouth, and fistulæ covered with scabs remain often for a long time, secreting a more or less abundant muco-pus.

The etiology of the process is as yet unknown. Syphilis has been assigned as the cause, but its connection with the affection is not clearly evident. The annoyances from the disease are almost confined to those arising from the enlargement and the limitation of the mobility of the lip. The internal use of iodide of potassium, cauterization, incision of the abscesses, and painting the ulcerations with tincture of iodine have been recommended. Deep incisions through the mucous membrane will probably contribute most to the attainment of a speedy cure.

Another form of cheilitis, which is best designated as *cheilitis exfoliativa*, has a certain resemblance to the above form. Here the process appears to be even more superficial. We have seen the disease only upon the lower lip. The mucous coat of the lip is relaxed, is of an intense scarlet-red color, and in places is stripped of its epithelium. The secretion is very scanty, but the lip is always moistened with a serous discharge. The substance of the lip is only slightly swollen, yet the lower lip generally appears considerably enlarged, because the patient always keeps it protruded and seeks to avoid contact of it with the teeth and with the upper lip.

The affection is extremely painful. The patient experiences a burning pain even when the lip is at rest, which is increased when the lip during speaking or eating is brought into contact with the neighboring parts. No changes are visible in the adjacent mucous membrane nor in the other parts of the mouth. We have never been able to discover anything definite as regards its etiology. The disease lasts for weeks or months, then disappears rapidly and, as it seems, spontaneously. In the cases seen by us we could not be sure that the treatment instituted had any essential effect upon it. The pa-

tients were considerably relieved by covering the lip with thin strips of cloth smeared with boric-acid salve, which were changed every three or four hours.

We have met with the disease in middle-aged men; it appears to be very rare.

The most common cause of chronic enlargement of the lips is eczema of the labial skin and mucous membrane. Such "scrofulous hypertrophies" of the lips occur especially in individuals who have tuberculous disease, particularly tuberculosis of the tissues of the head or neck, but with almost equal frequency in those with latent tuberculosis. The vermilion border of the lips shows the well-known tendency to crack; rhagades are formed which may penetrate more or less deeply into the mucous membrane, become covered with brownish scabs, and groove the border of the lips in all directions, dividing it in such a way as to suggest a resemblance to garden beds. (We may here remark, in passing, upon the frequent connection of such chronic eczemas with chronic rhinitis.) Erysipelatous or lymphangitic inflammatory processes frequently have their starting-point in these rhagades and lead to a still greater swelling of the substance of the lips. This swelling is, as a rule, more marked in the upper lip and gives the face a very characteristic appearance. The border of the lip is puffy, tense, shining, generally of a blue color, and covered with crusts and chaps. The lips no longer close completely, and project strongly. If, as is frequently the case, there is associated with this condition obstruction of the nose from hypertrophies of the mucous membrane or from enlargement of the pharyngeal tonsil, the external signs of mouth-breathing are also manifest, the result being the well-known "scrofulous habitus" of the face. The section on macrocheilia (p. 40) should be consulted in this connection.

The treatment is that of scrofulous eczema in general. The application to the rhagades of mild mercurial ointment (one per cent. of white precipitate) once or twice daily is especially to be recommended.

The mucous membrane of the cheeks is, more rarely, subject to diseases of the mucous glands which are quite analogous to those of the lips. Vigier[11] has called attention to the diseases of the lymph glands situated in the cheeks, which heretofore have hardly been noticed.

TUMORS.

Nearly all known forms of tumors are found occasionally within the mouth, some kinds even with especial frequency. And the different kinds of tumors have each their favorite localization. In

what follows the topographical relations of the tumors will be taken account of only to the extent of considering tumors proceeding from the jaw, which are characterized by certain peculiarities, apart from those of the soft parts. The more benign forms will be considered seriatim and then those which are malignant.

Benign Tumors of the Soft Parts.

Fibroma.

Pure fibromata are very rare tumors in the mouth. They are found most frequently upon the tongue. Here they may either be pedicled and resting, as it were, upon the surface or be situated deep within the substance of the tongue. Both forms may be situated in one of the lateral halves of the tongue or upon the median line posteriorly. The latter appears to be relatively the most frequent location. Most of the reported fibromata of the tongue belong to the hard forms. They consist essentially of tissue poor in cells which is sometimes almost indurated or cicatricial in character. Accordingly they have a firm feel, although if deeply situated, their hardness is masked to some extent by the overlying muscular layers. Other tumors are softer and richer in cells and in fluid. These are generally also more vascular. The pedicled tumors are usually of this sort. Corresponding to their location and to the development of their pedicles the appearance of these tumors varies greatly. Now they are seen as small polypus-like formations with broad pedicles, again they are found as firm, flat, elevated tuberosities upon the dorsum of the tongue (Albert). Or they may occur as large tumors from the size of a walnut to that of an egg, which are sometimes provided with a thin pedicle, sometimes have a broad base. Again, in other cases, at the first glance only a more or less considerable swelling of one-half or of the base of the tongue is noticed, which at first does not suggest a fibroma, and it is only upon palpation that the finger detects the hard and sometimes movable tumor deeply embedded in the tissues.

Common to all these tumors and their most essential diagnostic feature is their distinct demarcation from the surrounding tissues. This is easy to determine in those which are pedicled or sessile upon the surface of the tongue with a broad base, but sometimes very difficult in the case of those deeply situated. Moreover, it is to be remembered that the tumor sometimes, though rarely, sends out finger-shaped processes into the parenchyma which make the demarcation less apparent.

Slow growth is an important characteristic of fibromata. Many patients have had tumors for decades without becoming apprehensive on account of their growth. When the tumor has reached a certain size it sometimes suddenly begins to grow with rapidity, a fact which frequently first induces the patient to seek advice. The annoyance caused by fibromata of the tongue is in general trifling, so long as they have not reached a considerable size. If they are large they are troublesome by impeding swallowing (especially if located at the base of the tongue) or speech, in consequence of the imperfect movability of the tongue. A movable, pedicled tumor is occasionally caught between the teeth during mastication and is injured.

More rarely and chiefly in the softer and more vascular tumors their mucous surface, which is exposed to frequent mechanical injuries and thus has become thinned and less resistant, begins to ulcerate and then bleeds easily. Quite exceptionally even abundant hemorrhages may occur which demand operative interference (Haynes). In such cases the tumor, or rather its ulcerated surface, may occasionally give severe pain; generally this is not to be expected.

The age of patients with fibroma of the tongue varies extraordinarily. We find congenital tumors in little children (Barling) and on the other hand tumors, which have only recently attracted attention, in people sixty years old. Often in tumors of long duration there is no knowledge of the time when they originated. In general it is not an error to regard a good proportion of fibromata as congenital in their origin. According to the size which they attain and the rapidity of their growth, they are discovered at an earlier or later period.

The *diagnosis* of fibroma of the tongue is not always easy. If one finds a firm tumor which has a pedicle and has grown slowly, or a round hard nodule which is freely movable and well-defined from the surrounding tissues, the diagnosis is not difficult. But if the growth is comparatively soft and irregularly shaped, its surface, it may be, presenting an ulceration and inclining to bleed easily, mistakes may readily be made.

Broad flat fibromata, especially if multiple, are easily confounded with the sclerosing superficial glossitis of tertiary syphilis. In the case of the latter it is to be noticed that, as a rule, the papillæ over the sclerotic plaques appear atrophied, as if the mucous membrane were shaved (Fournier). Analogous appearances in the forms of fibroma above described have not been reported.

The deep fibromata are often difficult to distinguish from lipomata, sarcomata, and from deep or, if they are ulcerated, even from superficial carcinomata. They may also be confounded with the in-

filtrations of deeply situated sclerosing glossitis luetica, with incipient phlegmon of the tongue, with decubital indurations, and finally perhaps even with deep-lying lingual cysts. Some of these mistakes are easily prevented. An incipient phlegmon is known by the fever, the pain, and the disturbance of the general health. The decubital infiltration (or ulcer) is easily cured by the removal of the offending tooth. Deep lingual cysts will always give at least a hint of fluctuation. Other errors, such as confounding fibroma with lipoma, are of slight moment.

The distinction from gumma and sclerosing infiltrations is important. These latter may also exist unchanged for a long time, or with only slight growth, and may cause hardly any inconvenience, so that a mistake is easily made. When the history fails us and no other signs betray the existence of syphilis, the only aid in the differential diagnosis is the generally undefined limitation of the syphilitic products, but that is a very unreliable sign and often hard to determine. In such difficult cases antisyphilitic treatment is in place and will quickly clear up the diagnosis.

Of the other tumors, carcinoma, if ulcerated, can be excluded in the majority of cases by the peculiarities of the ulceration. In ulcerated fibromata we generally find upon the summit of the tumor a shallow erosion of a dirty-gray color—a flat surface upon the convexity of the tumor—not excavated in the surrounding infiltration as in carcinoma.

From deep as well as from superficial carcinoma, fibroma is distinguished to a certain extent by the fact that the former occurs almost always upon the edge, the latter in the middle of the tongue. Here, too, the absence of sharp demarcation of the tumor leads us to suspect carcinoma.

Confusion with sarcoma, which also is often sharply defined and is localized at the same points, can generally hardly be avoided.

Thus it is seen that the diagnosis presents many difficulties. But in practice, first an endeavor must be made to exclude syphilis. That being done, perhaps by the aid of mercury or iodide of potassium, if the diagnosis is still not quite clear, an operation is performed. If the case is one of fibroma, the tumor is very easily turned out, as a rule, after a simple incision. Cocaine anæsthesia suffices for this, and preliminary operations are not necessary. But if the enucleation is attended with difficulties, one may at first make only an incision and remove a small part of the tumor for microscopic examination. According to the result of this it will be easy to decide whether an operation of greater magnitude is necessary, and how it should be performed.

The other parts of the mouth are very rarely the seat of fibroma. Partsch[12] describes a soft-pedicled fibroma, the size of a hazelnut, having greatly dilated vessels and ulcerated in places, situated on the hard palate. He also observed a lobulated fibromatous growth at the junction of the lip with the alveolar process of the upper jaw, caused by the pressure of a badly fitting set of artificial teeth.

On the uvula and the palatine arch small polypous fibromata are not very rare. These never give any trouble and are only accidentally brought to notice.

Padieu[13] saw a cystic fibroma on the inner surface of the upper lip, the histological structure of which suggested an implication of the mucous glands of this region. Demme[14] has observed a fibroma, of the size of a walnut, upon the mucous membrane of the cheek, which during mastication and speaking was often caught between the teeth and, its surface having ulcerated as a result of these traumatisms, caused much pain and annoyance.

On the border between the palate and the palatine arches encapsulated and easily enucleated tumors, up to the size of a hen's egg, are found, which grow slowly and only give annoyance by narrowing the isthmus faucium. They are often described as fibromata. It appears, however, that many, even a majority of these tumors, belong to the endotheliomata, which are to be described below.

Lipoma, Myxoma, Myoma, and Transitional Forms.

Transitional forms between fibromata and lipomata (fibrolipomata) are found sometimes in the mouth, and most frequently upon the tongue (Poncet,[15] von Bergmann, quoted by Krausnick,[16] Albert[17]). They are generally situated within the substance of the tongue, but are very superficial and considerably elevated. They are from a clinical point of view equivalent to the unmixed lipomata, not being distinguishable from them, and no attempt is generally made to keep them separate.

Knoche[18] has made a collection of cases of true lipomata of the mouth and Krausnick[16] of those of the tongue, to which many more recent cases can be added.

Lipomata of the tongue are most often met with. They are almost always present singly (Barling and Malon each report one case of multiple lipomata), generally originate immediately below the surface, and may be considerably elevated above it. Occasionally they originate between the muscles and may reach the surface only after a long period of growth. They occur with relative frequency upon the tip of the tongue (see Mikulicz' Atlas, Plate XXXV., Fig. 3, Rydygier's case), next on the lateral margins, and rarely at the base.

They have generally a smooth, exceptionally a lobed surface and a round or oval form. Their size may become very considerable, almost as large as a hen's egg (Rydygier's case). The annoyance which they cause is often surprisingly slight and is due mainly to the mechanical impediments arising from the size of the tumor. But in these tumors, also, there may develop superficial erosions, ulcerations, and hemorrhages.

The diagnosis, on account of the generally superficial seat of the tumor, is not especially difficult. The yellowish gleam of the fat through the thinned mucous membrane might, it would seem, lead more often to a correct diagnosis than actually appears to be the case. Deeply situated lipomata can hardly be distinguished from fibromata. Practically this fact is of no importance.

On the other hand, a confusion with gummata might be unpleasant. These may have quite the same consistence and, if deeply situated, are very readily confounded with lipomata, the more so if their softened centre shines through the mucous membrane. But the lipomata are, as a rule, very sharply circumscribed, a fact which will generally protect the patient from an unjustified suspicion.

The treatment of lipomata presents no difficulties. They are easily shelled out after incision of the mucous membrane.

On the floor of the mouth lipomata are sometimes met with. They generally present a lobulated structure, sending out processes between the genioglossi and hyoglossi muscles, or, in other cases, beneath the skin of the neck. But these processes are demonstrable with difficulty or not at all by palpation.

Lipomata situated here cause considerable annoyance at an early period by pushing up the tip of the tongue and thereby interfering with the taking of food. The diagnosis can hardly present any material difficulties. Confusion with sublingual dermoid cysts is perhaps most likely to occur. But the latter are often located exactly in the median line, the former only very rarely. They may be situated on both sides of, and symmetrically with respect to the median line, but more frequently they are in the lateral portions of the floor of the mouth. In the case of dermoid cysts the palpating finger often leaves an impression which it never does in the case of lipomata.

Enucleation is here also always extremely easy.

Lipomata of the cheeks occur about as often as those of the floor of the mouth. They are also submucous and are to be distinguished from the tumors which are found in the fatty tissue of the skin of the cheeks on the corpus adiposum malæ.

The submucous lipomata may cause great annoyance from the injuries received during mastication, just as the above-mentioned

fibromata of the cheek. In this locality transillumination of the cheek may be utilized for diagnosis. A lipoma then appears dark, a cyst translucent.

On the lips pure lipomata are great rarities. Many of the tumors described as such appear rather to be cavernous angiomata with abundant deposition of fat. But lipomata do occur and are distinguished from angiolipomata by their more distinctly submucous seat, their sharper circumscription, the absence of compressibility, and their greater softness.

Lipomata of the gums and the palate are extremely rare. One of each has been observed (Knoche[18]). Both tumors were of the size of a pigeon's egg, were situated directly beneath the mucous membrane, and were easily enucleated.

As from the fibromata to the lipomata, so also from the former to myxomata a series of transitional forms are met with, but these latter are manifestly much more rare in the mouth than the fibrolipomata. Cardone[19] describes a papillary pure myxoma upon the buccal mucous membrane of a boy one and a half years old. He also mentions other similar observations. McLeod[20] has operated on a fibromyxoma of the soft palate which manifestly sprang from the fossa pterygo-palatina.

A quite unique case is one of fibromyoma of the base of the tongue, slowly developed, and having a broad base, which E. Blanc[21] operated upon and described. The interspersed unstriped muscular fibres give the tumor a quite exceptional character, so far as tumors of the mouth are concerned. An analogous case of Fith's is mentioned by the author.

Chondroma, Mixed Tumors, Teratoid Tumors.

Tumors in which cartilage or bone is found occur quite rarely in the soft parts of the mouth. As a rule they are seen rather by the pathologist than the clinician.

Unmixed chondromata are extremely rare. Tapie[22] described one, of the size of a nut, situated in front of the lateral incisor in the upper lip. The tumor which was movable was extirpated and proved to be a chondroma with formation of cancellous spaces, *i.e.*, beginning ossification.

Somewhat more common are various mixed tumors and transitional forms.

Of these the first to be mentioned is a quite typical but rare form of tumor which really only projects into the mouth, while its place of origin is in the tissues of the parotid gland or in th palatine

fossa. These are the tumors, described by F. Krause[23] as "hourglass tumors," which, growing slowly, push forwards so strongly the soft palate on one hand and the region of the cheeks on the other, and are so immovably fixed in the tissues, that one is led to suspect an exceedingly malignant tumor of the soft palate or of the parotid. These tumors may present various types of tissues. Von Bergmann regarded his cases as fibromata. Krause found a fibromyxoma. The case of McLeod[20] appears also to have been a fibromyxoma. An analogous tumor operated upon in the Breslau Surgical Clinic contained connective tissue, cartilage, mucous tissue, and a few epithelial elements. The knowledge of these tumors is not unimportant, since one might easily mistake them for inoperable tumors of the palate or pharynx. The slow growth of the tumor and the movability of the anterior palatal arch, and sometimes also of the skin of the cheek over it, may, however, lead to the correct diagnosis. The enucleation of the tumors can usually be effected easily and even for the most part without the use of the knife after incision of the mucous membrane, by the way of the mouth. Only in case of large hourglass-shaped tumors is an external incision demanded. Preliminary operations are not necessary.

The other mixed tumors have more pathological than clinical interest, but a knowledge of them is necessary in order to avoid mistakes.

Such formations appear to be relatively frequent at the base of the tongue. Tumors have been described in this situation by Zahn, Ziegler, Kraus, and recently by M. B. Schmidt,[24] which were all found at autopsies. They contain besides cartilage and bone also amyloid substance surrounded by coarse-fibred hyaline connective tissue. Many contain also sparse or abundant giant cells. Schmidt thinks, and probably with reason from an anatomical standpoint, that these formations are not true tumors but products of the metamorphosis of parachondral connective tissue. He compares them with the localized amyloid tumors of the conjunctiva and of the respiratory tract. If, however, they ever came under clinical observation they would certainly be regarded as tumors. They appear as hard, approximately spherical nodules, situated at the posterior portion of the root of the tongue and but slightly or not at all elevated. The mucous membrane is movable over them and they themselves manifest at least a limited movability with reference to the surrounding tissues. They occur singly or in small numbers. The reported cases were all those of middle-aged adults who had died from various other diseases. With regard to the clinical manifestations which the growths may have produced, nothing is known in any of the cases.

A fibroma with a cartilaginous centre has been described by T. Berry.[25] It was situated upon the right edge of the tongue and was considerably elevated. In this connection a series of tumors should be mentioned, which have been described under the names of lipo-chondromata, hairy pharyngeal polypi, and the like. O. Weber[26] observed a lipomatous infiltration in a cartilaginous tumor of the tongue, and Knoche (Malon) cites two similar cases. In one there were osseous centres, in the other masses of cartilage in the tumor. The last-mentioned tumor was covered with genuine skin and forms a transition to a fourth case in which the tumor, containing only fat, connective tissue, and vessels, was covered with epidermis.

Such tumors, consisting of fat and connective tissue and covered with typical epithelium and even more or less abundantly beset with lanugo hairs, have been repeatedly observed upon the anterior arch of the palate. Arnold[27] mentions many such cases, in part in individuals who were otherwise imperfectly developed, and so also does R. Otto.[28] Roncalli and Gradenigo[29] described a small tumor of this kind and Kafemann[30] one still more complicated, containing also gland lobules with small retention cysts.

Since the majority of such growths are certainly or probably of congenital origin, and since, moreover, those who have them are often afflicted with other developmental defects, it is certainly justifiable to regard these hairy polypi as teratoid tumors analogous to dermoids. This is not the place to discuss fully the subject of their position in oncology. Clinically they are for the most part of slight importance. The patient has often no suspicion of their existence. In some cases they have caused serious dyspnœa in the newly born infant and have required an operation. Their removal is not difficult, since they almost always have a pedicle.

In passing, the parasitic twin malformations, which are known as epignathi, may be mentioned. They are almost solely of interest from a teratological point of view.

Tumors of Vessels.

Tumors consisting of new-formed or dilated vessels belong also to the benign tumors of the connective-tissue series. They are divided into hæmangiomata and lymphangiomata, although in the mouth peculiar forms intermediate between the two occur with especial frequency.

Hæmangiomata.

Tumors consisting wholly or chiefly of blood-vessels occur in three types, which, however, are not sharply separated from one another.

Telangiectasis and Cavernous Angioma.

The telangiectasis or angioma simplex or plexiforme consists of a compact system of fine canals made by the abundant formation of small new vessels, which are coiled and interwoven with each other. Tumors of such tissue may be small, or sometimes they are massive and of great extent. They are especially found at points where em-

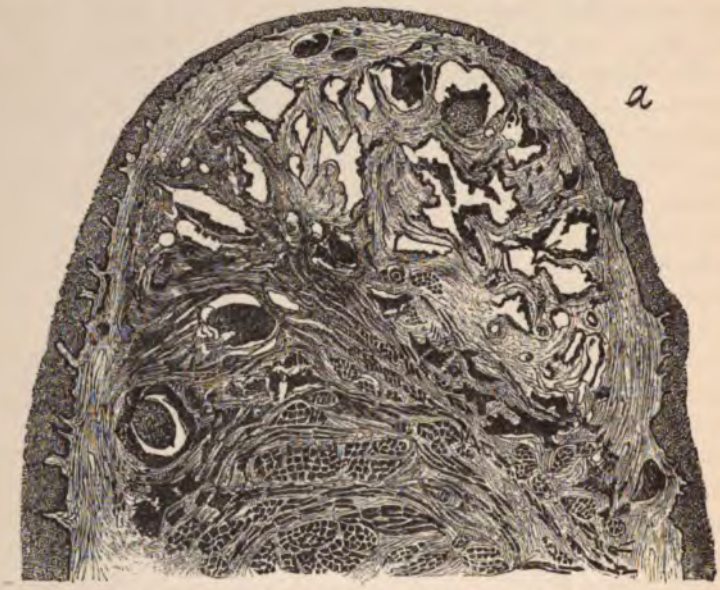

Fig. 2.—Represents a Section through a Cavernous Angioma of the Tongue. There are seen, especially at *a*, large and wide sinuses which in part still contain blood (the deeply shaded parts), and which are surrounded by fibrous connective tissue. In the deeper layers are longitudinally and tranversely cut muscular fibres. (Taken from Partsch, "Die Geschwülste der Mundgebilde," Fig. 114.)

bryonal fissures have become closed during the process of development, Virchow's fissural angiomata. They are clinically, and often also anatomically, difficult to distinguish from the second form, angioma cavernosum. This variety, also called the erectile tumor, is characterized by the fact that the vessels of which the tumor is composed are in the form of widely dilated, thin-walled sinuses (Fig. 2). These communicate with each other by wide openings, and thus a spongy tissue is formed, which is quite analogous to the corpora cavernosa of the penis.

After extirpation, when the blood has been evacuated, the telangiectasis shows almost no macroscopically recognizable cavities. It looks like a soft but solid tumor composed of separate lobules resem-

bling true glands. The cavernous angioma under the same conditions is much more reduced in size. It appears firm and tough and upon section numerous dilated spaces are seen, now reduced to narrow slits but plainly recognizable.

Clinically the telangiectasis appears as a smooth, bright red or bluish-red tumor, with very irregular but generally quite sharply defined borders. Some of the larger vessels on close inspection can be seen as red streaks. The cavernous angioma has a bluer color and an uneven, generally knobbed surface (at least in the larger tumors), and appears lobulated to the touch. The cavernous angioma is more easily emptied by pressure than the plexiform and swells more than the latter when there is any obstruction to the efflux of blood (in crying and the like).

Many transitional forms unite the two groups of tumors. A property common to both groups is that, besides the newly formed vessels, other tissues help to make up the substance of the tumor. This is especially true of the telangiectases. Almost uniformly large numbers of fat cells are embedded in the lobes of which these tumors are composed. Connective tissue, rich in cells, is also present in considerable amount. When these elements are especially abundant, the terms angiolipoma or sarcomatous angioma (the latter should not be confounded with the angiosarcoma to be described farther on) have been used. The clinical appearance of the tumors, however, is not changed by these admixtures.

The great majority of both forms of angiomata are certainly of congenital origin. But their chief growth takes place subsequently, as a rule. They attack the surrounding tissues indiscriminately and may even penetrate into the underlying bone. In so far they have a certain malignant character and they exhibit this also by the fact that they readily recur after extirpation. But they have not the power of forming metastases, and recurrence is always due to portions of the tumor remaining after the operation.

The site of these tumors varies much. In general, the favorite locality for the telangiectases is the lips, especially their exterior surface, occasionally on the vermilion border. The cavernous form occurs much more frequently, almost exclusively, upon the true mucous membrane of the mouth. We find it in all parts of the mouth, but here again the tongue is first in order of frequency. Jullian[31] has collected a series of cases of these tumors. They are often multiple, and are located, it seems, generally on the anterior part of the tongue, sometimes on the edges and sometimes on the tip. They are less common on the dorsum of the tongue, but do occur there, those situated on the edges often growing in that direction. Sometimes

they attain a very considerable size and then generally grow towards the floor of the mouth and may extend for some distance downwards in the submental region. But even without displacing the floor of the mouth such an angioma may attain the size of the fist (case of Landerer").

Cavernous angiomata have been observed on the cheek and the lips. In the former locality they are generally on a line with the corner of the mouth; on the mucous membrane of the lips, near or in the median line beside the frenulum linguæ—all "fissural angiomata." Those rarities, cavernous tumors of the palate, and especially of the uvula, belong in the same category. Not rarely it happens that angiomata are present in many parts of the mouth at the same time, and, further, there are such tumors which extend continuously over lip, cheek, palate, and even farther.

The clinical importance of angiomata is not slight as soon as they show a decided tendency to increase in size, as often happens. Then they may sometimes grow with great rapidity and attack indiscriminately all the surrounding tissues. But, as a rule, their growth is slow, though even then they sometimes attain enormous dimensions. They are rarely very large at birth.

Small angiomata cause annoyance chiefly because they begin to bleed after some injury, and the hemorrhage from the cavernous tissue is difficult to check, but as the dimensions of the tumors increase, the troubles which they cause become more serious. Tumors situated near the surface of the face may cause great disfigurement and may impede mechanically the taking of food. But a more dangerous complication is the thinning of their cutaneous or mucous covering, which sometimes occurs in cavernous tumors. This occurs, as a rule, only in certain portions of the tumor and is frequently caused by several cavernous spaces coalescing and forming a blood cyst which rapidly expands outwards, in the direction of least resistance. Although cavernous tumors in general belong to the venous system, the blood pressure within them is frequently considerable. This is most pronounced when, as the tumor grows, freer communication with arteries is established, and, it may be, an aneurysmal dilatation of the latter occurs. Then the hemorrhages from the tumor during an operation may be very severe (Landerer," G. Fischer"), and accidental injuries become dangerous.

Not much less serious are the occasional inflammatory processes which occur not infrequently in tumors covered with thin mucous membrane. These are attended with the same dangers as is phlebitis of varices of the leg—especially those of general septic infection.

If these dangers are considered, one is easily convinced that the

removal of these, in an anatomical sense benign, tumors is urgently necessary. This can be effected with success only by surgical interference.

For small and inaccessible tumors on the one hand and for the very large ones on the other, which cannot be extirpated with the knife, ignipuncture may be regarded as an effective method of treatment. With the Paquelin or the galvano-cautery at a red heat (white heat increases the danger of hemorrhage) a number of punctures, not too far apart and not too superficial, are made into the tumor. The eschar is eliminated aseptically and by the subsequent cicatricial contraction a number of the surrounding vascular spaces become obliterated. In this way with patience tumors of considerable size may be caused to disappear, and, unless the pressure of blood within the tumor is very great, without danger. It is necessary to be on the watch for the small recurrent tumors which appear in the vicinity of the original growth, and to destroy them before they have attained a large size. This applies with especial force to the telangiectases of children.

Excision of the tumor leads to a cure more rapidly and generally more certainly. When it can be done without danger, it is therefore the best procedure. But it is often a difficult, and not very rarely a dangerous operation (case of G. Fischer), and is therefore not always to be recommended. The deformity and the impairment of function due to extirpation are often, indeed generally, not greater than that after the cauterizations; but one must be sure that he can control the hemorrhage. On the lips and the cheeks that can be effected by compression during the operation; on the tongue, if necessary, Langenbuch's method of securing local anæmia by ligating the base of the tongue is to be recommended. When the tumor is once removed, the hemorrhage is generally easily checked, the danger being chiefly during the operation.

The older procedures—injections into the tumor of substances to cause coagulation and caustic applications—are hardly to be considered to-day, the former because it is dangerous, the latter because it is ineffective.

With regard to electrolysis, which has occasionally given good results, further experience is necessary.

Nævus.

There is still to be mentioned that peculiar form of congenital vascular dilatation which is allied to telangiectasis and may pass into it by easy gradations—the nævus vasculosus. It occurs on the oral mucous membrane probably only in combination with nævus of the

skin of the face, and is found generally on the mucous membrane of the lips and cheeks only, much more rarely on the gums, the palate, and the tongue. It is only cosmetically important. Therapeutically it is unnecessary to do anything for the condition as it occurs in the mouth.

Cirsoid Aneurysm.

The aneursyma cirsoides or racemosum, which occurs almost exclusively upon the head, is a mass of thin-walled, dilated, tortuous, and freely anastomosing arteries. Since for its formation a very abundant development of new vessels must occur, and since these newly formed vessels actually constitute a true tumor, it is justifiable to separate them from the aneurysms and class them with the angiomata. But for our subject they are of little importance. Tumors of this kind, arising on the head or face, may spread to the mucous membrane of the mouth, and here form considerable swellings. But this occurs very rarely. Naturally it is the cirsoid aneurysm of the external maxillary artery which most frequently reaches the mouth. The thin-walled arterial vessels may extend to the mucous membrane of the lips and gums and even to the palatal arches. They may, especially on the gums, as a consequence of mechanical injuries and after the extraction of teeth, become the source of profuse arterial hemorrhage which it is difficult to check.

LYMPHANGIOMA.

In the mouth and especially on the tongue there is found a tumor-like new formation of lymph vessels of three different forms—nodular or wart-shaped, diffuse, and cystic (E. O. Samter[34]).

The mode of origin of these tumors is still the subject of controversy. But of late, opinions seem to be inclining to the view that the primary condition is a new formation of lymph vessels and of lymphoid and fibrous connective tissue of congenital origin, and that subsequently these newly formed lymph spaces, especially under the influence of inflammatory processes, enlarge and spread through the surrounding tissues. We cannot enter more fully here into this pathologico-anatomical question. The work of Nasse[35] may be consulted with regard to it. It is, however, of interest that the lymphangiomata, at least those of the mouth, show a predilection for those places where embryonal fissures have united, just as do the "fissural" hæmangiomata.

Clinically it is important that the great majority of lymphangiomata are discovered in early youth and that they are probably without exception to be regarded as congenital. But they frequently long

remain small and then for a time grow rapidly. These periods of growth coincide with inflammatory processes which develop in the tumors. The sinuses which traverse the entire tumor and which everywhere freely anastomose with each other, extend to the surface of the mucous membrane. After traumatisms, and even without them, an infection of the superficial portions easily takes place, and this propagates itself through the entire system of sinuses. Thus, especially in the diffuse lymphangioma of the tongue, there frequently arise attacks of glossitis which lead to marked swelling of the tongue, and so produce serious disturbances. Subsequently the tumor returns but incompletely to its former size. These inflammatory processes are attended with considerable danger for the patient. Thus in a case depicted in Mikulicz Atlas' (Plate 28, Fig. 3) and also described by Samter (Case IV.) there had been previously an acute phlegmon of the connective tissue of the neck with œdema of the glottis, and it had been necessary to ligate both lingual arteries, to make incisions into the tongue and into an abscess which had developed beneath the jaw, and finally also to perform tracheotomy.

Since all forms of lymphangiomata occur most frequently in a typical way upon the tongue, they will be first described as they appear there.

The most peculiar picture is afforded by the nodular or wart-shaped lymphangioma. This occurs on the edges or upon the dorsum of the tongue in the form of rather firm tumors, varying in size, sometimes larger than a walnut. These have a papillary surface and at the first glance suggest an hypertrophy of the lingual papillæ of the region. On closer inspection, however, it is noticed that they do not present the fine hair-shaped growths of the hypertrophied papillæ filiformes nor the red knobs, of the size of the head of a pin, of the papillæ fungiformes, but that they are formed of small vesicles containing fluid. These are in part easily recognized macroscopically, in part only to be made out with a lens. They project above the surface as hemispheres. Their contents shine through the covering of the vesicles, being either quite clear and transparent or cloudy, of a whitish or yellowish color, sometimes, because of small extravasations of blood, even a bluish or reddish color. These hemispherical vesicles are sometimes crowded closely together in one place, forming a considerable tumor, or they may be irregularly scattered over the surface of the tongue, forming a number of small foci. Thus arise plaque-like nodules which simulate a solid tumor or smaller nodules like papillomata. Both of these forms are considerably elevated above the surface as knobby tumors. Confusion with solid

tumors is therefore easy, and special notice must consequently be taken of the presence of the characteristic vesicles.

The appearance of the tumor is also complicated by the fact that the patients as a rule first consult the physician when they have been attacked by a fresh exacerbation of glossitis. Then, in addition to

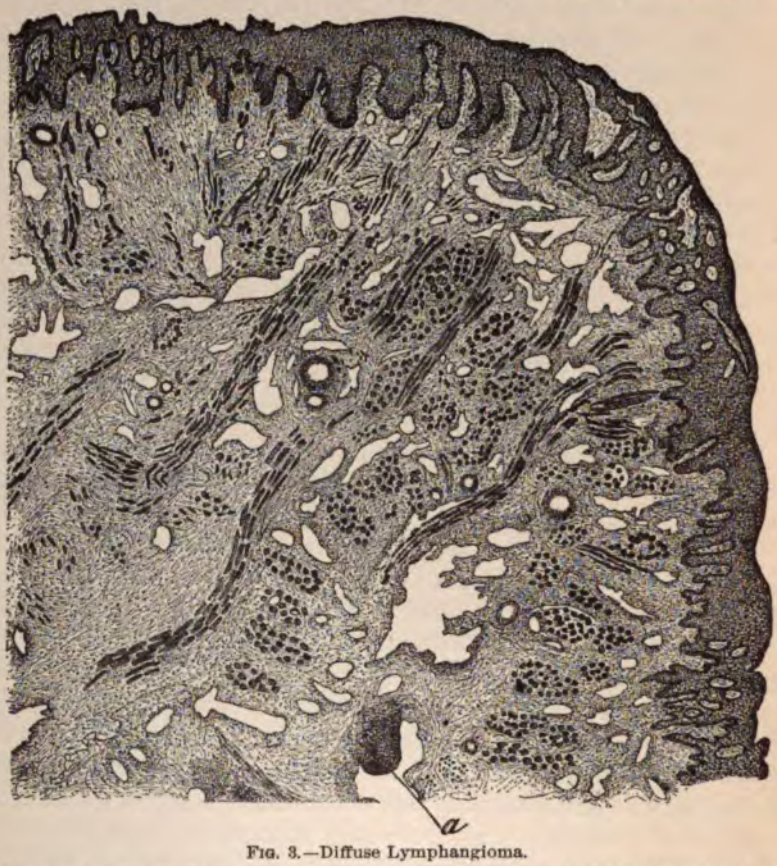

FIG. 3.—Diffuse Lymphangioma.

The illustration gives the macroscopic appearance of a tongue with the formation of diffuse lymphangioma. Cavernous spaces are seen, widely dispersed through the substance of the tongue and surrounded by connective tissue poor in cells. These are now empty, but during life were filled with lymph. The bundles of muscular fibres are widely separated by this tissue and are much atrophied. At *a* an infiltration with round cells is seen in the wall of a cavernous space. (After Partsch: "Geschwülste der Mundgebilde," Fig. 117.)

these knobs and warts, one-half or the whole tongue is found to be much enlarged, of a more or less vividly red color, and sensitive. If the infection is severe, fever, swelling of the glands, and even a phlegmon of the connective tissue of the neck may also develop. So in the presence of manifestations of severe glossitis one may easily overlook the perhaps inconspicuous tumors.

Materially different is the appearance of the diffuse lymphangioma which generally occurs as "macroglossia." Here, too, we find, as characteristic of the tumor, the small vesicles beneath and shining through the mucous membrane, the contents of which present the same differences as have been mentioned above. But here there are not formed out of these vesicles large aggregations which project above the surface as circumscribed tumors, but the whole affected part, generally a considerable portion of the tongue, is diffusely enlarged.

The wide diffusion of the tumor within the substance of the tongue

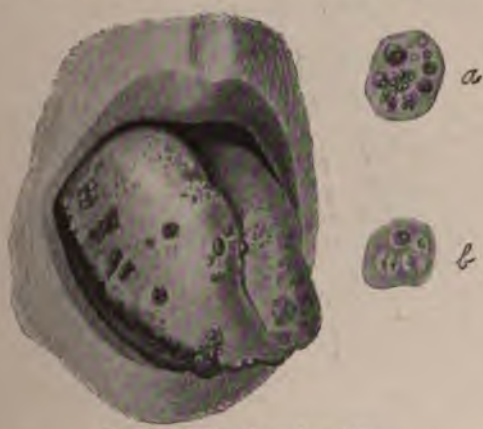

FIG. 4.—Diffuse Lymphangioma.

Fig. 4 represents a diffuse lymphangioma from a boy five and three-quarter years old. The right side of his tongue had been abnormally large from birth. For a year there had been exacerbations of inflammatory swelling of the whole tongue, which had lasted from eight to ten days. The tongue completely fills the mouth. Its right half is much thicker than the left, and the line of demarcation is represented by a deep furrow. On the edges of the tongue are shallow notches, also small ulcers, which correspond to the impressions of the teeth. In place of the papillæ filiformes are small vesicles (represented at *a* and *b* as magnified by a hand lens) filled with a pale red clear fluid, which at some places have become confluent. The whole tongue is hard. Fœtor ex ore is present. There is no fever. Speaking and eating are much impeded. After the disappearance of the inflammation two wedge-shaped pieces were excised and the cautery was applied, the operation resulting in cure. Microscopically the growth was found to be a typical diffuse lymphangioma. (From Mikulicz' Atlas, Plate XXXVII., Fig. 1.)

causes a considerable increase in the size of the whole organ, which, if it no longer can find room within the cavity of the mouth, protrudes between the teeth and may then remain permanently outside of the mouth. This position naturally causes many annoyances, which are increased by the fact that the portion of the tongue protruded through the lips is exposed to all kinds of traumatisms. In this way, too, the opportunities for infection are much increased, because the projecting portion of the tongue quickly becomes dry, the mucous membrane chaps, and the deeper tissues are freely exposed to patho-

genic organisms. Thus here, even more than in the other forms, occasion is given for the development of chronic relapsing glossitis. Moreover, the disease from the first occupies a much larger portion of the tongue, and such inflammatory processes will spread correspondingly more rapidly and widely. The teeth, especially those of the lower jaw, may be much displaced by the protruded tongue.

FIG. 5.—Macroglossia.

Fig. 5 represents a severe macroglossia from a woman twenty-five years of age. Her lower jaw was so deformed that, when the mouth was firmly closed, the incisors were separated from each other 2.5 cm. By reason of this separation and of a marked lengthening of the lips, the lower lip especially, the tongue was able after a fashion to remain within the cavity of the mouth. The teeth of the lower jaw, especially the incisors, were much displaced outwards and were almost horizontal. Besides the diffuse enlargement of the tongue there was on the right side of the lower surface of the tongue an abundant formation of circumscribed nodules. An operation was performed with the removal of several wedge-shaped pieces of the tongue. (Photograph from the Breslau Surgical Clinic.)

Even the alveolar process or the entire lower jaw may thereby become deformed.

Cystic lymphangioma, the lymph cyst, is a great rarity. In a case depicted in Mikulicz' Atlas (Plate XXVII., Fig. 1), the anterior part of the patient's tongue hung from his mouth and down upon his chin, as a tumor the size of an apple, covered with a dry and chapped mucous membrane, so changed as to resemble leather. Upon removal (by excision of a wedge-shaped piece) the tumor

proved to be a single cyst, the nature of the walls of which pointed unmistakably to a lymph cyst.

The same forms of tumors occur upon the lips, especially the upper lip, the cheeks, and the floor of the mouth, as upon the tongue. The inflammatory manifestations which occur so regularly in lymphangiomata of the tongue may also develop in these locations, but much more rarely. There can hardly be any doubt as to the diagnosis in the case of diffuse and cystic lymphangiomata. But the nodular form may be confounded with malignant tumors, especially in persons advanced in years.

The treatment of these tumors varies in the individual case, according to the type and the extent of the growth. For the nodular lymphangiomata, if their location admits, extirpation with the knife or the Paquelin cautery will be the most suitable procedure. The flat lymphangiomata, situated at the base of the tongue, generally give no trouble, and if they show no tendency to grow may be let alone. The removal of the portions of the tumor which project above the surface is not always sufficient, since the tumor has generally penetrated some distance into the deeper tissues. It is necessary therefore to excise a wedge-shaped piece of the tongue. In the diffuse form we shall not, of course, be able to remove all of the affected tissue by excising such wedges, but by the combination of several of such incisions carried in different directions, the tongue can be so far reduced in size that it can be returned to the mouth. The operation should not be done at a time when the tissues traversed by the lymphangioma are infected and infiltrated with inflammatory products. If no such condition is present, the wound surfaces can be at once united by deep catgut sutures without any danger. If it is necessary to excise several wedges, it should be done in successive operations.

When excision is impossible on account of the location or the extent of the tumors, or when the small size of the tumor does not appear to justify such severe measures, ignipuncture may be employed. Its action is precisely the same and the same rules apply in its use as in the case of hæmangiomata.

In conclusion mention should be made of the fact noted by Wegner,** that the lymphangiomata may become connected secondarily with blood-vessels. They erode, as it were, the blood-vessels, the thinned walls of which, as the result of trauma or during an exacerbation of inflammation, may burst and the blood is poured into the dilated lymph spaces. Thus arises a kind of mixed tumor which has been called hæmolymphangioma. This is not an extremely rare occurrence.

Macroglossia and Macrocheilia.

Considerable enlargements of the tongue, sometimes true elephantiasis of that organ, are met with occasionally at birth, in childhood, and also at later periods of life. Their causes may be of a quite various nature.

In the great majority the formation of a lymphangioma is the first stage, especially in macroglossia (see above), but other things also occasionally play a part.

FIG. 6.—Macroglossia.

The illustration was taken from an otherwise healthy woman, thirty-six years of age, in whom this condition had persisted unchanged from earliest infancy. The whole organ is much enlarged and deeply lobed. The main furrow corresponds about to the median line, the others run irregularly extending to the edges of the tongue and at the tip even to its under-surface. At the bottom of the furrows the mucous membrane is quite smooth, its elevated parts are abundantly covered with papillæ. The sense of taste is almost completely absent in the more deeply furrowed portions. The condition gave no inconvenience. (From Mikulicz' Atlas, Plate XXXII., Fig. 2.)

A rare but typical form is the purely muscular macroglossia.[37,85] The tongue has double or three times its normal volume, but otherwise with respect to its form and the appearance of the mucous membrane is approximately normal. The enlargement of the tongue is caused almost exclusively by a hyperplasia of the musculature. I have recently seen a case of this kind in a girl two years old. The tongue from birth was so enlarged that it could not be contained in the mouth and projected 2 cm. beyond the teeth. The enlargement of the tongue made the taking of food difficult but otherwise gave no trouble. A wedge-shaped piece, about 3 cm. long, was excised and the hemorrhage was checked by deep sutures applied immediately. Recovery was uneventful.

A peculiar variety of congenital muscular macroglossia is the so-called lobed tongue. The tongue as a whole is enlarged and preserves in a general way its normal configuration. But its surface does not appear smooth, being grooved by numerous more or less symmetrically disposed furrows. The median furrow over the dorsum is the deepest. The whole tongue has a peculiar lobular appearance (see Fig. 6). The prominent portions of the lobes have normal papillæ, but these are absent in the furrows. This condition has no

clinical importance, unless the tongue is greatly enlarged, and requires no treatment.

A moderate degree of macroglossia is not infrequently found in connection with cretinism.[37]

Sometimes such an hypertrophy or hyperplasia of the musculature affects only one-half of the tongue, and is then occasionally associated with hypertrophy of the whole of the corresponding half of the body (Maas[38]).

In other cases, macroglossia and, more rarely, macrocheilia are local manifestations of a general acromegaly. They are easily recognized as such on account of the striking appearance of that disease. This form seldom attains a high degree of development. The enlargement of the tongue in it, as far as is known, is due to increase of all the constituent tissues, especially the musculature.

Considerable permanent enlargements of the tongue may also be produced by other causes, especially chronic inflammatory processes (syphilis) and by tumors. The name macroglossia should not, however, be used for these secondary forms but should be reserved for the primary affection.

The lips also may present considerable and permanent enlargements from various causes. "Scrofulous hypertrophy" has been already mentioned. Cases of macrocheilia from the formation of lymphangiomata have often been reported.[39]

Simple and cavernous angiomata may produce similar enlargements. These may also be caused by hypertrophy of, or the formation of adenomata in, the glands of the lip, which will be treated of below. A not uninteresting disease is one connected with the later stages of syphilitic infection, especially in men; a diffuse swelling of one or both lips, which in every respect resembles that of lymphangioma diffusum. The sometimes very considerable swelling has a hard feel; circumscribed nodules cannot be detected in it. Often, at least for a time, there is a certain redness which is limited to isolated spots, and these reddened parts are usually sensitive to pressure and also, to a slight degree, spontaneously painful. In general the inflammatory symptoms show no great intensity, but occur in exacerbations and relapses as in lymphangioma. The supposition readily suggests itself that the process is a syphilitic affection of the lymph vessels which become obstructed and dilated, or perhaps a chronic hyperplasia of the connective tissue. Judging from the appearance of the disease, the former supposition is the more probable.

Anatomical investigations are necessary to settle the question. Under the use of iodide of potassium the swelling in the last-named cases showed a tendency to diminish, but the patients

could not be kept long enough under observation to obtain definite results.

Cystic Tumors.

In this chapter only those cystic tumors will be discussed which as true cysts are contrasted with the lymph and blood cysts, and which are distinguished from these by possessing a true epithelium, the nature of which varies, however, greatly in the individual cases.

Two groups may be found: the dermoid and dental cysts on the one hand, the gland cysts on the other.

Dermoid cysts are not frequent in the region of the mouth, yet they have been seen often enough to give rise to lively discussions with regard to their pathogenesis as well as to their treatment.[40] They are found almost exclusively on the floor of the mouth, both anteriorly and posteriorly. Exceptionally they have been seen on the under surface of the tongue.

The dermoid cysts are found either exactly upon or close to the median line. The former have been divided by Gérard-Marchant[41] into two chief types: the submental and the sublingual. Marchant designates them according to the place to which they frequently contract adhesions as "ad-hyoidiens" and "ad-géniens."

It is to-day settled beyond the necessity of discussion that these always, as far as can be determined, congenital and, in the mouth, quite heterotopic formations arise from epithelium included when the fissures closed during fœtal development, although this epithelial germ may be long in developing to any considerable size.

To make it more easy to understand about these fœtal fissures and about that which develops from the parts adjacent to them, we may refer briefly to the development of the mouth. For further details the reader is referred especially to His.[42]

The floor of the mouth is formed exclusively from the derivatives of the branchial arches. The lower part of the first branchial arch forms the lower jaw; the second supplies the styloid process, the stylohyoid ligament, and the lesser cornua of the hyoid bone; the third, the great cornua of the hyoid bone. These arches are originally in pairs and unite in the median line. In the hollow of the arch of the lower jaw arises the single centre of development of the anterior part of the tongue. Where the second and third branchial arches come in contact at the median line, there arises on each side a prominence. These two together furnish the paired centres of development of the root of the tongue.

It is possible, therefore, theoretically, that the inclusion of epidermoidal elements may occur, first, immediately before, in, and

behind the median part of the lower jaw (coalescence of the two halves of the lower jaw); secondly, in the same way at the median portion of the hyoid bone (coalescence of the two halves of the second and third branchial arches); thirdly, between the lower jaw and the hyoid bone in the median line or laterally (fusion of the first and second branchial arches); fourthly, between the cornua of the hyoid bone (fusion of the second with the third branchial arch).

Practically, however, it appears that we have to do only with the first three places of closure, and especially with the first and second. The mesal sublingual cysts originate at the first place, the lateral cysts at the third, the submental cysts at the second and third. In accordance with their mode of origin, the close relations or the adhesion of some cysts with the inner side of the lower jaw or the hyoid bone is explained. The details as to the formation of these dermoid cysts do not belong here.

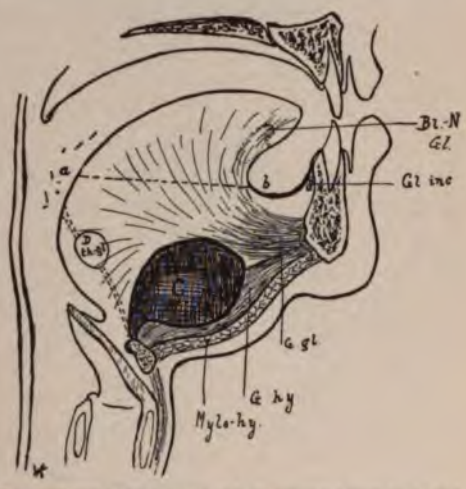

FIG. 7 shows the position of a submental cyst attached to the hyoid bone. Besides this, the course of the ductus thyreoglossus and the position of a cyst originating from its appendages are shown. The position of the Blandin-Nuhn gland at the apex of the tongue (which is not cut in a median section) is marked by a dotted outline. The glandula incisiva is also shown. (G. Marchant.)

Clinically, the appearance of the cysts varies greatly according to their location. The sublingual dermoids are of all the forms confined most exclusively to the cavity of the mouth.

The submental cysts (Fig. 7) cause more distinct deformity in the submental region, a kind of "double chin"; they may be very disfiguring. In general, however, the annoyances are greater in case of the sublingual dermoids (Fig. 8). They arch the region of the frenulum linguæ strongly forwards even at a very early period, and may extend as far as the edges of the lower incisors. Of course, when they have reached such a size they displace the tip of the tongue upwards and backwards, and may finally press it so firmly against the hard palate that only by widely opening the mouth does the patient secure sufficient space for taking food. Speech is then much impeded, and when the growth is very excessive the tongue may be pushed so far backwards that the breathing is also interfered with. But this is very rare.

The submental dermoids may also impede swallowing and respiration, and if they reach a great size, as is sometimes the case, they may even threaten life. In general, growth of the cysts is so very slow that the patients become habituated to their presence, and many notice the cysts only after a long period of development, and sometimes purely by accident. Quite rarely they attract attention soon after birth because the child is unable to nurse.

We have said that the dermoids grow very slowly, but we should add that many stimuli may cause a rapid development; above all, incomplete operations in which the cyst wall is not removed, but is infected. Other traumatisms and sometimes quite unknown causes may produce inflammation. Occasionally the inflammatory process is chronically recurrent as in lymphangioma.

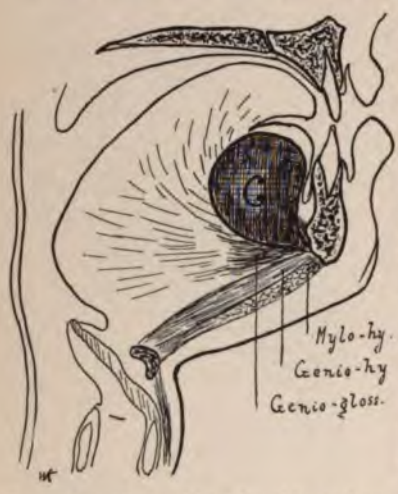

FIG. 8 shows the position of a sublingual cyst with attachments to the spina mentalis. Schematic drawing, a figure from Gérard-Marchant being used.

The appearance of the tumors, as seen in the mouth, varies greatly. The tumor is generally spherical or oval; if it is long and narrow, there is reason to suspect that in the direction of the greatest length a process descends downwards, and that the cyst has grown up out of its original location.

The overlying mucous membrane is always smooth and is movable over the tumor except when it is tightly stretched by it. If the cyst lies directly beneath the mucous membrane, covered by no muscles, its "atheromatous" contents are seen shining yellow through the membrane, or at least the latter presents a certain orange coloration. But this phenomenon is absent if the cyst, as is common, possesses a very thick wall. The color of the contents is never bluish as in glandular cysts.

Upon palpation the dermoid cysts present no fluctuation; their contents are too thick for this; only when a separation of their contents occurs, so that above there is a thinnish fluid and below a thick sediment, may true fluctuation be detected. The tumor is generally soft, seldom tense. Sometimes pressure with the finger leaves a pitting as in œdematous tissues. It is often possible to palpate the tumor bimanually and thus ascertain with greater exactness its mode of attachment and other peculiarities. Firm adhesion to the hyoid

bone, practically of the most importance, is manifested by movements of the tumor during the act of swallowing.

The displacements caused by these cysts have already been mentioned. It is they which make energetic treatment of these, in themselves quite benign tumors most often necessary.

The best *treatment* is unquestionably the complete enucleation of the cyst. Excision of a piece of the wall, puncture (by a *large* trocar) and washing out the contents with the subsequent injection of iodine, and drawing a silk thread through the tumor as a seton, are seldom successful in these generally thick-walled cysts. If it be decided, nevertheless, to try any of these procedures, it should be clearly understood that they all excite inflammatory processes in the vicinity of the cyst and thus may render the extirpation, which may eventually prove necessary, much more difficult.

The operation of enucleation is, as a rule, very easy in cysts which have not been inflamed. The question has been discussed whether it should be undertaken by way of the mouth or through the submental region; but this must be decided according to the nature of the individual case. If the cyst is not very large, and if it lies directly beneath the mucous membrane of the floor of the mouth, the incision will be made through the mouth. If, however, the cyst is covered with thick layers of muscular tissue, and if it projects considerably in the submental region, the incision must be made at the latter point. This incision should also be preferred for very large cysts which are difficult to remove by the way of the mouth, especially if for any reason there are grounds to suppose that there is a firm connection with the hyoid bone.

After extirpation from without, the wound may be closed at once by sutures. When the enucleation has been made through the mouth, it has been closed by immediate suture (after rubbing iodoform into it) by but few operators. In general it is preferable to pack the cavity with iodoform gauze and allow healing to take place by granulation.

If it is necessary to come to the assistance of the new-born child on account of interference with suckling, it is better to content one's self with puncture, repeated if necessary, deferring the operation of removal until a later time.

Dental cysts occur frequently in the mouth. But they are generally situated upon the jaw and will therefore be considered in connection with the tumors of that part.

Gland Cysts.

Gland cysts, *i.e.*, those the walls of which are covered with glandular epithelium (so far as this has not been lost secondarily), and the contents of which are a colloid or mucous fluid, are met with frequently in the mouth. Three chief groups can be distinguished: superficial cysts of the mucous membrane, occurring in all parts of the oral mucous membrane which contain glands; cysts of the floor of the mouth, going under the general name of ranula; cysts of the base of the tongue.

Superficial cysts of the mucous membrane are of frequent occurrence, although not much is said about them in the books. But Roser[43] remarks that the cysts of the mucous glands of the cheek, if of large size, may occasion difficulties in diagnosis. W. G. Spencer[44] and Chaslin[45] have described the mode of formation of these cysts. An illustration and description of such a cyst of the lip may be found also in Mikulicz' Atlas, Plate I., Fig. 3.

These cysts rarely come under the observation of the physician. The patients generally notice only by accident the slowly growing tumors, which seldom reach any considerable size, and in many cases they try to effect a cure themselves by puncturing the cyst. That the formation contains fluid is, on account of its superficial location, clear, even to the layman.

Inflammatory processes are not only frequently the primary cause, since they lead to a blocking up of a chief or one of the smaller excretory ducts, but they may also be the cause of increased disturbances from the tumor, through infection of the contents of the cyst or of the cyst wall. The latter may then be destroyed by suppuration and cast off. It may then easily happen that the pus-containing cavity, the abscess, alone comes under observation, its origin from a cyst being no longer recognizable.

The most frequent location for these cysts is naturally those portions of the mouth where the glands, from which they originate, are most abundant and most highly developed. These are the inner side of the lips in the vicinity of their vermilion border, the mucous membrane of the cheeks in its entire extent, the dorsum of the tongue especially in the region of the circumvallate papillæ, and the under surface of the apex of the tongue.

The cysts are generally covered only by a very thin, transparent membrane, through which appears the bluish color of the fluid. Sometimes processes of the cyst, or smaller cysts adjacent to the chief cyst, penetrate more deeply, for example, among the fibres of the buccinator muscle (Roser).

These little tumors cause annoyance as a rule only when they are inflamed, but the patients, when they have noticed the abnormality, often wish it removed. This is generally accomplished without difficulty on account of the superficial seat of the tumors, by enucleation. Cocaine makes the task easier.

Cysts of the floor of the mouth (to which the name *ranula* adheres, almost like a curse) have been the subject of countless articles. Since the publication of the work of von Recklinghausen[46] and earlier

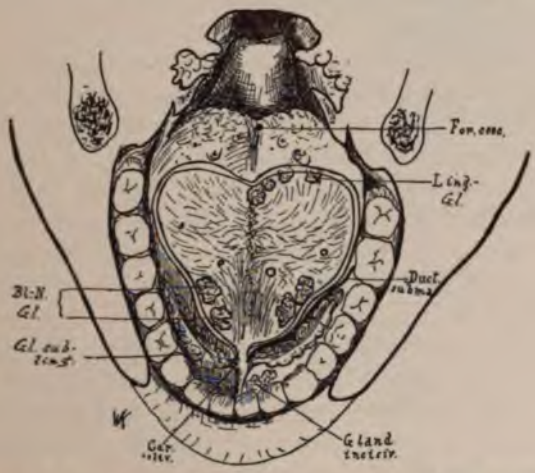

FIG. 9, drawn from a dissection, with the aid of Merkel's Fig. 208 ("Handbuch der topographischen Anatomie"), and somewhat schematic, shows the position of the structures which are under consideration. The tongue has been cut through almost horizontally in the direction *a–b* (Fig. 7). The cheek and jaw were cut horizontally at the level of the angle of the mouth. On the left the mucous membrane of the floor of the mouth has been dissected off. The individual structures are easily to be recognized by the affixed names or lettering.

that of Pauli[47] an enormous literature upon the subject has accumulated and the contest is not yet at an end. Albert's sarcastic designation of this literature as "*noli me legere*" has a certain justification.

For practical needs the following remarks should suffice and these may be made more readily intelligible by reference to Figs. 7 and 9.

Such a tumor may arise; first, by inclusion of epithelial elements during closure of the branchial fissures; secondly, by closure of the excretory ducts and of the acini of various glands.

Strohmeyer's view that they are true hygromata need not be discussed, now that it is known that the bursæ mucosæ, manifestly discovered *ad hoc* by Fleischmann, do not exist.[48] As little will it now be supposed that they are due to a mucous extravasation from the bursting of the submaxillary duct.

With reference to the formation of these cysts by inclusion of epi-

thelial elements, all the possibilities enumerated above for the dermoid cysts may occur, but of course parts must have been included from the intestinal side of the branchial pouches. We need merely refer to what was said above as to the situation of such displaced epithelial germs. Besides these, such inclusion might occur along the lines of union of the anterior centre of development of the tongue, the tuberculum impar (His), with the two posterior centres. In this last mode of origination a quite closely defined location of the cyst must be assumed, viz., between the foramen cæcum or the circumvallate papillæ, *i.e.*, the triangle of the tongue on one side, and the hyoid bone on the other; consequently deep in the tissues of the tongue itself.

For the second mode of formation, a large number of glands are to be considered, the situation of which are to be seen in Figs. 7 and 9: *a*, the sublingual gland with its excretory ducts; *b*, the submaxillary duct; *c*, the often very numerous small mucous glands which surround the terminal portion of that duct; *d*, the group of glands described by Suzanne and designated by Merkel[48] glandula incisiva. It lies at the neck of the median (sometimes the lateral) incisor; one can often detect its presence in the living subject, as a slight thickening of the mucous membrane at that point; *e*, the Blandin-Nuhn gland in the apex of the tongue. This may extend as far as the point of union of the mucous membrane of the floor of the mouth with that of the tongue; *f*, the irregularly, or apparently irregularly scattered glands of the mucous membrane of the mouth.

From which of these in the individual case the cyst has arisen, will generally remain uncertain in the living subject even after operation. By keeping clearly in mind the relative position of the different parts which are to be considered, one can often make at least a reasonable supposition as to the mode of origin. It is much to be desired that some new anatomical investigations of ranulæ be made upon the cadaver by one without any prepossession for any particular mode of origin. The often very considerable displacement of the parts makes a clear understanding of the matter difficult in the living subject. But the fact cannot be too much emphasized that the histological examination of the cyst wall or of its epithelium allows by itself of no far-reaching conclusions. The epithelium may be transformed in the most varied ways here[48] as in other cystic formations.[49]

In theory, we may regard every one of the above enumerated modes of origin as possible. The question is, which of them can be really proved? This has as yet been done with certainty only in the case of cysts which originate from the Blandin-Nuhn gland (von Recklinghausen.[48])

The location of these ranula cysts corresponds to their place of

origin, but a certain displacement may occur gradually, if the growth is of considerable size. The cysts are found in the median line only exceptionally. In this case an inclusion may always be assumed. As a rule, they are situated near to the median line. In either case, after they have attained any considerable dimensions, they lie usually immediately beneath the mucous membrane. So it almost always happens that the physician, when first consulted, finds between the tongue and the lower jaw a roundish, translucent tumor of a bluish-red or bluish-white color, which displaces the organs situated upon the floor of the mouth more or less in one or the other direction. If the tumor is mesally situated, the frenulum linguæ is stretched over it. The lateral swelling which corresponds to the sublingual gland generally runs over the mesal as well as the lateral cysts (Figs. 9, 10).

Fig. 10.—Ranula.

This cut shows a typical ranula from a girl eighteen years of age, which had been noticed for a year and had been in the mean time evacuated spontaneously several times. The tumor lies symmetrically on both sides of the frenum, presses the tip of the tongue upwards and backwards, and is bluish and translucent. A seton was inserted, after which the cyst slowly collapsed, with the evacuation of a clear, stringy, honey-like fluid. (From Mikulicz' Atlas, Plate XXVIII., Fig. 2.)

Conclusions may sometimes be drawn as to the mode of origin by the direction in which the parts are displaced.

The cyst walls are generally quite tense. But fluctuation is usually easily detected, best by bimanual palpation (one finger under the chin and one upon the oral surface of the cyst). The diagnosis can therefore hardly be mistaken. Only very deeply situated cysts could, for lack of sufficient means of discrimination, be confounded with dermoid cysts.

The symptoms which a ranula produces clinically depend entirely upon its size. The tumor grows generally very slowly, but may have attained a considerable size even in fœtal life, and still more frequently soon after birth. It may often remain unnoticed or at least unregarded until the time of puberty, but the physician is rarely consulted about it in later life.

Precisely like the sublingual dermoids the ranula presses the apex of the tongue upwards and backwards against the hard palate. Thereby the suckling of infants and even the taking of food by adults

is rendered difficult. There are also impediments of the speech; sometimes the voice has a peculiar metallic tone. In other cases the speech becomes constantly indistinct on account of the difficulty of moving the tongue. When greatly enlarged, the cyst also presses the root of the tongue backwards and may then cause serious difficulty of breathing.

In consequence of these annoyances many ranulæ are operated upon; but formerly the operations were not very successful. Evacuation of the cyst, on account of its superficial location, is generally very easy, but, as a rule, it just as readily refills.

There are many methods of breaking ranulæ of which we will mention only puncture, with or without the subsequent injection of tincture of iodine, and incision of the cyst, with or without subsequent cauterization of the sac with nitrate of silver or other caustics. In some isolated cases these methods are successful, and in fact the cyst may even heal spontaneously, bursting and becoming obliterated. In the majority of cases this certainly does not happen and therefore recourse is had to more energetic means of destruction—chloride of zinc and the thermo-cautery. The former has not approved itself on account of the inflammation which is apt to follow its use, and which is not without danger in the floor of the mouth. The thermo-cautery is less dangerous, but is not less liable to be followed by recurrence.

The best procedure for all simple cases is the insertion of a seton, a stout silk thread, which is tied over the tumor. This method was especially recommended by Billroth in recent times and made widely known by his school. The cyst then empties itself slowly. The efficacy of the method depends on the fact chiefly that the thread remains *in situ* six to eight days (until it has cut through the portion of tissue ligated by it), and for this length of time keeps open a communication between the sac of the cyst and the mouth. This time suffices, as a rule, to effect at one place at least a direct union of the epithelium of the cyst and that of the mouth. If this has occurred a refilling of the cyst is prevented. Occasionally a relapse occurs, in which case a repetition of the procedure is generally successful. If it is not, excision of a portion of the sac may be considered, but this presents no material advantage over the seton. If this first-mentioned method fails, nothing remains but the complete extirpation of the sac. From this operation a permanent cure may be expected, but it has its difficulties. The wall of the cyst, in the majority of cases, is very thin and hard to separate from the surrounding tissues, so that the cyst is easily cut, and when it is empty the enucleation becomes doubly difficult. When a tolerably abundant hemorrhage results, for the sac can hardly ever be enucleated without the use of

the knife, it often happens that the intended enucleation must be given up, and, instead, a simple excision of a portion of the cyst wall is performed.

The operation cannot well be performed under general and deep anæsthesia on account of the hemorrhage unless it be done with the head dependent. But one can do very well with the aid of cocaine. Félizet[50] proposes in addition to the superficial applications to the mucous membrane to inject cocaine solution at several points in the vicinity of the cyst and then at the same place to inject a larger quantity of sterilized boracic-acid solution. In this way an artificial œdema is created about the cyst while this latter remains untouched and distinctly discernible. Félizet incises the cyst as soon as he has freed it superficially, and packs it firmly with a wad of gauze or cotton. He then has a solid tumor to enucleate and the task is consequently easier. If a very weak solution of sodium chloride and cocaine (0.01 per cent.) is used for infiltration-anæsthesia, after Schleich's method, the same end is attained in a simpler way. The intentional evacuation of the cyst and the extirpation of its wall, while it is made tense by the finger, as in the removal of dermoid cysts and other cystic growths, has long been the practice of many surgeons. The wound cavity is best packed with a tampon of iodoform gauze, which should remain until it falls out spontaneously.

For the sake of completeness the cases of so-called *acute ranula* should be mentioned here. These are cases of acute inflammation of the excretory duct of one or both submaxillary or sublingual glands. These excretory ducts are blocked by the swelling, by pus, or, as in the most recently reported case,[51] by fibrinous deposits, and the glands in question are swollen and painful. Pressure upon the glands evacuates saliva, sometimes mixed with pus from the opening of the duct. But a genuine cystic dilatation of the excretory duct seems not to have been observed. The disease presents most resemblance to pyorrhœa salivalis (see p. 13).

Cysts at the base of the tongue are very interesting formations from a pathological point of view, but are clinically of almost no importance. They seldom reach a considerable size, the largest are described as of the size of a hazelnut or a cherry. With regard to the details of the pathology of these formations reference may be made to the most recent and thorough investigation by M. B. Schmidt.[52]

The places of origin of these cysts are the superficial mucous glands, which are thickly clustered at the base of the tongue, the large "serous glands" (von Ebner) which open about the circumvallate papillæ, and finally the appendages of the lingual duct, the upper part of the ductus thyreoglossus. This duct, as is well known, forms

in the embryo the canal which runs from the foramen cæcum to the hyoid bone. The portion of this canal which persists varies greatly in length. About its upper portion, as Schmidt has demonstrated, are aggregated a varying but often very great number of small mucous glands, and from its lower end there proceed a series of wide and sinuous ampullæ which branch repeatedly and which may extend posteriorly beneath the valleculæ and forwards and downwards to the vicinity of the body of the hyoid bone.

Cysts may originate from all these parts of the ductus thyreoglossus. Schmidt has collected examples of all these forms. The position of the cyst accordingly varies. Some are superficial and elevated beneath the mucous membrane in the vicinity of the foramen cæcum, or more posteriorly beneath the valleculæ in or near the median line. Others lie deeply embedded in the parenchyma of the tongue between the fibres of the genioglossus muscle, extending perhaps to the hyoid bone. All these cysts are distinguished by the fact that some at least of the cells which line them are ciliated. Schmidt thinks that the formation of almost all of the cysts is due to an obliteration of the excretory duct of one of such glandular appendages.

Clinically, as has already been remarked, these cysts, which as a rule remain of small size, do not cause any annoyance. They are consequently all discovered by accident, generally at autopsies. The operative removal of the tumors, if it should ever become necessary, would be quite difficult on account of the inacessibility of this region. Yet if it should be necessary to operate, the insertion of a seton, the method already described for ranula, would be applicable to these so superficially situated cysts. In this situation it would of course be necessary to use the laryngeal mirror. Kirstein's direct laryngoscopy ("autoscopy") under anæsthesia, with head dependent, should be employed here.

In connection with the cystic tumors of the ductus thyreoglossus, the "accessory thyroid glands" of the base of the tongue should be mentioned. The ductus thyreoglossus is a remnant of the deep infolding of epithelium from which is formed the unpaired upper centre of development of the thyroid gland. These accessory thyroids have also been described by Schmidt.[52] To the cases cited by him is only to be added that of Bernays.[53] These tumor-like formations are very rare, and there are as yet but eleven cases reported in the literature. They appear as smooth or slightly nodulated tumors of a soft and somewhat elastic consistence and covered by normal mucous membrane. They are sometimes flat, sometimes elevated, even hemispherical. Their position is generally strictly in the median plane be-

tween the hyoid bone, the epiglottis, and the foramen cæcum. They form a transition to the accessory thyroid glands which are situated in and near the hyoid bone[54] in the further course of the ductus thyreoglossus.

These accessory thyroid glands have been observed in female patients only, and generally at about the time of puberty. They may give trouble by interference with deglutition and, to a certain extent, with speech. But they grow slowly and a partial extirpation, which can frequently be performed by the way of the mouth, generally suffices to remove the disturbances which they cause. They show but a slight tendency to recur (Butlin[55]). Larger nodules situated in the muscles nearer the hyoid bone must, however, be removed, if operation is required, from without, which is not especially difficult (Bernays[53]). It should be noticed that they are often found to be multiple.

With respect to the diagnosis, confusion with a cyst at the base of the tongue, which can occur only in the case of soft tumors, may prove serious. The attempt to evacuate such a tumor by incision may easily lead to a fatal hemorrhage.

Papilloma.

M. Kahn[56] has made recently a comprehensive study of the papillomata which occur on the oral mucous membrane. Besides a relatively small number of cases from the literature he reports eighty-three observations of his own.

As appears from his article, papillomata are a very frequent form of tumor in the mouth; but the patient very rarely seeks advice on account of them. They are generally discovered only while the mouth is being carefully examined for other conditions. Kahn enumerates fifty-eight male and only thirteen female patients—the same preponderance of the male sex that is observed in so many diseases of the mouth, and which there is reason to ascribe to the misuse of alcohol and tobacco. Papillomata occur very frequently coincidently with other affections of the mouth, pharynx, and nose. Of seventy-one patients only twenty-one were free from such diseases. The majority of such affections are of the nature of chronic inflammation. One is therefore very naturally inclined to regard papillomata as the result of conditions of chronic irritation.

The favorite seat of papillomata is the mucous membrane of the uvula, and, next, that of the palatine arches; more rarely the tongue and the tonsils are involved. On the tongue the base is the most frequently affected locality. They are often multiple, sometimes very numerous.

The structure of papillomata of the mouth differs in no way from that of other analogous tumors. The chief mass of the tumor is formed by thick layers of pavement epithelium which penetrate deeply into the relatively scanty connective tissue of the tumor. These tumors are generally provided with a typical, often a very long pedicle, but papillomata occur which have short pedicles or even none at all.

The greater number of papillomata are small, from the size of the head of a pin to that of a pea, very rarely they attain the size of a hazelnut (Kahn's Case, No. 44), and this is probably the reason why they cause their possessors so little inconvenience.

FIG. 11.—Papilloma of the Uvula.

This tumor occurred in a youth eighteen years of age. It was found accidentally during an examination for adenoid vegetations and was cut off with scissors. The microscopical examination confirmed the diagnosis. (From Mikulicz' Atlas, Plate XXVII., Fig. 4.)

Papillomata of the base of the tongue give the most annoyance, confined here to paræsthesias. In a case of Seifert's (see Kahn) papilloma of the base of the tongue even provoked attacks of dyspnœa of a nervous character, and in a case of Herzfelder attacks of hystero-epilepsy. But these are exceptions.

The diagnosis of papillomata is not difficult; their surface, thickly dotted with fine and coarse elevations, can hardly be mistaken for anything else. An error may occur, however, when the spaces between the papillæ are filled with mucus, and we should therefore always wipe the surface of any tumor we are examining. A confusion of these growths with isolated adenoid hypertrophies is of little practical moment. In cases of frequently recurring papillomata, we should remember that carcinomata may in the beginning resemble such warts very closely, especially in elderly persons. We may here refer to a case reported by W. K. Simpson,[56a] of New York. After the extirpation of multiple papillomata of the uvula, there occurred a very malignant sarcoma of the soft palate which speedily caused death. Simpson was of the opinion that here the originally benign papilloma was transformed directly into a malignant growth, a sarcoma.

If a papilloma causes any inconvenience, removal by the knife or scissors is the simplest and best treatment. Scraping or cauterization is more disagreeable for both physician and patient.

Albert [57] includes among papillomata two other conditions which

we, with Kahn, are not willing to class with them. These are: first, "the painful papilloma of the papilla foliata," *i.e.*, an hypertrophy of this structure, which is usually not well developed in man. Violent neuralgic pains, especially during eating, characterize this disease. The papilla foliata is enlarged and reddened and presents erosions. The disease is certainly not common. It cannot be regarded as a papilloma in the pathologico-anatomical sense of the word. The same is true of the second condition, hypertrophy of the papillæ fungiformes (Albert's "flat papillomata"), which are associated with no such symptoms.

The so-called "horns" of the skin (cornua cutanea) are to be considered as a peculiar form of papillomata with a strong tendency to cornification. These occur, though rarely, upon the lips and always proceed from their external surface. A typical case of the kind has been operated on at the Breslau Surgical Clinic.

The deformity and interference with function caused by a cutaneous horn in this location will probably always induce the patient to seek an operation. Local recurrence occurs frequently after removal, and since this tumor is relatively more common in elderly people, the suspicion that the new growth is malignant is not unnatural.

Adenomata and Tumors Resembling Them.

Until recently certain benign solid tumors, which are especially frequent in the salivary glands and on the mucous membrane of the palate, have been regarded as true adenomata or as mixed forms between adenoma and tumors of the connective tissue of various kinds.

But it has been demonstrated of late that some of these tumors are of endothelial or occasionally of epithelial origin. R. Volkmann even assumes this origin for a class of tumors which by other investigators would certainly be regarded as true adenomata.

Since this question has not yet been decided and cannot be decided here, we will separate this clinically well-marked group from the adenomata in our discussion, and first consider some other forms of tumors.

Unmixed hypertrophies of the glands of the lips, which only in part possess distinctly the character of tumors, have been observed in a few cases.[*] The mucous glands on the inner side of the much enlarged lips are felt distinctly as a chain of nodules, the size of peas. In E. Fränkel's case a viscid secretion dropped continuously from their openings.

The affection is probably essentially identical with the "double

lip" described by many authors, an hypertrophy of the submucous tissues adjoining the vermilion border of the lip. Thereby a second lip, as it were, projecting from behind the true lip, is formed, which causes great deformity and may seriously impede the speech. Its operative removal is therefore often necessary, and is easily effected by longitudinal incisions which remove a wedge-shaped portion of the labial tissues.

Adenoma of the sublingual gland, which M. Zeissl [59] has described, appears more distinctly as a tumor. In the case reported the tumor was fully as large as a hen's egg, rather easily enucleated, and its structure was clearly a reproduction of that of the salivary glands.

R. Volkmann[60] describes a benign epithelioma of the cheek proceeding from the cutaneous surface, which is perhaps to be explained as an adenoma of a sebaceous gland, but which is very similar to the doubtful tumors which are to be described below. Many tumors described as adenomata may be regarded as analogous growths proceeding from the mucous membrane.

Endotheliomata.—The great majority, however, of tumors described as adenomata in the literature, especially by the French, should, according to the investigations of R. Volkmann[60] and according also to many earlier authorities, be regarded as derived from the endothelium.

The structure of these endotheliomata, which is quite analogous to that of the cylindromata, myxofibromata, myxosarcomata, enchondromata, and chondroadenomata (all these names are in use for this group of tumors) of the salivary glands is very complicated.

There is always found a network of bands or nests of cells which are separated from each other by interstitial tissue poor in cells. The bands of cells exhibit the most various formations: flat, spindle-shaped, polygonal cells, and some which resemble epithelium. When these latter forms lie crowded together in a wide space one would hardly have any doubt as to the glandular nature of that space. Notwithstanding this, and although such forms have been pretty universally regarded as adenomata, Volkmann demonstrates their origin from endothelial elements and thus classes them with tumors of the connective tissue. The resemblance to glandular structures is in numerous cases increased by the fact that the cells are capable of a kind of secretion—they deposit within the bands colloid or mucoid hyaline products, which appear like the secretion in glandular vesicles.

The interstitial connective tissue is as a whole poor in cells but fairly well developed. It undergoes numerous transformations, to which the variegated appearance of these tumors is chiefly due. Sometimes we find fibrous connective tissue, sometimes mucous tissue, sometimes cartilage; even the formation of true bone occasion-

ally occurs. This is not the place, however, to enter into the very numerous histological details. The reader is referred for these especially to Volkmann's very thorough presentation of the subject. Whether Volkmann is right in including the greater part of the tumors of the salivary glands, known as chondromata, adenomata, myxosarcomata, etc., among the endothelial tumors also cannot be discussed here. This much is certain, that tumors quite analogous to those of the large salivary glands occur rather frequently in the small glands of the oral mucous membrane. They present the same histological and clinical characteristics and have had as manifold interpretations as those of the salivary glands.

Above all, such so-called adenomata are found proceeding from the glands which lie thickly beneath the mucous membrane of the hard and soft palate, especially numerous at the junction of these two structures. The tumors are situated quite constantly laterally from the median line, thus corresponding exactly with the position of the glands. They form generally flat, more rarely hemispherical, projecting, smooth, or, at most, slightly nodular swellings, which are generally covered by smooth and intact mucous membrane. Quite rarely the mucous membrane is stripped of its epithelium or ulcerated. The form of the tumor is round or oval. It generally has a firm feel, and may even be as hard as cartilage, according to the nature of the intervening tissues. Isolated spots where considerable cysts have formed may also give distinct fluctuation. The mucous membrane over the tumor is generally movable, at least as long as the tumor has not attained great size. The tumor likewise is movable over the underlying tissues. It can, as a rule, be determined by palpation that the tumor is sharply circumscribed.

The ulcerations upon the surface, which only rarely occur, are always quite shallow and are manifestly produced by mechanical injuries of the thinned and badly nourished mucous membrane.

The resulting discomfort is also purely mechanical in its origin, caused solely by the size of the tumor. Pain is always absent. Interference with speaking and swallowing is the chief source of annoyance.

The rate of growth of these tumors is almost as slow as that of fibromata and lipomata. They often exist for years before they cause any trouble. But after attaining a certain size they sometimes quite suddenly acquire a malignant character. They break through their capsules and may then be equivalent to the most malignant sarcomata. While the neighboring lymphatic glands are never enlarged as long as the tumor retains its primary benign character, they swell quickly on the occurrence of sarcomatous degeneration, just as they

do in primary sarcoma, a fact which may be of great importance in the diagnosis.

In general the diagnosis of these tumors is easy. They are most readily confounded with fibromata and lipomata, which, on account of the absolutely identical indications for treatment, is a matter of comparative indifference.

If they have become very large a malignant sarcoma may be suspected. The mistake might be in so far injurious to the patient that it would cause the difficulties of operative removal to be overestimated and extensive preliminary incisions might be made in order to gain free access to the tumor. The sharp demarcation of the tumor, especially with respect to the overlying mucous membrane, ought, however, to prevent such errors in most cases.

The extirpation of the so-called adenomata is generally very easy. After incision of the mucous membrane they can usually be turned out without further use of the knife. As a rule they are but slightly vascular. Their tendency to local recurrence is rather great, but the prognosis is not rendered worse thereby. General metastases and indiscriminate involvement of neighboring organs are almost never observed.

But, as has already been mentioned, after existing for a considerable time they may suddenly assume a more malignant character and then resemble the worst sarcomata. Keeping this in mind, it is necessary to give a cautious prognosis and especially to endeavor to remove them by an early operation.

Analogous tumors occur in other parts of the mouth which are rich in glands, although with much less frequency than upon the palate. R. Volkmann mentions such tumors, resembling adenomata and in part containing cartilage, of the upper lip and the cheek. Similar cases on the tongue also are mentioned by Voyer[61] and Monod (Larabric[62]). A cylindroma of the lateral portions of the floor of the mouth, which subsequently recurred locally in the tongue, has been seen at the Breslau Surgical Clinic.

And finally we may mention a very singular symmetrical affection of the glands of the palate and the sublingual glands, which acquires its peculiar character chiefly from the similar and contemporaneous disease of the lacrymal and large salivary glands. It has been especially described by one of the present writers,[63] and is evidently very rare. The strange physiognomy of the patient produced by the considerable enlargement of the lacrymal and salivary glands so attracts the attention that the disease of the palate and sublingual glands may sometimes be overlooked, although it is striking enough. On both sides of the deeply grooved median line of the palate lie two

thick, somewhat pear-shaped swellings, an extreme thickening of the whole mass of glands. The sublinguals likewise present a thick swelling on the floor of the mouth on both sides of the tongue. The swellings in the submaxillary and lacrymal glands which we examined were due essentially to the deposition of a uniform dense infiltration of lymphoid tissue between the almost unaltered acini of the gland. We may assume the presence of similar changes in the palatal glands.

The etiology and the general pathological relations of this strange disease are by no means well determined. The reader is referred to the original article[93] for the literature, the significance, and the details of the disease.

MALIGNANT TUMORS OF THE SOFT PARTS.

Sarcoma.

Tumors of a malignant or suspicious character belonging to the connective-tissue series are found very frequently on the jaws, but seldom proceed from the soft tissues of the mouth.

In the majority of recorded cases of sarcoma the tongue was the part affected. More than twenty such cases have been reported.[94] Among these is a case of congenital origin (Jacobi), but this, as in the case of sarcomata in general, is a rare exception. They are observed at all ages and in both sexes with about the same frequency. They may spring from any part of the tongue, but the larger number arise from the root, although such tumors have been seen on the tip, on the edges, and in one lateral half of the tongue.

Their external appearance varies greatly. Two tumors depicted in Mikulicz' Atlas had thick pedicles (Plate XXXV., Fig. 2), the remainder were embedded in the parenchyma of the tongue, projecting above its surface to a varying degree as flat-topped or hemispherical elevations (Plate XXXVII., Fig. 3). The mucous membrane which covers them is only rarely ulcerated. When this is the case the erosion seems generally to be shallow and to be due to mechanical injuries, from mastication and the like; only after a long duration does the ulceration penetrate more deeply into the tumor itself. Sometimes the overlying mucous membrane is thickened and presents papillary excrescences, also probably caused by mechanical irritation. The size of the tumor varies from that of a hazelnut to that of one removed by Poncet which weighed 400 gm. (14 ounces).

With respect to structure, the majority are small round-celled sarcomata, some have larger cells and others spindle-shaped cells—

fibrosarcomata. A case described by Santesson approached in its structure the endotheliomata already described.

The clinical symptoms depend, at least in great measure, upon the extent of the tumor. But even small tumors may give annoyance on account of their situation, which renders them liable to be caught between the teeth. Otherwise there are no disturbances worth mentioning produced by the tumor until it becomes large enough to interfere with speech and deglutition.

The pains which are often recorded, but which are generally absent in the thoroughly benign tumors, are not unimportant from a clinical standpoint. They often shoot into the ear of the affected side and may become very severe.

The consistence of the tumors varies greatly. They may be so soft that there is pseudo-fluctuation; on the other hand, the fibrosarcomata may be very hard.

The rate of growth of the tumors may be very slow to a certain point when a greater vigor of growth is manifested, occasionally quite suddenly. This sometimes coincides with the appearance of an ulceration, sometimes also with the development of more severe pains. In this case, of course, the difficulties of a purely mechanical nature also increase rapidly.

The demarcation of the tumor from the surrounding tissues is sometimes so sharp that enucleation is not difficult. In other cases, however, such an operation is manifestly impossible, and it is necessary to remove the tumor by excision of the affected portion or by amputation of the entire tongue.

Metastases occur in only a relatively small number of cases. The lymphatic glands especially are rarely diseased; the submaxillary more frequently than those of the neck. Extensive tumors of the glands are observed almost exclusively in tumors which have recurred after operation. Metastases in other organs are seldom seen.

To a certain extent, therefore, sarcomata of the tongue are to be regarded as benign. They recur locally very frequently after operation, but repeated operations upon the recurring growths have led to an apparent cure.

The diagnosis of these tumors is sometimes easy. If the tumor is large and firm, not very sharply circumscribed, or merging diffusely into the surrounding tissues; if it causes severe pain; if the overlying mucous membrane is not ulcerated, and if the adjacent lymphatic glands are not affected, the other conditions with which it might be confounded are only deep-seated tuberculous or syphilitic nodes. From these two formations a distinction is often well-nigh impossible, but in tuberculomata the neighboring lymphatic glands

are often diseased; syphilomata are recognizable sometimes by their multiple growth.

In other cases excision of a bit of tissue for microscopical examination may lead to a correct diagnosis. But Scheier calls attention to the fact that discrimination between syphilitic and sarcomatous tumors by means of the microscope is often very difficult. Trial of antisyphilitic treatment is certainly advisable in many cases. But, as will be repeated with even greater emphasis when we come to speak of carcinoma, the matter must not be overdone, the favorable moment for operation must not be lost by long-continued antisyphilitic treatment.

If the ulceration is extensive a distinction from carcinoma is generally impossible, but is practically of no importance. Smaller tumors, on the other hand, may easily be confounded with fibromata and the like, especially if they are distinctly circumscribed. But this error is also of no great importance if an operation is performed.

The absence of inflammatory manifestations and of a sharp-edged carious tooth will prevent confusion, on the one hand, with deep glossitic, and on the other, with decubital infiltrations.

The treatment consists only in the operative removal of the tumor. If the diagnosis of sarcoma is quite certain, it is best to follow the same rules as in cancer of the tongue. But if this is not the case, and if the tumor in question may be benign, it is advisable to attempt its enucleation. When there is suspicion of gumma a rational antisyphilitic treatment is in place, which should not, however, be too long-continued unless there is manifest improvement in the first two weeks. In inoperable cases the administration of arsenic may cause at least a temporary benefit (Kundrat[65]).

Sarcomata in other parts of the mouth are rarer. On the palate, especially at the junction between the hard and soft palate, and in that vicinity, tumors which are regarded as sarcomata are not uncommon, but they for the most part manifestly belong to the above-described type of endothelial mixed tumors.

Besides these forms other tumors are contained in R. Volkmann's tabulated series of cases which are called round-celled and spindle-celled sarcomata. They are not especially different from the malignant sarcomata of other parts of the body, generally grow rapidly, and invade indiscriminately the surrounding tissues. Their removal often requires a more or less extensive resection of the upper jaw. The case of W. K. Simpson has already been mentioned in the section on papillomata (p. 54).

Some cases of melanosarcoma have also been reported. To those collected by Volkmann, one observed by himself, is to be added also

that depicted and described in Mikulicz' Atlas, Plate XIV., Fig. 2. In another case we have observed a peculiar and surprisingly slow development of a melanosarcoma on the hard palate and the gum. For almost two years the singular sharply limited dark grayish-brown discoloration of the mucous membrane which is peculiar to these tumors lasted without any demonstrable swelling or thickening. Then wart-like prominences appeared, and finally a diffuse and rapidly growing tumor.

As a rarity may be mentioned the occurrence in two cases of tumors resembling sarcoma in which there were cells analogous to those of embryonal striped muscular fibre. In E. Wagner's[66] case there was a sharply circumscribed nodule, of the size of a small pea, in the submucosa of the posterior surface of the soft palate. In the case depicted and described in Mikulicz' Atlas, Plate XXIV., Fig. 2, an extremely malignant tumor, which had evidently grown rapidly, lay between the layers of the soft palate extending along the side wall of the pharynx to the base of the skull. These tumors could only have been discovered by microscopical examination.

With regard to the other parts of the mouth, two myxosarcomata of the cheek have been observed, both of which, however, may possibly belong to the above-described group of endotheliomata (Zahn, Hiepp[67]).

A sarcoma of the size of an apple and in a state of fatty degeneration was enucleated by Hortelrup[68] from the cheek of a lady, forty-six years of age. A fibrosarcoma of the cheek, the size of a walnut and easily enucleated, situated 2 cm. behind the commissure of the lips, was observed at the Breslau Surgical Clinic. The annoyances caused by the growth were similar to those from other tumors of the cheek, and the case presented nothing clinically which is worthy of remark.

An observation of Röttger[69] may also be mentioned. In the case of a man, forty-eight years of age, there was found a node over which the skin was movable, and which led rapidly to a diffuse hard thickening of the entire upper lip. An excised piece was examined microscopically and was decided to be a "lymphoma." Soon afterwards a tumor of the size of a hazelnut developed in the right cheek. Both disappeared spontaneously after some time, fever and an erysipelatous reddening of the face being present. There was no recurrence at the end of a year. The blood and the spleen were normal. Only one swollen (supraclavicular) lymphatic gland was detected. The case is not quite clear. Whether they were pseudoleukæmic tumors, as Röthger supposes, is doubtful.

Carcinoma

The structures of the mouth are unfortunately among the parts most subject to carcinoma. The lips and the tongue especially are very frequently attacked by cancer. A series of facts are to be considered which apply equally to all the special localizations of cancer in the mouth and which are much discussed in the inquiries as to the origin of carcinoma. We will speak of these at the outset before proceeding to describe the individual forms of the disease. Certain statistical data, first of all, claim attention. Here we cannot bring forward extensive statistics of our own but must take the figures for the most part from the earlier investigations.

What excites surprise is the fact that the male sex so largely preponderates in cases of cancer of the mouth. Thus it has been calculated that only 7.4 per cent. of all patients (31 out of 418 cases) suffering from cancer of the lip are women. In Wölfler's statistics of cancer of the tongue from Billroth's clinic there were out of 115 patients, only 4 women, *i.e.*, 3.4 per cent. But out of 92 cases collected by English and German statisticians 30 were women, that is, 32 per cent.

In spite of the great variations in these figures, the fact of the preponderance of men among subjects of this disease will have forced itself upon the attention of every observer, and it demands an explanation. In general, the same law obtains for the majority of diseases of the mouth. It has been sought, very naturally, to explain this fact by supposing that the chief cause is the misuse of alcohol and tobacco, to which, with us, the male sex is particularly addicted. But von Winiwarter has opposed to this apparently obvious explanation the statement that Oriental women who are accustomed to the use of tobacco are as little subject to cancer of the tongue as the women of the Occident. And Wölfler has further shown that English women who do not smoke, as a rule, are relatively much more frequently afflicted with cancer of the tongue than the women of Vienna who are equally or less abstinent.

The influence of smoking, at least, is consequently not quite clear, and with regard not only to cancer of the tongue but also to that of the lips there are many facts which are opposed to the assignment of such importance to this certainly often overestimated etiological factor. Besides the fact emphasized by Thiersch that many men with carcinoma of the lips have smoked moderately or not at all, the peculiar relation of cancer of the upper and lower lip to the sex of the patient should also be mentioned. Whereas, according to Bergmann's statistics, in 100 cases of cancer of the lower lip only 9.22

per cent. are women, in those of Eschweiler in 61 cases of the much rarer cancer of the upper lip 24, *i.e.*, 39.3 per cent., were women—a percentage more than four times as great.

We can as yet only confirm without explaining these strange facts. Possibly much more extensive statistics would yield different results.

Some other influences which are regarded as of etiological importance may be interesting to mention. Thus Partsch calculates that among 88 patients with cancer of the lips 37.5 per cent. were agriculturists, and 73.8 per cent. people whose occupations exposed them constantly to the inclemencies of the weather.

We have already learned to know that other diseases of the mouth predispose in a high degree to cancer; first of all, leukoplakia, next, carious teeth, chronic eczemas, and scars of all kinds. Gummata also, it would seem, may degenerate into cancers. The transformation of benign tumors into cancer (and here the papillomata especially come into question) has not as yet been actually proved so far as the mouth is concerned. But since this change has been proved to take place, though rarely, in the larynx, the possibility of its also occurring in the mouth cannot be lightly dismissed.

But all diseases, all injurious influences which are accused of being causes of cancer befall large numbers of men who do not acquire cancer. We must, therefore, suppose still another especial cause, from the knowledge of which we still appear to be far removed.

That cancer is in general a disease of advanced age is especially true of it as it affects the organs of the mouth. The sixth decade is especially exposed to lingual and labial cancer; in later and in earlier periods of life the liability is much less. Yet B. Destot[70] has observed a cancer of the upper lip in a young man of twenty-two years, and on the other hand, W. H. Talland[71] operated upon a man one hundred and two years of age for a cancer of the lower lip. The patient died nine months later of a recurrence.

All carcinomata when primary are almost always single. But this rule also has exceptions. Von Bergmann,[72] among others, has seen carcinomata which developed simultaneously on corresponding portions of the upper and lower lip. Gerwe[73] has reported carcinomata located, at the same time, upon the under lip and at both corners of the mouth. Here the question arises whether we may not have "inoculation metastases." At all events, such occurrences are extremely rare.

Cancer of the Lip.

Cancer is much more common upon the lower than upon the upper lip. The ratio is variously stated as 1:12 or 1:25.5. We have already spoken of the relative frequency in the male and in the female sex.

Labial carcinoma generally begins on the lateral part of the lip. Its commencement is rarely observed by the physician, and is quite variously described by the patients. Sometimes a wart, sometimes a small nodule, and sometimes a blister is reported to have first made its appearance. In the earliest stages in which we observe carcinomata, they appear generally as small hard spots in the vermilion border of the lip, more often near the edge of the skin, which are either flat-topped elevations or are somewhat sunken. Even at this time the epithelial covering is no longer smooth and unbroken; the surface is "raw"; it secretes a thin, somewhat sticky fluid, which dries readily and forms scabs which intentionally or unintentionally become torn off. When this happens, the surface bleeds easily and becomes uneven and cracked, notwithstanding attempts to cause it to cicatrize. But it must be especially emphasized that flat scars do occasionally form on the edges of slowly growing cancer of the lips which point to a partial spontaneous healing. Attention is to be paid to this point particularly with respect to the differential diagnosis. As a rule, very soon, but occasionally only after a long duration, the fact becomes recognized that we have not to deal with a simple ulcer or erosion. The vicinity of the ulcer becomes infiltrated and feels decidedly hard. Frequently this is first distinctly apparent after the removal of the hard and firmly adhering scabs. If the surface of the ulcer is closely examined, a certain yellowish-red coloration of its base and especially of its margin is observed, which depends upon the deposition of epidermoid elements in the tissues. Often these are recognizable as distinctly isolated dirty yellowish pale plugs of the size of the head of a pin, in the midst of a vividly red tissue.

As a rule, the ulcer with its surroundings then begins to rise steadily from the surface of the lip, and so acquires even more distinctly the character of a tumor. The margins become elevated and separate the ulcer-like walls from the surrounding tissues ("wall-like margins"). At the same time the hard infiltration about the ulcer spreads more widely, and as the latter grows invades larger and larger portions of the lip. The resulting stiffness and immovability of the lip trouble the patients, who have hitherto been but little

annoyed by the growth, more and more, and even the most negligent now frequently decide to seek medical advice. Hitherto the affection has generally progressed slowly (sometimes years pass before this stage is reached), but now, if there is no treatment, or if the treatment is unsuitable, it usually develops with much greater rapidity. Perhaps in this the frequent traumatisms to which the stiff and unyielding lip is exposed play an important part. The nature of the tumor becomes more apparent from the cancerous infiltration which constantly extends. Soon the neighboring glands begin to enlarge; at first generally the submaxillary, then the submental glands. At a later time the glands of the neck become involved. The first beginnings of infiltration of the glands are, as a rule, not easily made out. If they are superficial, palpation discovers them as hard nodules of the size of a pea, which are freely movable, or are adherent to the edge of the jaw. If, on the other hand, they lie buried between the muscles of the jaw and the submaxillary region they can only be detected with any degree of probability from a slight increase in the resistance of the affected region. In this examination bimanual palpation may be of great value.

The sufferings of the patient are much increased when the infiltration passes from the lip to the gums and the tumor begins to adhere to the jaw bone. Tears then occur at the adherent portions as soon as the ulcer which progresses with the infiltration reaches them. The pains, at first only moderate and occasional, become steadily more violent, almost incessant, and are made much worse by attempts to eat. When the extent of the tumor has become great the surface of the ulcer is no longer covered with scabs but with superficially necrosed portions of the tumor. These latter quickly undergo the putrefactive processes, which develop so readily within the mouth, and so the malady advances, becoming steadily more unendurable. The lymphatic glands swell into great nodes which may unite with the primary tumor. The taking of food through the mouth, the tissues surrounding which have been converted into one uniformly stiff immovable mass, becomes more and more difficult. Suppuration now becomes a prominent feature. The cancerous lymphatic glands may also suppurate.

As a rule, even when cancers of the lip are far advanced, the tumor formation predominates. Even when the tumor is ulcerated over its whole extent, the surface of the ulcer forms a thick mass distinctly raised above the normal level. Exceptionally, especially when the nutrition of the patient is much impaired, the ulcerative processes may follow so closely upon the heels of the carcinomatous infiltration that at the first glance one sees only widespread destruction of

the lip and the neighboring skin and mucous membrane. Not until a more careful examination is made is it noticed that the base and the margins of the ulcer are formed of a moderately indurated layer of cancer tissue, a few millimetres thick.

In general the patients die from defective nutrition and the drain

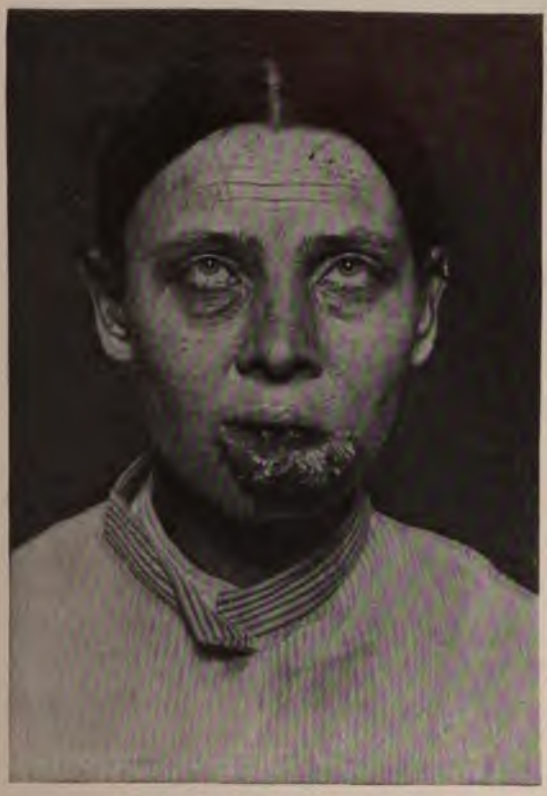

FIG. 12.—Carcinoma of the Lower Lip.

This figure represents an extensive carcinoma of the lower lip in a woman forty-five years of age. It was removed, and a plastic operation was performed on the lip after Langenbeck's method. A rather extensive operation for the removal of lymphatic glands was also necessary. A typical flat-celled epithelial carcinoma. (A photograph from the Breslau Surgical Clinic.)

upon the strength by the profuse suppuration from the ulcerated surfaces, often also from pneumonia due to the inspiration of infectious secretions.

The duration of cancer of the lip is relatively long. Partsch calculates the average duration of life to be 3.72 years. If it be further considered that even in the relatively extensive and fatal cancers, metastases, as a rule, occur only in the neighboring lymphatic glands, very rarely in the internal organs, it is certainly correct to count can-

cer of the lip among the more benign carcinomata. It is analogous to the flat cancers of the skin in this clinical relation, as also with respect to its purely histological characteristics. Microscopical preparations of it generally present a typical picture of the cornifying, epidermoid, flat-celled, epithelial cancer.

In view of its relatively benign character, an early operation is doubly important. Cases of extensive cancer of the lip are still seen in which the patients have been delayed by treatment with caustics, ointments, and the like until the tumor had become so enormous as to force the diagnosis of cancer even upon the layman. Nowhere is there perhaps a greater misuse of caustics than in cancer of the lip, and the statement is so frequently made by the patient that after such cauterization the growth of the tumor had been much more rapid, that one is forced to ascribe this unfortunate result to the cauterization.

Even in case of small and doubtful tumors an attempt to remove them by cauterization should be strongly discountenanced, since it is precisely tumors of this class which are removed so easily without disturbance of function or severe operation by the excision of a wedge-shaped portion of the lip. It is quite reprehensible to let this favorable moment pass and to permit the formation of more extensive infiltrations and of metastases in the lymphatic glands. In larger cancers the interference is more extensive on account of the extirpation of the glands and the plastic operation upon the lip which become necessary.

The patient's salvation then depends essentially upon an early diagnosis. The characteristics of an incipient labial cancer have already been described and it only remains now to exclude those conditions which may possibly be mistaken for it. There are not many of these; tertiary syphilitic and tuberculous ulcers are rarities upon the lip. The latter, moreover, are rarely present singly. In the beginning of the disease a benign papilloma might be thought of, and, indeed, if such a tumor has a broad base, the differentiation might be hardly possible. It is well, however, to regard papillomata with great suspicion, especially in elderly patients, and the unnecessary extirpation of such a tumor is certainly a less serious mistake than the failure to remove an easily operable carcinoma.

In young patients of both sexes cancer of the lip is so extremely rare that a suspicious induration of the lip would lead one rather to think of an initial syphilitic sclerosis which, in fact, presents a certain similarity to an incipient infiltrated carcinoma. But also in older patients, especially women, if the history is insufficient or untrustworthy it is well to consider this possibility. The relatively

rapid development of the syphilitic primary lesion will, as a rule, prevent this error. In doubtful cases it is well to inquire whether the patients have had the care of nursing infants who might possibly have congenital syphilis. The swelling of the lymphatic glands, which in case of the primary lesion of syphilis is generally great in comparison with the size of the lesion itself, will occasionally guide to the correct diagnosis.

In extirpation of the tumor it is most important to operate in the healthy surrounding tissues. In small cancers it is easy to follow the usual directions and keep 1 cm. from the margin of the growth and to effect the removal by excision of a wedge-shaped piece of the lip under local anæsthesia after Schleich's infiltration method. With tumors of greater extent it is better to perform an extensive operation than an incomplete one. Above all, the diseased lymphatic glands should be removed.

With such an operation the chances of permanent recovery are good. Partsch calculates that thirty-five per cent. are cured. In a small series of cases collected by Odenthal[11] (from Trendelenburg's clinic) as many as 70.3 per cent. of the patients whose subsequent history could be learned remained free from recurrence.

Inoperable cases, especially cases of recurrence, constitute a terrible malady, in the treatment of which the narcotics must be extensively used. Artificial feeding with the œsophageal catheter, if necessary introduced through the nose, is sometimes required. All remedies are powerless for the ichorous suppuration of the ulcerated surfaces. Iodoform is still the most effective application, and compresses soaked in acetate of aluminum may be of service both for the suppuration and the pain.

Cancer of the Tongue.

Although not so common as cancer of the lip, cancer of the tongue has a much more ill-omened significance, because, upon the average, it grows much more rapidly and reaches at an earlier period a stage in which the radical operation can produce no, or only very temporary, good results. This especial malignancy cancer of the tongue owes mainly to the proximity of the floor of the mouth, which it often invades early in its course. The loose connective tissue, rich in lymphatics, opposes very little resistance to the local extension of the cancer, and soon the lymphatics over an extensive area are infected. The spread of the cancer is probably also materially favored by the mechanical massage effect of the movements of the tongue. It is proper, therefore, from a clinical standpoint to

separate cancer without from cancer with implication of the floor of the mouth.

The differences as to the site of the primary localizations are also of importance. Wölfler's[75] statistics show that of 48 patients the original site of the cancer in 22 was on the edge of the tongue (13 of these on the right edge); 6 each were upon the apex, the under surface, and the frenulum of the tongue, 2 were on the right side of the base of the tongue, 1 was upon the dorsum, and 1 in the centre of the upper surface of the tongue; in 4 cases the primary localization was upon the floor of the mouth. Seventeen out of 40 cancers were confined to the tongue; in the remaining cases the floor of the mouth was implicated. The remarkable preponderance of the right half of the body is as yet inexplicable.

From an anatomical point of view we may distinguish cancers which originate in the epithelium of the tongue's surface and those which originate in the glands of the tongue. Those of superficial origin are certainly in the large majority. They are typical, often cornifying, flat-celled epithelial cancers, which from the first are inclined to ulcerate and which extend superficially rather than deeply.

The deep "gland cancers" are at first nodules situated beneath the mucous membrane and not ulcerated. But they soon infiltrate the mucous membrane and ulceration results; they then present no clinical differences from the other form. Their epithelial constituents do not show the numerous layers, and above all have not the tendency to cornification of those of the other form. With respect to malignancy there is little difference between the two forms. Yet the flat-celled carcinoma is generally soon noticed and it is then easy to thoroughly extirpate it. Most gland carcinomata come under the observation of the surgeon at a later, generally an inoperable stage.

The development of true cancer is often preceded for a long time by diseased conditions which, as it were, prepare the soil for the cancer. This is especially true of leukoplakia and simple ulcer, the decubital ulcer already described. In carcinoma of the tongue areas of leukoplakia are often still found in other parts of the mouth, frequently most developed in the immediate vicinity of the cancer. How far this pathological proliferation of epithelium may merge directly into cancer, it is as yet impossible to say with certainty. But the one disease follows the other with such surprising frequency that a causal connection between the two must be supposed.

That the first beginnings of a cancer of the tongue were represented by an ulcer caused by a decayed tooth is so often stated by the patients that it is not proper to assume that in all cases the car-

cinoma was the primary condition and that the carious tooth had simply caused its ulceration. But it is true here as of leukoplakia, that no one has yet demonstrated with certainty histologically the transition of the one process into the other.

At all events the soil is prepared frequently in one of these ways and long in advance for the development of the cancer. With or without this preliminary stage the first evidence which the patient notices of the new and severer malady is either a small flat ulcer or a firm nodular infiltration which is more or less elevated above the surface of the tongue. At first the nodule or the ulceration is

FIG. 13 represents a microscopical preparation from a very small cancerous node of the tongue. The nests of epithelial cells are seen, in part, disposed as true cancroid pearls. Around them is a considerable infiltration of small round cells. (From Partsch's "Die Geschwülste der Mundgebilde," Fig. 108.)

sharply defined with respect to the surrounding tissues, but with its continued growth, which usually takes place more rapidly after the tumor has reached a certain size, the boundary becomes less and less marked. This phenomenon depends upon the filling of the neighboring tissues with an abundant cellular infiltration, which becomes more and more prominent a feature as the tumor rapidly increases and which shades off gradually into the surrounding healthy tissues. This infiltration is the cause of the hardness of the base of the ulcer, which is often in the early stages one of the most characteristic signs of the disease.

Very soon there develops a wall-like thickening of the margins of the ulcer, caused by the epithelial plugs which grow from the whole base of the ulcer into the underlying tissues and which are crowded together in thick masses in the margins.

At this stage the positive diagnosis of cancer is generally possible. As a striking and always very suspicious symptom, sharp

pains may even now occur. It has often been stated to be characteristic of these pains that they dart to the ear of the affected side. But this symptom remains absent in a great number of cases, not only at this but also at later stages of the disease. On the other hand, the hemorrhages which are an unpleasant feature of lingual cancer may occur at an early stage. They are either spontaneous or are caused by slight injuries, which so easily befall the hard, unyielding, and at the same time brittle new growth.

The ulceration and infiltration constantly extend, but these processes are relatively slow so long as the cancer is confined to the

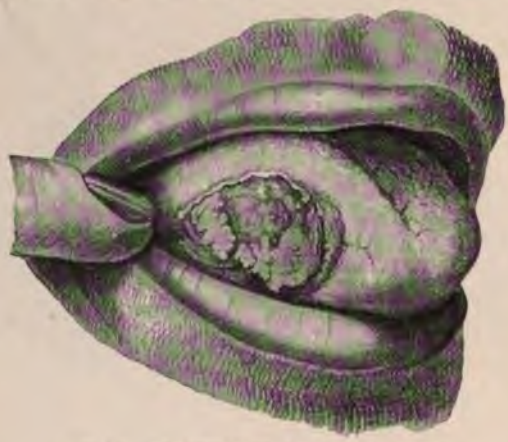

FIG. 14.—Epithelial Cancer of the Tongue.

This figure represents an ulcerating flat-celled epithelial cancer of the tongue in a woman fifty years of age, who had first noticed it two years before as a hard and but slightly painful nodule, of the size of a lentil. Previously to this the tongue had repeatedly become sore at this point by rubbing against sharp edged roots of the teeth. Nothwithstanding the removal of these roots the nodule continued to grow. The tumor is of the size of a pigeon's egg and is elevated several millimetres above the level of the tongue. Its surface is partly ulcerated, partly covered with swollen, milky white epithelium. The margins are raised. The mass feels flat and hard to the touch, and pretty sharply defined. The lymphatic glands cannot be detected on palpation. After preliminary ligature of the lingual artery the tumor and a few glands at the angle of the jaw were removed. The healing was uninterrupted. Seventeen months later, the patient having remained well in the interim, there was an extensive recurrence in the tongue and in the lymphatic glands. (From Mikulicz' Atlas, Plate XXXVI., Fig. 1.)

tongue proper. As long as this remains true of the tumor the lymphatic glands also are generally not infected, according to the statements of Wölfler. He found them diseased in only four cases out of seventeen. (The retromaxillary glands were the ones affected.) But it would seem that Wölfler had paid little attention to the glands on the floor of the mouth, which represent the first station of the lymphatic system of the tongue. They are frequently diseased very early.

The movability of the tongue, consequently its usefulness during mastication and deglutition, is up to this time but slightly impaired.

But in the following stage the picture of the disease becomes very different, when the cancer attacks the loose mucous membrane of the under surface of the tongue and also, as is generally the case, the floor of the mouth. Then the organ becomes fixed to the jaw and to the floor of the mouth, its movability becomes steadily less, and mastication and deglutition are increasingly more difficult. A recess is formed from which the movements of the tongue can no longer sweep fragments of food. These stagnate and decompose and very quickly there develops an extremely repulsive ichorous suppuration, a torture to the patient and his friends. Antiseptic mouth washes, which are generally tried, the patient cannot apply upon the especially important parts, and he finally gives them up as useless. If the floor of the mouth is implicated, the swelling of the lymphatic glands is rarely absent. According to Wölfler it is most marked in the submaxillary, and less often in the retromaxillary glands.

The now rapidly growing carcinoma soon makes the patient's condition unendurable. The pains become more and more severe, and are almost incessant. They are increased by attempts at taking food. Aside from this, mastication and deglutition become increasingly difficult and the patient contents himself with soft or fluid food. In many cases hemorrhages also occur, sometimes very alarming ones, and so the patient's strength declines more and more. A new obstacle to swallowing is the implication of the anterior arches of the palate, which is rather common, especially if the carcinoma is situated on the posterior part of the tongue.

Since the patient cannot swallow the saliva, which on account of the decomposition is formed very abundantly, this ill-smelling fluid escapes at the corners of the mouth and also backwards into the respiratory tract. If he acquires, in consequence, a fatal pneumonia, that can really only be regarded as a release, which, however does not soon fall to the lot of every patient. Sometimes the cancer also attacks the alveolar process and from that or from the palate advances to the intermaxillary fold. Then a kind of lockjaw results which makes the condition of the patient still worse. The patient finally dies from inanition or from metastases to the internal organs.

The disease, which attracts so little attention at the beginning, is the source of most terrible sufferings in its subsequent course. Hence it is especially necessary with reference to it to endeavor to operate at the earliest possible moment, while there is still a tolerably favorable prospect of a permanent cure. For the whole disease runs its course, in general, quite rapidly. Its average duration until death is only a little more than one year, if no operation is performed (Wölfler).

Diagnosis.—The most important prerequisite for a timely operation is an early recognition of the affection. In most patients the operation is hopeless because they themselves and, unfortunately, often their physicians also, fail to recognize soon enough the serious significance of the disease, and so the favorable time for operation is allowed to pass.

It is true that the diagnosis may be quite difficult even in the later stages. The not yet ulcerated nodule can be mistaken for a benign tumor, a sarcoma, a deep-seated tuberculous node, a gumma, or a focus of actinomycosis. If ulceration has occurred, besides the conditions already mentioned, the cancer may be confounded with a decubital ulcer, and, under certain circumstances, also with a syphilitic primary lesion, and even with a nodular lymphangioma. The differential diagnosis from cysts which is often discussed is probably never necessary in practice, nor that from scleroma, leprosy, and glanders. The latter rarer affections are sufficiently distinguished by their mode of extension (already described in Volume VIII.) and by the almost invariable development of multiple foci.

The gumma in its deep and nodular, as well as in its superficial and ulcerated form, is the affection most commonly confounded with, and the most difficult to distinguish from cancer. But gummata which have not ulcerated can be confounded only with the deep gland carcinomata, and these, in general, quite rare tumors can, in fact, hardly be distinguished from gummata, if there is no history in favor of the latter. They are as ill-defined as the syphilomata; they show the same tendency to develop towards the surface, and may occur in any part of the tongue, precisely like the gummata. In such cases it is proper to institute antisyphilitic treatment or to examine a bit of tissue from the node microscopically—but not if glandular swellings are already present in the floor of the mouth.

A flat cancroid might be confounded with a superficial sclerosing glossitis, but these cancroids, as Fournier remarks, never form the "plateaux d'induration" which are so characteristic of sclerosing glossitis.

Most difficult is the distinction between an ulcerated gumma and a cancroid; it may often be impossible. Sometimes general considerations throw a certain light upon the diagnosis (Fournier): 1. While cancroids occur, as a rule, only with advancing years, generally after the fiftieth year, we often find gummata in young persons; 2. The occurrence of carcinoma in previous generations, especially in the parents of the patient, speaks in doubtful cases in favor of carcinoma; 3. The existence previously of symptoms which point to a leukoplakia also makes carcinoma the more probable; 4. A very

considerable and rapid loss of bodily strength, especially if it is not directly dependent upon impediments to the taking of food, has the same significance.

But these points can only support the diagnosis, or cause carcinoma to be suspected. A series of diagnostic points, however, may be established which oppose the diagnosis of gumma: 1. A location on the under surface of the tongue occurs almost never in gummata; their tendency, even in the later stages of the disease, is always to develop towards the dorsum of the tongue. Ulcers so situated are almost never gummatous, unless a sublingual or a Blandin-Nuhn gland becomes diseased. That is, however, very rare and the disease is easily recognized; 2. The occurrence of hemorrhages spontaneously or after slight injuries is opposed to the diagnosis of gumma. The lardaceous surface of the ulcer of gumma is not easily caused to bleed; 3. Severe pains, especially before the disease has extended widely, do not occur in gummata spontaneously nor are they easily provoked by examination; they also do not occur during eating or are not made materially worse by it; 4. Swelling of the adjacent lymphatic glands almost absolutely disproves the existence of gumma.

For the diagnosis of a gumma and against that of carcinoma are: 1. Multiplicity of foci. It has already been remarked above that this is an extreme rarity in carcinoma; still it does occur, and Fournier, for example, reports two such cases; 2. Location upon the dorsum or at the base of the tongue is more probable for a gumma than for a carcinoma.

Besides these diagnostic points the appearance of the focus itself is especially to be noted. However marked the ulceration, carcinoma always appears as a tumor, *i.e.*, as an independent swelling with which the firm infiltration on the floor of a gummatous ulcer is not at all to be compared. The examining finger can, as it were, lift the cancerous ulcer, together with the tumor on the summit of which it is situated, out of its bed, separate it from the underlying tissues, notwithstanding the absence of well-defined demarcation. That cannot be done with the gumma, which always appears as a loss of substance.

For the same reason the gumma, moreover, forms an ulcer with abrupt, steep edges; in carcinoma the margin as well as the base of the ulcer is tumor tissue, consequently the margin is thickened like a wall and firm like the hem of a garment, and slopes gradually. To use a comparison, the cancerous ulcer is not hollowed like a soup plate but like a saucer.

The floor of a gummatous ulcer is filled with a greasy but firm

lardaceous mass, as the bottom of an empty ditch is filled with mud. A carcinomatous ulcer has an uneven floor covered with large and small nodules. It has not the uniform yellow color of that of gummatous ulcer, but appears somewhat mottled, yellowish spots alternating with red streaks. If an attempt is made to wipe off these yellow spots, they are often found to be plug-like comedones or crumbly caseous material which can be lifted out and under the microscope proves to be masses of flat epithelium, cancroid pearls.

Extensive processes of cicatrization may also be important in the diagnosis; these do not readily occur in carcinoma without operative treatment. Limited cicatrization does not, however, absolutely oppose the diagnosis of cancer. It is sometimes distinctly apparent in cancroids.

Extensive infiltrations, firm thickenings of the neighboring tissues which effect a fixation of the diseased parts to the lower jaw or floor of the mouth and much impair the power of protruding the tongue, almost never occur in gummata but are common in advanced carcinomata.

If these points are kept clearly in view in the examination of the patient, the diagnosis can be made from them alone in the majority of cases. In some cases, however, the question still remains unsettled, and then we must resort to antisyphilitic treatment or to the excision of a piece of the tumor for microscopic examination.

But in instituting treatment for syphilis the old fact, emphasized by Fournier, must be kept in mind that mercury stimulates cancer, communicating to it a new vigor of growth. Too often, unfortunately, we see patients who have been treated with iodide of potassium and mercury, sometimes for whole months, before the diagnosis of cancer is made and an operation is advised, which is then, alas, too late!

The warning, therefore, against the purely routine treatment of cancer patients with mercury and iodide of potassium can hardly be made too strong. Fournier says rightly that such treatment is only justifiable when there is "some hope, even some gleam of hope, however feeble it may be, in favor of syphilis." But frequently iodide of potassium is administered for long periods when there could really be no doubt of the presence of cancer, if the patients were thoroughly examined; which is, of course, very reprehensible. It is for this reason that the differential diagnosis has been discussed at such length.

But if it is felt to be necessary to institute such treatment, let it be done energetically. Fournier recommends large doses of iodide

of potassium—3, 6, even 10 gm. (gr. xlv.–cl.) per diem. Large doses take effect more rapidly and certainly should be used in such cases where everything depends upon saving time. Mercury should be administered cautiously, else the patient who very often cannot keep his mouth as thoroughly clean as is necessary during a course of mercurial treatment acquires to his misfortune a mercurial stomatitis and the conditions become still more difficult for diagnosis. An energetic treatment with iodide of potassium alone for fourteen days is sufficient to clear up most of the doubtful cases.

In many cases the microscopical examination of a fragment is much to be preferred to antisyphilitic treatment. It is not difficult to distinguish a carcinoma from a gumma under the microscope, always provided that a suitable portion of the growth is selected for excision. The removal of a portion for examination can easily be accomplished with the aid of cocaine.

Next to mistaking for syphilis, the most easily made and most serious error is confusion with the products of tuberculosis. Deeply situated and isolated tuberculous nodes present great similarity to gland carcinoma which has not yet ulcerated. But if no tuberculous disease of other organs is discovered the excision of such nodes is as justifiable in tuberculosis as in carcinoma; practically, therefore, such a mistake is of little importance. But tuberculous ulcers also require to be distinguished from cancer, and here one point is especially important: In the vicinity of tuberculous ulcers a marked inflammatory reaction is seldom wanting; the margins of the ulcer are surrounded by an often quite broad zone of deep, sometimes cyanotic reddening. Moreover, distinct miliary nodules are frequently found around the tuberculous focus, which leave no doubt as to its tuberculous nature. If, in addition to this, several separate ulcers are present, carcinoma is almost absolutely excluded.

The diagnosis is generally easy if the two following peculiarities of the tuberculous ulcer of the tongue are borne in mind: (1) it generally appears in the form of rhagades, and (2) the floor of the ulcer and the surrounding tisues lack the firm infiltration of cancer.

In tuberculosis of the tongue, even of long duration (aside from acute miliary tuberculosis of the organ) the neighboring lymphatic glands are not, as a rule, implicated. If this is exceptionally the case, and if there is doubt of the diagnosis, it may be decided to excise a gland and examine it microscopically. It must be remembered, however, that the first beginnings of carcinoma, as of tuberculosis, in the lymphatic glands are often very difficult to detect, and, on the other hand, that tuberculous glands may happen to be present with an undoubted carcinoma, as was once observed in the Bres-

lau Surgical Clinic. It is safer, therefore, always to excise tissue for microscopical examination from the primary focus.

Carcinoma will hardly ever be confounded with a benign tumor, a fibroma, a lipoma, or the like. The question could only arise in case of the gland carcinomata, and they are rarely so sharply defined from the surrounding tissues nor is the mucous membrane over them so movable as in fibroma. Sarcoma can more easily cause difficulties in the diagnosis. If the tumor has not yet become ulcerated, a distinction is quite impossible. If ulceration is present, the independence of the ulcer from the tumor may lead to the recognition of sarcoma. In carcinoma the ulcer is formed from the outset at the expense of the tissues of the tumor.

Actinomycotic nodules may present an extraordinary resemblance to cancer, especially by their hardness, by their tendency widely to infiltrate the tissues, and by their adhesions to neighboring organs. But actinomycosis occurs frequently in young subjects and sometimes the history (its origin from the penetration of a husk, etc.) points to this affection. Moreover, there are also found here and there upon the surface of the tumor of actinomycosis small yellow points, pus foci, which lead to the correct diagnosis.

Finally, the primary lesion of syphilis should be mentioned. By palpation it is hardly to be distinguished from carcinoma, but the color and the general appearance of the surface of the lesion are aids in the diagnosis. The rapidity of its development will generally exclude carcinoma. It is to be remembered that infection from syphilis due to the occupation of the patient, as in the case of nurses, frequently occurs at the age most liable to cancer.

The *treatment* of cancer, when once recognized, consists solely in its removal by operation. The results with respect to a cure are not so unfavorable as is often supposed. Wölfler estimated fourteen per cent. of cures in cases from Billroth's Clinic of more than three-fourths of a year's duration. To-day the percentage is certainly still higher now that we operate in the healthy tissues at a greater distance from the tumor and remove also, nearly without exception, the neighboring lymphatic glands.

This is not the place to discuss the operative methods. Only the most essential points which must guide us to-day in fixing the indications for the operation, in the prognosis of the operative treatment, and in the technique of the operation will be briefly touched upon.

First of all, it should be stated that the formerly serious danger of the operation is to-day reduced to a minimum. In the first place, the checking of hemorrhage from any point is made certain by the present technique (preliminary ligature of the lingual artery, imme-

diate deep suturing of the wound of operation), and hence the inhalation of blood is almost excluded. In the past ten years I have had no fatal cases in consequence of the operation for cancer of the tongue. I operate always with the patient semi-anæsthetized and in a sitting position. The reflexes are then so far preserved that blood which reaches the entrance of the larynx is at once coughed and hawked up. Hence I make no preliminary tracheotomy.

In the second place, we are now able to exclude with certainty the infectious processes which formerly so frequently attacked the wound and its vicinity and, secondarily, the lungs (aspiration pneumonia). The sovereign remedy for this is still iodoform. The wound cavity is tamponed with iodoform gauze, which is left in the mouth undisturbed for six to ten days. In this way the after-treatment is far less painful than was formerly the case. The patient can take fluid food by means of a feeding-cup from the first without any especial difficulty.

The question, whether a case should be operated upon or not, is, as a rule, difficult to answer. Besides the general condition of the patient, there are two points which decide it: first, the local extent of the tumor; second, the extent of the gland metastases.

It is necessary that at least 1 cm. of healthy tissue in all directions can be removed with the tumor. In this regard we shall seldom encounter any obstacles, since it is easy, so far as the technique is concerned, to remove the whole tongue, the floor of the mouth, and even the neighboring portion of the lower jaw. But so extensive an operation is almost always contraindicated because the lymphatic glands are at the same time seriously involved. This fixes a limit for our interference at a much earlier period than does the local extension of the cancer. I regard the prospect of radical cure, *i.e.*, the absence of recurrence, as excluded as soon as the deep glands of the neck become infected. For this reason I cannot approve of too extensive operations for cancer of the tongue. The cases in which I would perform excision of the entire tongue are extremely rare.

Only in the case of very small carcinomata on the tip of the tongue do I content myself with local extirpation; in all other cases, even in the small carcinomata of the edge of the tongue, I remove the glands of the submental and of one or both submaxillary regions, although no glands may be perceptible on palpation. This operation is, of course, preceded by ligation of the lingual artery.

Some surgeons demand the removal of the entire tongue in all severe cases and justify it by the fact that this causes relatively slight disturbances of function. But although the disturbances are only relatively slight, to the patient they seem sufficiently

severe. It must be remembered that the large majority of relapses do not occur in the scar. Of the thirteen patients with relapses concerning whom Wölfler reports, the recurrence took place certainly only in the lymphatic glands in seven, and but twice with certainty in the scar, and of these two patients one was still alive two years after the removal of the recurrent tumor. My experience fully agrees with this.

From the preceding it appears that better results are obtained from a very early operation, the cancer being diagnosticated before the lymphatic glands become diseased.

For the inoperable patients, who are truly a crux medica, extensive use must be made of narcotics. But they should not be used too freely at first; the subcutaneous injections of morphine especially should be reserved for a later stage in which the patients are tortured day and night with severe pain. I have several times known patients, in whom morphine injections had lost their effect, to be driven to suicide.

For the ichorous suppuration of the ulcer the insertion of iodoform gauze between the teeth and the ulcer, which is changed once or twice a day, is sometimes of benefit. In severe hemorrhages ligation of the lingual artery is occasionally necessary. This may, however, be attended by enormous difficulties if there are great bunches of enlarged glands in the neck. The same is true of section or resection of the lingual nerve which has been repeatedly undertaken for the relief of the terrible pains.

To nourish the patient fluids or gruels are introduced by means of the feeding-cup as long as possible. At a later stage it is necessary to use the œsophageal sound inserted through the nose.

Cancer of the Floor of the Mouth.

Although the floor of the mouth is often secondarily implicated in cancer of the tongue, it is very rarely the primary site of cancer. Of forty-four patients with primary cancer near the tongue, Wölfler found only four in whom the primary location was on the floor of the mouth.

Here also are found both cancers proceeding from the surface epithelium and deep-seated gland cancers. Thus, for example, the sublingual gland may be their place of origin. But because of the slight thickness and small power of resistance of the overlying mucous membrane the differences between the two forms of tumor are obliterated at an even earlier stage of the disease than elsewhere. The two relatively most frequent points of origin are the region of the

sublingual gland and the median line near the frenum linguæ. The first appearance of the disease is almost always in the form of an ulcer, rarely of a nodule, which, however, quickly becomes "raw." The cancer is at first not different in its appearance and course from that of the tongue, but it acquires an alarming malignancy as soon as it has penetrated into the submucosa and has found in its loose tissues the extremely favorable conditions of growth which have already been alluded to.

The functional disturbances in cancer of the floor of the mouth become quickly very considerable. The tongue soon becomes adherent to the floor of the mouth or to the lower jaw, and speech and mastication are much impeded. But although the patients are thus led to notice the disease at a relatively early period, they generally first come under observation with an already extensive growth of the tumor and with metastases in the glands—a sign of the rapidity of growth of these carcinomata.

With respect to the diagnosis, nearly everything applies which has been said above concerning cancer of the tongue. The (tertiary) syphilitic disease of the salivary glands which Neumann describes (see Vol. VIII., p. 47) could easily be confounded with cancer of the floor of the mouth unless great caution were exercised.

The prognosis must be very guarded even for small foci and with an early operation. Permanent recovery is rare. It is especially in place here to act rapidly and to cause no unnecessary delay by prolonged treatment for syphilis.

Cancer of the Cheek and of the Intermaxillary Fold.

Carcinoma of the buccal mucous membrane, even more frequently than that of the tongue, is located near carious teeth, especially carious second molars and wisdom teeth. Here it seems always to occur as flat epithelial cancer originating from the surface, with regard to the appearance, growth, etc., of which little can be added to what has already been said. Carcinoma of the cheek is especially formidable when, as is usually the case, it is situated in the posterior portions of the buccal pouch. It then, as a rule, very quickly invades the intermaxillary fold, where in fact it sometimes originates. The consequence of the dense infiltration of this tissue is very soon lockjaw, which prevents thorough examination of the diseased parts and is responsible for the fact that very often in such patients the condition is recognized and operation is performed too late.

The distinction from a decubital ulcer of dental origin is the most difficult, the more so because when the jaws are locked the extraction

of a carious tooth, if one is present, and so the diagnosis *ex juvantibus* is hardly possible. But in purely inflammatory trismus, by very gradually forcing the jaws apart one can, as a rule, obtain sufficient space for inspection. In carcinoma the attempt is generally not successful. This may sometimes serve as a hint as to the diagnosis.

Of other ulcerative processes which may make a diagnosis difficult tuberculosis and actinomycosis are especially to be considered (gummata are hardly ever located here). But both processes develop much more slowly than carcinoma and frequently also in young subjects. They are always accompanied by inflammatory processes in the surrounding tissues. Moreover, the discovery of tubercle bacilli or actinomyces kernels will not infrequently confirm the diagnosis.

Carcinomata of the cheek are far less malignant than those of the floor of the mouth and long remain localized. But they have one unfortunate peculiarity besides their frequent difficulties of diagnosis, which is, that their extirpation is complicated by the necessity, after the removal of the tumor, of doing a sometimes very extensive plastic operation to replace the lost portions of the cheek, unless one is willing to take the risk of a serious cicatricial locking of the jaws. For this purpose double flaps are to be raised, mostly from the skin of the neck, and the operation may be quite difficult and extensive. It is therefore advisable if the cancer is extensive to adopt the method proposed by Mikulicz of lateral pharyngotomy with resection of the ramus of the lower jaw. It affords the advantage that in case of recurrence the painful locking of the jaws at least does not occur.

Cancer of the Palate.

Cancer of the soft parts of the palate is probably not so rare as Kronacher[76] states, who found besides his own case only one other described. In the tables of Eisenmenger and Volkmann (see above) are five certain cases, besides five which are doubtful, and to these seven others can be added from the literature. One case is depicted in Mikulicz' Atlas (Plate XIV., Fig. 1), in which, however, the cancer appears to have originated in the alveolar process, and in the Breslau Surgical Clinic two cases have been observed in which the cancer certainly, and two others in which it probably originated in the palate.

Here also two forms are found: the flat-celled epithelial cancer proceeding from the surface of the mucous membrane, and the gland carcinoma which originates in the abundantly present glands of the palate.

For both forms the place of origin is most frequently the pos-

terior part of the hard palate. While the superficial cancroid has quite the same appearance and the same course as in other parts of the mouth and very quickly becomes a "cancerous ulcer," the gland carcinoma, which is more frequently observed in this situation than elsewhere, before the stage of ulceration presents an essentially different picture.

We find (Himmelreich[77]) a flat-topped tumor projecting into the mouth, with a smooth surface covered with intact mucous membrane and of a firm, even hard consistence. The tumor frequently penetrates into the nasal cavities at a very early period, even before its overlying mucous membrane is destroyed, an occurrence which causes this kind of carcinoma to resemble most closely gumma and which makes the diagnosis, difficult in any case, still more confused. It also very easily penetrates into the antrum of Highmore; in the case mentioned both antra were attacked by the tumor. Having reached these large cavities, the tumor finds abundant space for rapid growth, and at a later period it is often difficult to decide whether the palate or the mucous membrane of the antrum was the point of origin.

The cancroid shows the same tendency to perforate upwards, but in this case the perforation does not occur at so early a period.

The recognition of gland carcinoma before ulceration occurs may be very difficult. But, as a rule, the lateral extension of the tumors is so great that at least a gumma, which in general has a spherical form, can be excluded. Sarcomata which do not implicate the entire body of the jaw are rare in this region, and would demand the same treatment. The same is true of the more malignant forms of endothelioma. The distinction from the two last-named tumors will hardly ever be possible without microscopic examination.

If ulceration has occurred or if the tumor began primarily as an ulcerated cancroid the cancerous ulcer, as a rule, presents the already frequently mentioned characteristics and makes a positive diagnosis possible. It should also be remembered that gummata are almost always situated in or near the middle line, carcinomata much more frequently laterally. As their growth continues the latter do, it is true, extend over the median line, but at this stage they are not so easily confounded with gumma.

Tuberculous ulcerations which are also often laterally located and may occasionally lead to perforation are generally characterized on the palate by a surrounding zone of miliary nodules and by numerous smaller ulcers separate from the principal focus.

For the treatment it is very important to determine whether the carcinoma affects only the palatal process or whether the antrum is

primarily or secondarily affected. In the latter case it is better to decide at once upon resection of the jaw; in the former resection of the palate process, a much less serious and disfiguring operation, will suffice. The decision is often not at all easy. As a rule, however, the implication of the maxillary sinus is betrayed by a bulging and softening of its nasal wall. The tumor sometimes breaks into the nose from this cavity and thus clears up the situation. In doubtful cases we may commence with the resection of the palate process, and if it should appear that the disease extends far upwards the resection of the upper jaw can be performed.

In general, cancer of the palate is distinguished by its relatively slight malignancy, especially the gland carcinoma. It grows rather slowly, has a considerable tendency to recur locally, but it is only rarely and at a late stage that it forms extensive metastases. Even the lymphatic glands quite frequently escape involvement in the disease. But the resection of the jaw necessary for its removal, in view of the impairment of the patient's strength from defective nutrition, is an operation attended with considerable risk.

Two cases of carcinoma confined to the uvula are to be found in the literature, those of Malmsten[78] and of Katzenstein.[79] Both were about the size of a walnut and were situated upon the tip of the uvula. Amputation of the uvula was performed. The second case was certainly a cancroid with metastases in the lymphatic glands of the neck; the first case is called an epithelioma. Another quite analogous case was observed by Rydygier.

Tumors of the Jaw.

Tumors of the jaw are, in general, of purely surgical interest. We therefore will here speak only of their chief characteristics, so far as these manifest themselves by changes within the mouth.

Fibroma and Granuloma of the Alveolar Process.

On the edge of the alveolar process small tumors frequently develop which take their origin sometimes from the gum in proximity to diseased teeth, sometimes from the alveolus or the pulpa dentis after extraction of the teeth, and which consist of connective tissue abundantly infiltrated with fluid and with round cells. They have generally a thin pedicle, are attached in the immediate vicinity of the diseased tooth or within the alveolus, and their bulk generally fits closely in the carious cavity. They are granulation tumors, quite similar to the analogous tumors of the middle ear and other

localities. It appears rather doubtful whether they may be regarded as genuine fibromata.

Their clinical importance is slight. They may produce inconvenience by getting between the teeth during mastication when they are easily injured, and give rise to slight hemorrhages; otherwise they cause no trouble. They are readily snipped off with scissors, but are apt to recur so long as the carious tooth is present. When the tooth is extracted they are often torn off with it; if not, they generally shrivel up spontaneously.

Similar larger fibromata or fibrous hypertrophies, manifestly like the preceding, produced by continuous mechanical irritation may cause their possessors considerable annoyance by interference with mastication and still more with speech, as, for example, the lobed fibroma described by Partsch[12] in the fold between the upper lip and the alveolar process. Such large fibrous proliferations require operative interference more frequently than the above-mentioned smaller growths.

These fibromata situated near the teeth have been included in the large group known as epulis. But since the name really has only a topographical significance it is well to speak always of an epulis fibromatosa.

The growths are more characteristically tumors which spring from the periosteum of the alveolar process and from the inner walls of the alveoli and which, generally as the result of calcification and the deposition of bone, possess great hardness. They sometimes have pedicles, sometimes are sessile with a broad base, and may attain a considerable size and crowd the teeth before them out of the alveoli. In these tumors, as in all which are situated upon the margins of the alveoli, superficial injuries during mastication are common, and considerable hemorrhages and extensive ulcerations sometimes occur. Being benign growths, they are characterized by their slow development, which is generally unattended with pain and by the relatively slight disturbances of function which they cause, even when of considerable size.

But the fibromata which proceed from the body of the jaw itself, especially of the lower jaw, produce much greater disturbances. They also are distinguished by their hardness and by the frequent presence of calcification and ossification, and, growing slowly with the production of little discomfort, they may attain a colossal size. On account of their large size operation for their removal is not rarely difficult, but can generally be completed without great injury to the surrounding tissues.

Endosteal forms, originating in the interior of the bone, occur es-

pecially upon the lower jaw. They gradually swell the bone to a considerable thickness, then often break through the thinned shell of bone in places, push the periosteum before them and excite in it the formation of bone so that sometimes the entire tumor, sometimes only portions of it, are covered with a thin lamella of bone. This condition may cause the well-known phenomenon of "parchment crackling," but this disappears gradually as the tumor increases in size. Both forms of fibromata are often, and the larger tumors even usually, not sharply defined with respect to the periosteum or bone. But there occur in the interior of the bone, especially of the lower jaw, some completely encapsulated fibromata which are brought into genetic connection with the teeth germs.

All fibromata, especially the larger fibromata of the jaw, may occasionally present metamorphoses into cartilaginous or myxomatous tissue. They may also recur, and the recurrent tumors may exhibit the characteristics of sarcoma.

In the upper jaw the fibromata may make their way into the antrum of Highmore, find space for development in this and fill it completely.

From a malignant tumor fibroma is distinguished, above all, by its always very slow growth. It may be more easily confounded with chondroma and osteoma, but the latter is distinguished from fibroma by its stony hardness, the former by its nodular structure, the fibroma having a smooth surface. The diagnosis may, however, be very difficult, but is of slight importance since the treatment is the same in all these tumors.

Chondroma, Osteoma.

Cartilaginous tumors occur quite rarely upon the jaws, but present the same characteristics with respect to their location and development as the periosteal and endosteal fibromata. They also may reach an enormous size and they proliferate more freely than the fibromata through the bone and into neighboring tissues and cavities. Notwithstanding this and the fact that they form recurrences, they are in themselves of a benign character. Diagnostically they are distinguished by their peculiar hardness and their lobed nodular structure. The extirpation of extensive enchondromata may present considerable difficulty and may leave large cavities.

Osteomata are also found upon the jaws. On the lower jaw osteomata are met with which are encapsulated and quite separate from the remainder of the bone. These are thought to be connected with the processes of evolution of the teeth. But the majority of these tumors pass directly into the surrounding bone and are only distin-

guished from it by their greater hardness (osteoma eburneum). Within the mouth such tumors are found only upon the lower jaw.

There also occur exostoses upon the alveolar process, for the most part due to conditions of inflammatory irritation of the teeth, and generally in youthful individuals.

The peculiar form of enlargement of bone, which has been named leontiasis ossea, sometimes is more highly developed on the jaws, especially the lower jaw, than elsewhere. But as an affection of great rarity and one incapable of cure, it is of little practical importance.

The mixed tumors of the connective-tissue series which are more closely allied to sarcoma will be considered in connection with it.

Cysts.

Tumors on and about the jaws which contain fluid owe their origin, in great part, to disturbances in the evolution of the teeth.

One group of these cysts represents cystic degenerations of normal, or supernumerary embyronic tooth-sockets. These develop only during the period of dentition, but may especially in case of the wisdom teeth produce well-marked symptoms in later years, even as late as the third decade. These are the follicular cysts of Magitot.

The second group, the so-called periosteal (Magitot), or, more correctly, periodontal, or root cysts (Partsch[*]), arise from epithelial elements belonging to the root sheath (which is analogous to the enamel epithelium) left behind during the development of the root. From these residues are developed the "radicular fungosities," consisting in part of a purulent fluid, in part of firm granulation tissue, which are so often found, during the extraction of a tooth, adhering to the apex of the root. From these same aggregations of cells, which correspond to the epidermoid epithelium, may also be formed cysts, especially on the upper jaw. These cysts may attain a very considerable size, especially if they penetrate into the maxillary sinus. They first swell the bone, then cause its absorption, and may then, like other endosteal tumors, yield the "parchment crackling."

Inflammation may be excited by organisms which enter by way of the teeth and change the usually clear, yellowish, fluid or jelly-like contents into purulent or puriform fluid.

The follicular cysts are much rarer, and may occur at some distance from the alveolar process, sometimes enclosed deeply within the body of the jaw. They have been observed on the palate bone and even in the nose and the orbits. They always contain in their

walls imperfect or tolerably well-developed teeth. Their growth is very slow and, as a rule, painless. When they are deeply situated the diagnosis may be quite difficult. There are rare and peculiar cases in which cystic degenerations of strayed embryonic teeth occur in different parts of the jaw, as, for example, in the case of Hildebrand[91] in which there were about one hundred and fifty teeth in the upper jaw.

Allied to these unilocular are the multilocular cysts, which are very rare and with regard to the origin of which no certainty has as yet been reached. Becker's supposition[92] that they also are derived from the above-mentioned "epithelial residues" is not improbable.

The clinical symptoms of all these cysts first appear at a late stage when the tumor in its slow growth has attained a considerable size. Sometimes violent neuralgic pains, which often induce the patient to have even sound teeth extracted, long precede other manifestations. At a later time the tumors reveal their presence by considerable swellings of the bones of the jaw. The symptom of parchment crackling is often particularly distinct in them.

Dermoid cysts have been found in the lower jaw in quite isolated cases. Mikulicz[93] refers their origin to displaced epidermal germs of the period of the first formation of the teeth embryos. They contain plates of cholesterin, flattened cornified epithelium, and hairs. The structure of their walls is analogous to that of other dermoid cysts. The histological diagnosis is hardly possible without an operation.

Sarcoma and Mixed Tumors.

Sarcoma and carcinoma occur with about equal frequency on the jaws, and together they constitute the majority of the tumors of that region.

But it must not be supposed that all sarcomata are highly malignant. On the contrary, the sarcomata of the alveolar process have indeed a great tendency to recur locally, but form metastases infrequently.

By far the greatest number of these sarcomata of the edges of the jaw belong to the class designated by Virchow as giant-celled sarcomata and present the classical type of these tumors. They belong in general to the period of youth, especially the second decade. Their point of origin is given differently by different authorities, the majority supposing it to be from the deeper layer of the periosteum in the space between two alveoli. While the tumors are small they are often covered by an envelope of periosteum which partly ossifies and thus forms a shell about the tumor. In the tumor itself there

are round trabeculæ of bone almost without exception reticularly disposed between which lies the soft and very vascular tissue formed of spindle-shaped and round cells. Scattered through this, but especially in layers on the trabeculæ, are found the giant cells and large and generally much pigmented masses of protoplasm, with a varying but usually considerable number of small and irregularly disposed nuclei.

The origin of these giant cells is a question which has been much discussed since the writings of Robin and Virchow were published,

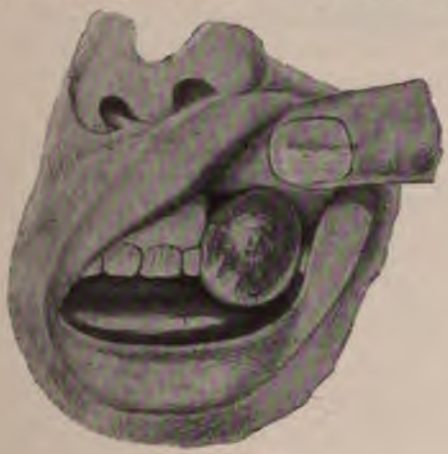

FIG. 15.—Epulis.

This figure represents a giant-celled epulis from a girl fifteen years of age. The tumor was first noticed a year before, when it was of the size of a pea. Three months later it was as large as a hazelnut. It was cut off, but soon grew again to its original size. One month before the picture was taken it had begun to grow more rapidly. The tumor is spherical, larger than a hazelnut, is of a firm consistence and in some places covered with pale epithelium, in others with dark red granulations. It is attached to the gums by a very short pedicle, 5 to 6 mm. in thickness. The teeth are intact. It was removed together with the subjacent periosteum of the alveolar process. It was found to be a typical giant-celled sarcoma. (From Mikulicz' Atlas, Plate XII., Fig. 1.)

but which is not yet settled. This form of cells occurs here and there in other fibrous tumors of the jaw, but not so regularly and typically. They have been supposed to be connected with the formation of bone, with its destruction and with the blood-vessels. There is much to be said for and against all these explanations, but the question cannot properly be discussed here.

It is, at all events, noteworthy that they are found especially in the tumors of the alveolar process and that quite similar tumors are occasionally developed from the wall of a root cyst (Partsch[89]). Perhaps in the case of Trendelenburg, cited by Becker, a similar phenomenon occurred in a multilocular cyst.

Clinically, as has been said, these giant-celled sarcomata, the

typical tumors of the alveolar process, *epulis sarcomatosa*, are quite benign. They may, though the patients rarely permit it, attain a considerable size and destroy much tissue. As a rule, however, they are operated upon at an early period, and do not recur very readily—at least, notwithstanding the statements in the text-books, such recurrence is rarely seen at the clinic. Small giant-celled sarcomata often have pedicles which are generally thick, and they are very easily removed. For larger tumors considerable resections of the alveolar process may be necessary.

The annoyances caused by the sarcomatous epulis are, as a rule, not insignificant. The tumor is easily injured during mastication and extensive ulcerations then sometimes form. This is particularly true of the larger tumors, which generally spread over the alveolar process like a broad cap and push the teeth out of their sockets. After this protection is gone, the tumors are exposed to all sorts of traumatisms and often become the seat of widespread ichorous ulcerations. Such large tumors are generally found in patients in the fourth and fifth decades.

Unmixed spindle-celled and round-celled sarcomata of the alveolar process are much rarer. They are similar to the giant-celled sarcomata in the symptoms which they produce, but are perhaps of a more malignant character.

Sarcoma of the body of the jaws is more frequently endosteal than periosteal. On the lower jaw the spindle-celled sarcomata preponderate, on the upper the much more malignant round-celled sarcomata which are among the most dangerous of tumors. Rapid growth and indiscriminate destruction of the surrounding tissues characterize them only too distinctly. They are difficult to distinguish from carcinomata of the upper jaw, which originate in the antrum of Highmore. That is, however, of no especial practical interest. Early and extensive resection of the upper jaw is for both the only hope, and it too often proves deceptive.

The central sarcomata of the lower jaw frequently cause very violent neuralgic pains from pressure on the inferior dental nerve. If on that account teeth are extracted, the tumor often proliferates through their alveoli and assures the often hitherto difficult diagnosis. But, in any case, it gradually breaks through somewhere on the outer or inner surface of the jaw and is then recognized as a harder or softer tumor which yields to pressure, but not in the same way as cysts.

Sarcoma of either the upper or lower jaw demands always extensive, generally complete, resection of the half of the jaw affected, often also of other bony parts.

The variously composed mixed tumors, chondromyxoma, myxosarcoma, myxochondrosarcoma, etc., are clinically closely allied to the sarcomata. Their names give the most important facts as to their structure which may sometimes be extraordinarily complicated. Through a more or less extensive formation of bone both their names and their structure may be still further complicated.

Clinically they may, in general, be regarded as rather malignant tumors, though with only a moderate tendency to form metastases. In their appearance, their course, and their diagnostic characteristics they are quite analogous to sarcoma, and they must also receive precisely the same operative treatment.

Carcinoma.

The carcinoma which occurs most frequently on the jaw is the flat-celled epithelial cancer of the alveolar process, the typical cornifying cancroid. It makes its appearance in just the same way as in other parts of the mouth, ulcerates quickly and extensively on account of its exposed situation, and may possibly be confounded with sarcoma of the alveolar process. But it seldom forms such extensive fungous masses as the latter, and is rather a flat ulcer with much thickened edges and all the characteristic peculiarities of cancerous ulcerations. As a rule, too, it attacks individuals more advanced in years than does sarcoma.

Central cancers of the jaw also occur. They are flat-celled epithelial cancers, like those last mentioned. They are quite rarely met with and only upon the lower jaw.[84]

They originate from the paradental epithelial residues. A histological diagnosis of these tumors is probably never possible before removal, unless they have broken through into the mouth.

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DISEASES OF THE INTESTINES.

(EXCLUDING INFECTIOUS DISEASES, PARASITES, AND HERNIA.)

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DISEASES OF THE INTESTINES.

GENERAL THERAPY.

It may be well to precede the systematic discussion of the diseases of the intestines by a general survey of the methods of treatment applicable to these morbid conditions, since these methods group themselves according to a moderately small number of indications which present themselves repeatedly in the special diseases of the intestinal tract. We have in this connection to deal chiefly with those agents which are directed against the two prominent symptoms of all intestinal diseases, namely, constipation and diarrhœa. Of course, every indication is not fulfilled by these remedies alone, but the other therapeutic measures, which are perhaps necessary—for example, the antiphlogistic, astringent, sedative, or analgesic remedies—do not bear a specific character, but are subject to the generally accepted principles regarding their application. The methods of treatment of the intestinal diseases may be divided into the three large groups of dietetic, mechanical, and medicinal.

Dietetic Measures.

The functions which the intestine has to fulfil towards the chyme, which is brought to it from the stomach, are exceedingly manifold, and only by the prompt co-operation of the various factors of a mechanical or kinetic and chemical or bacteriological nature, which come into play during its long and labyrinthine course from the pylorus to the anus, can justice be done to the normal process of intestinal digestion. As soon as one of these factors is wanting or acts ineffectively, the entire relation of the intestine to its contents is disturbed. One of the problems which is necessary for the treatment of such disturbances, and frequently the most important one, is to combat this mischief as much as possible by a judicious regulation of the ingesta.

It is to be regretted in the greatest measure that we are so little acquainted with the finer processes of such disturbances and their clinical symptoms; at the best, the fact only but not the nature of the individual processes from which these result can be recognized by us.

At the same time we may be permitted to set up certain dietary rules which depend partly on the composition of the various foods, that is, upon their specific character, partly on the manner of their preparation, and partly upon their physical properties. Accordingly food and drink may be classified in the following manner:

Laxative Foods.—Articles of diet which promote the intestinal movements by the products contained in them, or which are formed from them in the intestine, usually organic acids and gases. To this group belong cold water, cold carbonated beverages, most fruits (both raw and cooked), and fruit juices, so long as they do not contain astrigent substances such, for example, as are found in the bilberry (vaccinium myrtillus). In these cases it is the malic, tartaric, or citric acid of the fruit which in most individuals promotes intestinal peristalsis. The proportion of the contained acid varies in different fruits: plums, for example, contain as high as 1.27 per cent. of free acid, peaches as high as 1.58 per cent., strawberries 1.6 per cent., gooseberries 1.36 per cent., while pears only contain 0.07 per cent., and apples 0.53 to 1.0 per cent. Nearly all fruits and vegetables, with the exception of those belonging to the bulbous or leguminous class and cereals, provided they contain an abundance of water, as, for example, melons, cucumbers, tomatoes, pumpkins, and the different varieties of cabbage, promote peristaltic action by the formation of acid or gaseous products. The same effect is produced by fresh not yet fully fermented beer, cider, sour milk, kefir, unripe fruit, high game, and the like. We must, however, take cognizance of many individual idiosyncrasies in the action of these substances. Thus the juice of the grape acts on some persons rather in a constipating way than as a laxative, but its action manifests itself in the latter way whenever there is disease of the intestine which has a tendency to increase the number of intestinal evacuations. The same may also be said of the different varieties of sugar, such as candy, syrups, honey, apple sauce, and the like, which are absorbed in a healthy stomach as is well known, but which undergo fermentative changes in cases of disorders of the stomach, and act as laxatives through the formation of lactic or carbonic acid.

Constipating Foods.—Foods which act so as to retard intestinal peristalsis in any other than a mechanical way are very few in number. They are such as contain a large proportion of astringent substances, particularly tannic acid. Among these are usually classed dried bilberries (although I have never found any reference to the amount of tannic acid they contain); also those wines which contain tannic acid, particularly French red wines; tea, which contains ten to twenty per cent. of tannic acid, especially when drawn for some time;

cocoa, which may contain as much as six per cent. of an astringent principle; the different acorn preparations, as acorn coffee and acorn cocoa (containing about five per cent. of tannic acid). In how far solution of egg albumin, scraped raw meat, scraped raw ham, veal soup with rice, also rice water, rice flour, sago, tapioca, which are all practically approved foods for arresting diarrhœa, act medicinally or only benefit by furnishing the irritated intestinal canal with non-irritating food must remain an open question.

There is another group of food-stuffs which certainly act in a constipating manner only by their consistence, inasmuch as they contain but little water; such are the dried cereals, the leguminosæ or their preparations, such as rice, thick starch gruel, buckwheat, maize, millet, chestnuts, macaroni, nudels, all those which readily part with their water and leave a voluminous residue, as potatoes, carrots, cauliflower, spinach, asparagus, and the like. Alimentary substances of this kind also act quite differently in different individuals, so that one person may take large quantities of them without experiencing any particular effect, while a comparatively much smaller quantity may produce the desired result in others.

Midway between these representatives of both extremes there are many food-stuffs for which as a general rule no specific effect on the intestine can be claimed. To these substances belong, above all, meats and fish of various kinds, if served without seasoning, particularly in the form of jellies or purées; eggs and their preparations; also well-baked bread of wheat or rye flour, and pastry made from the same, as zwieback, cakes, and the like; also fats, provided they are taken in small quantities and no specific idiosyncrasy exists in regard to them. There is sometimes an idiosyncrasy in regard to fat, as in the case of a man whom I knew who had but to eat a trace of butter to provoke one or more stools. In this group milk may be included, although it really belongs in a class apart. Most persons may partake of large quantities of milk, as high as three to four pints during the day, without experiencing any particular influence on the intestinal function, the only effect being that the stools are lighter in color; others become markedly constipated, even after taking small quantities of milk, so that if the continuation of this diet for any reason seems desirable a slight laxative must be ordered to keep the bowels regular.

Latterly, an extraordinary benefit has been hoped for in diseases of the intestines by the administration of sterilized milk, and an asepsis of the intestinal canal in contra-distinction to its antisepsis has been spoken of. The latter has, however, not yet been accomplished, as is shown by the experiments of different observers (Bouchard,

Stein, Shiller, Albu, and others). It is, however, easy to see that such a recommendation, in so far as it has not in view the destruction of the pathogenic germs, such as tubercle and typhoid bacilli, which are sometimes in the milk, is based rather on a preconceived opinion and cannot be made of much practical utility. For the intestine, particularly in pathological conditions, contains such a wealth of the most various kinds of bacteria (of which Mannaberg alone enumerates no less than fourteen kinds of bacilli, nine of cocci, and four varieties of hyphomycetes) that the few microbes which might possibly be introduced by well-boiled milk can be of no consequence whatever. It is furthermore not known that sterilized milk is an exceptionally poor culture medium for the intestinal bacteria, and could thus be of any use in promoting asepsis. The idea of making the intestine perfectly free from germs, through the administration of purgatives or of irrigations with antiseptic solutions, would hardly be entertained seriously by even the most enthusiastic disciple of intestinal antisepsis, although putrefaction of the intestinal contents might be somewhat moderated by this treatment. Macfadyen and Nencki [1] reported, in proof of the persistence of these intestinal bacteria, the case of a woman with an intestinal fistula, in whom, after she had not had an evacuation from the rectum for more than two months, numerous cocci and bacilli of putrefaction were found in the large intestine, although the latter had been irrigated with a sterilized salt solution. We use sterilized milk, and rightly too, for an altogether different reason, namely, to protect the organism, and particularly the susceptible one of the infant, from possible pathological germs. This has no connection at all, however, with any asepsis of the gastrointestinal tract; and doubtless the claim that the use of sterilized milk in intestinal diseases is of service in diminishing bacterial dissemination and growth in the intestine will turn out to be one of the many exaggerations which are met with in this department of medicine.

There is another class of alimentary substances which act mechanically upon the intestinal mucous membrane. Cellular fibres which are not softened and dissolved by cooking or by the secretions of the stomach, husks, granules (for example of the poppy), the seeds of fruits, coarsely baked bread, raw cabbage, cresses, celery, and the like, are believed to further the peristaltic action by mechanical irritation of the intestinal wall from the pressure of the hard mass. I must confess that this view does not seem very plausible to me, and I believe that the coarse, undigested masses which are left in the intestine from the different articles of food in various quantities have a tendency rather to cause intestinal obstruction than to increase peristal-

tic action. According to my opinion, many of the previously enumerated substances act upon peristalsis through the formation of large amounts of lactic or oleic acid. Doubtless there may sometimes be an irritation mechanically produced by the presence of this mass of undigested matter, but if we consider that all these substances are enveloped within the intestine by mucus, and are thus made smooth and slippery (witness the passage of spicula of bone and similar objects through the intestine without causing any noticeable effect), then we are forced to regard with suspicion this theory of the mechanical action of food-stuffs.

The reverse may more likely be possible, namely, that the undigested cellular masses remain lying in the intestine, whose peristaltic power is not sufficient to move them onwards. The examination of old contents of the bowels, which have been removed by irrigation or are found at the autopsy in the pouches of the colon or in the lumen of the gut, proves that they consist in great measure of interwoven plant fibres, which are mixed with all kinds of different things, cellular detritus, bacteria, crystallized exudates, etc.

These remarks, which are in truth nothing more than a recapitulation of facts recognized by every layman, may suffice to give the thoughtful physician a hint regarding dietetic treatment. It is hardly necessary to mention that the question of diet plays an exceedingly important part in the treatment of intestinal diseases and that "rest and forbearance," above all, must be the first principles of rational treatment in these cases.

It has become more and more the fashion during late years to issue so-called diet cards for the most varied diseases of the stomach and intestinal tract, made out in the most painstaking manner for every day in the week, and so to produce a schedule which is to save the doctor all individual consideration, something after the manner of the homœopathic booklet, where one need only to search for the catch-word, cough, dropsy, painful limbs, or something similar and at once find named the proper tincture or pellet. It is the more impossible to regard such methods with favor as even the best prescription of this kind is, in a complicated case, made useless by the individual peculiarities of the patient, his "personal equation." The physician should rather obtain a minute knowledge of the composition of food-stuffs, their preparation, and their physiological action, so that he may be able at any moment to turn it to account. He should be competent to control his instrument like a good musician.

Mechanical Procedures.

Injections and Irrigations.

Simple as it may seem to administer an injection—that is, to throw a fluid into the bowel—there are certain measures to be observed at the same time which, though seemingly insignificant, still possess great practical importance. We make a distinction between a clysma and an irrigation. By the first-named process comparatively small quantities of watery, oily, or mucilaginous liquids—six to nine ounces or at the most one pint—are passed into the lower segment of the large intestine; with the irrigation larger quantities of water or watery solutions, one or more quarts, are passed up as high as possible into the bowel. It is of no consequence whether use is made for this purpose of a fountain syringe or a compressible bulb or other form of syringe. The essential thing is that the pressure under which the liquid is forced into the bowel be an even, slow, and not too great one, otherwise reflex muscular contractions of the intestines may be set up which would lead to a rapid expulsion of the injected liquid. The latter ought, so to speak, to creep into the bowel, and should not be forced into it by shocks. Of still greater importance is the construction of the delivery tube which is passed into the rectum. Short tips of horn or gutta-percha are especially to be avoided, for in the first place they do not enter deeply enough into the bowel to pass the closed internal sphincter, and furthermore, when unskilfully handled, particularly when the patient is restless and struggling, they may give rise to some lesion, which might produce an inflammatory and suppurative process in the cellular tissue. Some years ago I saw a patient die from the effects of a periproctitis which had been caused by an unskilled nurse during the giving of an enema. The anal tube should consist of soft, vulcanized rubber, about 35 to 40 cm. (14 to 16 inches) long, of the thickness of the little finger, having at its extremity one large, and on its sides a number of smaller openings, and should of course be well oiled before being introduced. It is not at all necessary to touch the patient himself for this purpose; it is only necessary, holding the tube as a pen is held, to push it with a slightly rotary movement through the anus into the bowel, when it will be possible, under normal conditions, to pass it up its full length without any hindrance, making sure thereby that it has passed beyond the third sphincter. It may happen that the tube will curl on itself in the ampulla or become caught in the crescent-shaped duplication, which in some individuals juts out, like a great ledge, at the junction of the sigmoid flexure and descending colon. This will be made evi-

dent by the distinct feeling that the tube has deviated from the straight line either to the right or left. It will only be necessary then to withdraw it slightly and push it on again in the right direction in order to achieve success.

It is quite a different matter when the lower part of the bowel is filled with stone-like, hardened fæces, so that the tube either cannot be passed at all or only for a short distance. In this case the finger must be used to get rid of the fæcal accumulations after having crushed them in the bowel by pressing against the sacrum, a procedure which is as disagreeable to the physician as it is frequently painful to the patient. Should the intestine be filled with tough and soft masses, the perforations in the anal tube become readily occluded; this may, however usually be avoided, by allowing the irrigating fluid to pass into the bowel under light pressure during the introduction of the tube. Small injections are to be made with the patient lying on his back or side; high enemata are best administered in the knee-elbow position, for the reason that this position of the patient facilitates the flow.

The question as to how far up irrigations may be made to enter the intestine, and to what extent the valvula Bauhini is permeable to liquids and gases, has been extensively discussed in the literature on the subject.[2] Simon[3] reported some years ago the case of a patient suffering from a fæcal fistula which was situated above the ileocæcal valve, in which the water which was injected into the rectum rapidly passed through the whole length of the large intestine and flowed out through the fistula. In experiments on the dead body it is possible in a great number of cases, though not always, to overcome the pressure of the valve. That air which is blown into the rectum passes onwards to the small intestine can easily be demonstrated in tolerant patients.

According to some recent experiments by Grützner,[4] it has seemed possible that clysmata, especially when solutions of common salt are used, might pass up above the valve, even into the stomach, by an antiperistaltic action of the small intestine.

Dauber[5] avoided a source of error which invalidated the experiments of Grützner (namely, that the animal licked its anus and so got some of the salt solution in its stomach) and showed that there was no such backward flow of the injected fluid. The one experiment of this kind which Grützner made in the human subject with apparent success can easily be disproved; he had not thoroughly cleansed out the stomach of the person experimented upon, and discovered remnants of starch in it which he attributed to a starch clysma that he believed had passed through the intestine into the stomach.

Irrigations may be used, first for the purpose of causing a stool,

or secondly to produce a local effect on the mucous membrane of the intestinal canal, and lastly they may have a purely mechanical action. This is not the place to enter more minutely into this subject; it will rather be reserved for discussion when we come to the individual diseases of the intestines. It is not unusual, during high irrigation of the bowel, to find that the patient can retain only a comparatively small quantity of water, either because the sphincter muscle relaxes or because the *vis à tergo* is too great. Under these circumstances the favorite plan, to press a firm tampon of cloth or cotton batting against the anus or to press the anal fold against the tube with the finger, is, according to my experience, not very satisfactory. Better results are frequently obtained if a rubber ball, which may be filled with air or water and which is perforated in the centre by the anal tube, is passed folded into the ampulla and there filled (Quincke,[8] Boas[7]). Such a contrivance which will press against the pelvic wall or the perineum like a ball valve will keep the water well back, but the pain produced by it generally becomes at once so intolerable to the patient that the ball, and with it the fluid which has been retained by it, must be again removed.

Another plan of distending the intestines is by the insufflation of air, which may be done with any bulb syringe. It should, however, be attempted only after a thorough irrigation of the intestinal canal. It may give very valuable information concerning the position, the size, probable contractions, adhesions, or occlusions, tumors, abnormal communications of the intestine with the stomach, the bladder, etc.

In stenosis of the intestine the air will escape when only small quantities have been insufflated. This is a pretty sure sign of an intestinal stenosis which is not situated very far above the anus, for the intestines under normal conditions, or otherwise, are remarkably tolerant of insufflated air, at least to the extent that the air is not again expelled, although pretty severe pain may be caused by strong insufflation, earlier in one person than in another, according to the size and tolerance of the intestine. Should auscultation be practised over the abdominal parietes during insufflation, we can convince ourselves of the perviousness of the intestines by the cooing sounds which extend over the abdomen, just as we may see the gradually filling coils of intestine through abdominal walls, provided they are not covered with too much fat and are not too tense. It has also been proposed to fill the intestine with carbonic-acid gas instead of air, causing its development within the intestine (v. Ziemssen[9]), or to make use of liquids containing gas or carbonic acid (Schmetter[6]). There is no particular advantage connected with this method, and it may result in

a great irritation of the intestines, with resulting paresis of the intestinal muscular fibres.

Massage, Gymnastic Exercises, and Sports.

Massage should be practised only by well-trained and experienced persons. The "wild massage" which is now frequently practised by persons who seek thereby to obtain an easy and independent occupation very often produces more harm than good. On the other hand, it is altogether unreasonable to expect that physicians who have not occupied themselves specially with massage should apply the same to their patients. Massage is an art which must be acquired and cannot be practised by every individual, if for no other reason, on account of the muscular effort necessary. The physician ought, however, to be so far acquainted with the method as to be able to give the masseur general directions for treatment, and to control his management of the case.

Gymnastic exercises and sports, when they can be practised, are extraordinarily well adapted to stimulate and strengthen the abdominal as well as the intestinal muscular fibres. Rowing, particularly in boats with sliding seats, has given me lasting results in cases of men suffering from chronic intestinal torpidity, while horseback riding, for example, as well as cycling is usually effective only so long as the body has not habituated itself to that kind of exercise.

Electricity.

It seems to be a fact that electricity will excite movements of the intestines, that is, true peristaltic action, and not merely a localized contraction of one portion only; and the experiments of Ziemssen, Baudet, Larrat, Schillbach,[10] Erb, and many others, have shown in animals as well as in man that local contractions and peristaltic waves may be induced in the intestine by this agent. For this purpose the percutaneous method may be used, in which one electrode is placed in contact with a so-called indifferent point, and the other electrode is passed successively over different portions of the abdominal wall; or in place of the movable electrode a cylindriform or a very large disc-shaped electrode may be used; or one pole may be passed into the rectum or even, as I have recommended, one electrode may be passed into the stomach and the other one into the bowel. For the latter purpose use is best made of a rectal tube of soft rubber, as described above, through the centre of which a spiral wire the size of a knitting needle is passed, the distal extremity of which is supplied with a suitable contrivance for screwing on the electrode. This tube should be connected at the same time with a lateral short tube, to

which the irrigator may be attached. Through this latter tube the intestine, *i.e.*, the ampulla of the sigmoid flexure, is filled, after the passage of the tube with a one-per-cent. solution of common salt, after which the current is turned on. It is always necessary to precede the application of electricity by an irrigation for the purpose of cleansing the bowel.

It is altogether wrong to expect an immediate influence on the intestinal function from electrical treatment. When this is the case the application has acted simply as a clysma. The object of the electric current is rather to strengthen the muscular fibres of the intestine, and for this purpose the employment of electricity should be continued for a long period, as is the case in atrophic or paretic voluntary muscles. These seemingly well-founded views have, however, latterly been opposed by Moritz[11] and Meltzer,[12] the first, however, only in relation to the stomach. Meltzer comes to the conclusion, from experiments which were, to be sure, performed by him only on rabbits and dogs, that the mucous membrane of the digestive tract opposes a strong resistance to the passage of the faradic current to the muscular fibres, and that this resistance is strongest in the mucous membrane of the stomach. The percutaneous as well as the direct faradization of the stomach and of the intestines is therefore powerless to produce contraction of these viscera. Meltzer's experiments, however, are burdened with many contradictions, and at all events it is certain that the practical application of electricity in man is praised by all authors, of whom only the latest, Goldschmidt, of the University Klinik of Munich, need be mentioned. Schillbach arrived at the conclusion that the action of the galvanic current is, as a rule, superior to that of a faradic current of equal strength, and stated that local contractions usually predominate at the cathode, and peristaltic waves, particularly those in an ascending direction, at the anode, although the direction of the current exerted no influence on intestinal contractions. Other authors, however, believe that the faradic current influences chiefly the functional action, while the galvanic current is more effective in the sensory sphere.

METHODS OF EXAMINATION.

Inspection.

Certain coarse changes of the abdominal organs can already be recognized by simple inspection of the patient. We can thus learn whether the abdomen is retracted or distended; whether the integument retains its normal condition or is unusually smooth and shin-

ing, or tense; if, particularly in women, a pendulous abdomen is present or the adipose tissue of the abdominal region is abnormally developed; whether, lastly, local prominences or depressions are present, and whether or not these change their position. Knowledge thus obtained gives us a hint upon which we can base our further investigations, but it does not by any means allow of diagnostic conclusions. Thus it would seem, for example, that in the case of boat-shaped retraction of the abdomen, which is, as a rule, caused by a contraction of the intestinal coils or by their descent into the false pelvis (enteroptosis), no ascites would be present, and yet I have at different times found under such conditions appreciable quantities of fluid in the abdominal cavity, and especially in cases of small pelves. So also, on the other hand, may a tumor be simulated by a localized contraction of a section of the rectus, transversus, or obliquus abdominis, or of the quadratus lumborum, whose true character can be determined only by placing the patient under the influence of an anæsthetic. However, these are trite remarks, which should be familiar to every practitioner. Inspection is only of decisive value when the mucous membrane of the large intestine can be directly brought into view, and this can only be done in the region of the rectum. It will depend on the nature of each case whether we confine our inspection to the lower part of the bowel, as far as it may be exposed by the finger or as the patient may protrude it by straining, or whether we apply one of the well-known specula of Ferguson, Sims, Allingham, or, more practical and simple although necessitating the presence of an assistant, of Cusco, as modified by Ricord, consisting of two separate, grooved blades. Very great corroboration of other diagnostic measures may be obtained by observing, in the case of relaxed or sunken abdominal walls, the progressive inflation of the intestinal coils with air introduced through the rectum, or in exceptional cases, by way of the stomach. In this manner we are frequently able to recognize with distinctness the seat of a stricture causing obstruction in the intestine or to determine the mobility of a tumor or its position in relation to its surroundings.

Percussion.

Percussion of the abdominal walls also, can only be pursued with advantage, as far as regards the intestine, after inflating the large intestine with air, inasmuch as the relation of the latter to the small intestine and to probable new growths or dislocated organs of the abdominal cavity, can only then be positively determined. Of course, we might for such a purpose distend the bowel with water, but apart

from the fact that this would be more troublesome, we would at the same time lose the advantage of distinguishing solid formations by the percussion sound, as could be done if the intestine contained gas.

Auscultation.

Noises are produced in the intestine by its own movements or occasionally by certain manipulations of the individual (rapid, spasmodic contractions of the abdominal muscles), and are either audible at a distance or may be heard by means of the stethoscope applied to the abdominal wall. They may also be produced passively whenever large quantities of air or fluid are present in the intestine. They are particularly heard in three places: in the ileocæcal region; in the region of the navel, that is in the centre of the transverse line, connecting the anterior superior spines of the ilia; and in the left inguinal region. They are most easily produced by quick taps of the fingers of the hand applied flat to the region mentioned. I have found particularly characteristic of an abnormal condition of the intestine a swashing or splashing noise, sounding as though air and water were forced through a narrow space in the ileocæcal region. I have very rarely found it in this location in healthy persons, but quite frequently in those who were suffering with chronic intestinal indigestion. The descending colon may also be the seat of these sounds, particularly if the bowel is abnormally dilated and atonic. These sounds may be easily produced, according to Boas, by passing a small quantity of water, say 150-200 c.c., into this part of the bowel. In healthy persons this sound can usually not be so produced, but this statement is by no means without exception, as Boas thinks. As regards the noises in the transverse colon it is necessary to employ certain precautionary measures, such as a previous emptying of the stomach, in order to prevent confusion with the gastric splashing sounds. According to my experience, these sounds are met with in proportionately greater frequency than any others in healthy individuals.

By a *direct auscultation* of the intestines it is possible to convince one's self chiefly of the presence of peristaltic movements or of a paralysis of certain portions of the intestine. Auscultation during the passage of air forced in by way of the rectum will give us an idea of the permeability of the lower portion of the intestine as far as the ileocæcal valve, occasionally even of that of the small intestine. Lastly friction sounds in the case of inflammatory exudates, and metallic tinkling or amphoric sounds may be heard either directly or on percussion. The latter may be caused by the presence of air in the

abdominal cavity, but just as frequently they are produced within the intestines by a high tension of their walls, so that caution must be exercised in their interpretation.

Palpation.

This method of examination is without doubt the most important. To be able to perform palpation well and with certainty is an art which can only be acquired by constant practice at the bedside. It is not only necessary to possess great certainty in the interpretation of tactile sensations and the greatest delicacy of touch, but also a perfect knowledge of all the conditions with which the examiner may be confronted during his examination. The problem of palpation consists in the proper combination of a sense impression and an abstract train of thought. The examiner should, so to speak, be able to look into the abdominal cavity with the tips of his fingers. A condition of success is that the abdominal parietes of the patient are not too thick and his muscles not too tensely contracted. The first condition is frequently more rapidly brought about by the disease itself than the patient himself is pleased to see. Frequently it is possible to demonstrate during the progressive emaciation of the patient tumors or other abnormalities of the abdomen, which could in no way have been discovered earlier. The contraction of the abdominal muscles can be overcome in most individuals by placing the patient on the back, with head raised, legs bent at the groin and mouth open; the tension may sometimes also be removed by diverting the patient's attention, or by raising the pelvis by means of a pillow placed under it. In certain cases, in order to facilitate the palpation of tumors, etc., in the abdominal cavity, the patient may be placed in a warm bath which causes a relaxation of the abdominal muscles (v. Chlapowski). In other cases nothing can be done but to bring the patient fully under the influence of an anæsthetic. This should always be done in doubtful cases in which the conditions demand an accurate diagnosis. Only a few days before writing these lines I examined a case in which the attendant, an elderly and experienced physician and myself, thought that we had discovered a distinct tumor in the left hypochondriac region, but under chloroform narcosis it was shown that we had to deal with a circumscribed contraction of the transversus abdominis, which had made its appearance during palpation. In questionable cases palpation in the knee-elbow position should not be neglected, the patient being then examined from behind. In this position the parts lying normally deep in the pelvic cavity are thrown forwards, and may be easily palpated.

Palpation should under favorable circumstances disclose, first, the position of the individual portions of the intestines; second, the presence of abnormal conditions of the same; and, third, the relation of the other abdominal organs to the intestines.

That portion of the intestines which is distinguished by the relative constancy of its position, and by the facility of its demonstration, is the ileocæcal portion, which is found nearly without exception in the right iliac fossa. Under the favorable conditions mentioned above, a swelling is felt here, lying deep within and parallel to the rim of the pelvis, and in which, by reason of its contents of air and fluid, will be produced the various sounds above described. Under favorable conditions the appendix vermiformis may be felt lying towards the inner side and horizontally; this is particularly possible if there is any pathological enlargement, of which we shall speak in detail later on.

Edebohls[1] has written an article on the possibility of palpating the processus vermiformis. Of especial importance here is "McBurney's point," lying in the first third of a line passing from the anterior superior spine of the ilium to the umbilicus, and also the position of the right common and external iliac arteries, which lie in an imaginary line drawn from the umbilicus to the middle of Poupart's ligament; the beginning of the vermiform appendix is found somewhat outside of this line which crosses it. It goes without saying that the appendix can be felt only on deep and firm palpation when it is possible to press it against the rim of the pelvis, and Edebohls justly remarks: "Pressure deep enough to recognize distinctly the posterior abdominal wall, the pelvic brim, and the structures lying between them and the examining finger, forms the whole secret of success in the practice of palpation of the vermiform appendix."

A second portion of the intestine, which is also usually constant in its position, is the lower end of the large intestine, which, as a rule, is situated in the left iliac fossa. However, we meet here with a number of variations of position. The descending colon is secured normally by a short and firm mesentery to the posterior wall of the pelvis; but if this stretches as it may at times, the intestine falls over towards the median line, or may even lie to the right of the bladder or the uterus. Even greater variations are met with in the position of the transverse colon, which normally passes obliquely just above the umbilicus towards the arch of the ribs, but which may sink deeply down into the pelvis, or become U-shaped and form adhesions with the bladder or the uterus. In such a case it is possible to get an idea of the position of these organs only by insufflation of air or water through the rectum, for simple palpation or percussion will not

give us any definite information concerning its position. Thus it may be altogether impossible under some circumstances to differentiate the pancreas from the transverse colon by palpation, and the mistake has been made even by Glénard of confounding the two.

What we have said as to the variable position of the large intestine applies even more to the small intestine. As a general rule we may say that the small intestine lies in the middle of the abdomen in the umbilical region, but this is true only in a general sense and without distinction of the individual portions of the small intestine. When we reflect how difficult it is to get our bearings in abdominal operations, with the presenting intestinal coils before our eyes, we dare not promise ourselves too much from simple palpation of the abdominal wall.

We shall discuss the methods of detecting abnormal conditions of the intestines when we come to treat of the individual intestinal diseases.

The relation of the intestines to the solid abdominal organs, whether they be normally or abnormally situated, will also be discussed in a later section. Here we need only remark that abnormally situated organs or neoplasms of parts other than the intestines will, under the pressure of the intestines filled with air or water, return to the position which the organ normally occupies. Thus tumors of the kidneys or spleen will be pressed up under the diaphragm, tumors of the liver and of the stomach will be forced upwards, and those of the large omentum towards the front and downwards, while retroperitoneal tumors, tumors of the pancreas, of the spinal column, or of the pelvis will remain fixed.

Rectal Examination.

Of very great importance, however, is the exploration of the rectum by palpation and digital examination. This aid to the diagnosis of diseases of the intestine is made use of altogether too infrequently. Although every physician inspects the throat in diseases of the respiratory organs, the examination of the rectum in digestive or constitutional diseases is usually neglected by reason of a certain feeling of shrinking from this dark region, and I am cognizant of numerous cases in which in unexplained conditions of debility, after the patient had for a long time been treated for stomach and lung trouble, the simple passage of the finger into the bowel cleared up the diagnosis by revealing the presence of a carcinoma of the rectum. This examination should always be made while the patient is in different positions, that is to say, on the back, the side, and in the knee-elbow position,

whenever we do not arrive at a satisfactory conclusion after having employed a single method of examination detailed above. In this manner alone can the large intestine and its environment be palpated in every direction. It may also be necessary in women to make a vagiual examination as well as a bimanual rectal one. In certain cases, as when there is grave suspicion of a neoplasm situated high up, there is nothing else to do but to introduce the whole hand into the rectum after Simon's method. This is to be done under anæsthesia, and in its execution the examiner who is possessed of a very small hand with long fingers will have the advantage. The examination by sounds, whether of wax, of whalebone, or of ivory, which has been frequently advocated, does not give satisfactory results. Not only are we uncertain as to the nature of what the sound impinges against, but there is also danger that the point or the button of the sound may be caught in the folds of the intestine, so that we may not even reach the locality which we wish to explore.

Examination of the Fæces.

Necessary as a minute examination of the fæces may be for the recognition of certain pathological conditions within the intestine, and as little as it should be neglected in every serious disease of the intestine, yet the results are usually unsatisfactory, whether the question is one of the recognition of certain general types of disease or whether it is the diagnosis of individual affections. This is true of the macroscopic, but even more so of the microscopic and chemical examination. Here we obtain satisfactory and useful results only in the smallest number of cases. This examination may, however, give us information concerning the presence of parasites or of their eggs, of particles from tumors, of concretions, of bile pigments, or of certain specific bacilli, such as those of typhoid fever or of cholera. The presence of other abnormal admixures, as, for example, blood, mucus, pus, fat, or large quantities of undigested food, allows us to form a general conclusion as to the presence of a disease of the intestine, or perhaps even to recognize its nature, in so far as we have to do with certain general conditions, such as constipation or diarrhœa, and to determine the character of the latter; possibly also we may by this means be enabled to conclude as to the nature of certain functional disturbances, for example, deficient digestion of meat; but a definite knowledge of the more intimate causal lesions cannot be so obtained. I have examined the stools in many hundred cases of disturbed intestinal function, without the microscopical findings having brought me one step nearer to the diagnosis or to the indications for treatment.

This statement is not intended to underrate the value of the examination of the fæces, nor to advise its neglect, but in view of a certain fussiness which is so frequently met with, it may be permitted one of many years' experience to say that everything that may be learned from the examination of the fæces, particularly in the ordinary run of diseases, in by far the greater number of cases may be seen with the naked eye. Of course we must always in doubtful and difficult cases resort to the more delicate methods of examination, but it is unnecessary to impose upon the physician a minute and painstaking examination of the fæces in every case.

Normal Stools.

Even the normal stools, those of a person not suffering from any form of digestive disturbances, may vary greatly in shape and consistence as well as in their frequency. There are, as is well known, persons whose habit it is to have two or even three stools daily, while there are others in perfect health who perform this necessity normally only every second or third day, and a few "sensational" cases are on record in which the intestine rid itself of its contents only every eighth or tenth day. In like manner the consistence and color of the fæces may vary considerably within the bounds of good health, depending evidently on the nature of the food taken, as well as on the abundance of the secretion of bile, and on the length of its sojourn in the bowel. The different shades of light yellow (milk stools), grayish-brown, yellowish-brown, reddish-brown, to black or dark green may be seen in normal stools. On being exposed to the air the fæces become darker on the surface from progressive oxidation, and then appear to be of a lighter color interiorly. The dejections of young children, even after they have been taking mixed food for some time, are always much lighter colored than those of adults. The products of change of the pigments originating in the bile, bilirubin or biliverdin, which are converted by the putrefaction of the fæces into biliprasin and urobilin (hydrobilirubin), modify, according to the amount of each present, the color of the dejections.

The shape of the stools also varies. They may be solid or loose (not necessarily diarrhœal), smooth, or in layers with the impression on them of the sacculations in the colon, sausage-shaped, flat, ribbon-like, or round, without our being conscious of any noticeable disturbance or of any reason why one shape should be present at one time rather than at another. Some persons pass a soft stool year in and year out, and are conscious of a certain discomfort whenever the stool becomes firm.

Microscopical examination usually reveals a brownish, flaky, or

lumpy detritus, in which are found scattered intestinal epithelia, sparse, partly digested fibres of muscle, tendon, or elastic tissue, and, according to the vegetables eaten, plant cells, spiral fibres, and kernel-like formations. It stands to reason that the volume and nature of these remnants are altogether dependent on the digestibility of the food taken, and that when the diet consists of milk, eggs, very tender meats, very tender vegetables free from cellular tissue, the fæces will hardly, with normal digestion, contain any such constituents. It is remarkable that undigested starch granules are hardly ever found in the fæces. Of crystalline admixtures, neutral phosphate of lime, phosphate of magnesia, and carbonate or oxalate of lime may be mentioned. All these substances are usually present only in isolated specimens; the oxalates occur only after the ingestion of certain vegetables, as, for example, wood sorrel and spinach.

Micro-organisms are present in abundance in every stool, and bacteriological investigators have endeavored to isolate the single species and to examine them in regard to their specific action. Although the researches of Nothnagel, Bienstock, Mannaberg, Stahl, Escherich, and others,[14] have shown the presence of an exceedingly rich fauna of fungi and schizomycetes (with the bacterium coli at the head) in the intestine or rather in the fæcal masses, we have thus far learned only that their absolute number increases greatly under pathological conditions; but a positive relation of these bacteria to definite diseases, if we except the specific micro-organisms of typhoid fever, cholera, and tuberculosis, has not yet been proven. Mannaberg, the latest worker in this line, concludes the account of his investigations[15] with the following words: "From the observations hitherto made, we can with great probability conclude that the catarrhal diseases of the intestinal mucous membrane are not caused by specific micro-organisms, always of the same species in each case; it seems rather as if the numerous intestinal bacteria, whether constantly or casually present, possessed the capacity, under circumstances which are as yet unknown, to produce the diseases mentioned. Furthermore, it seems as if one and the same variety of bacteria may occasion at one time a slight, at another time a more severe affection of the intestine, or again cause no disturbance. Such, for example, is the case with the bacterium coli commune, which is tolerated without detriment by the majority of persons, and yet occasionally appears as the cause of catarrh of the large intestine, dysentery, cholera nostras, etc."

The odor of the fæces is caused, according to researches by Brieger, mainly by skatol, discovered by him, and in less degree by indol. The odor of rotten eggs is caused by the formation of large quantities of sulphuretted hydrogen and carburetted hydrogen gas, which may

also be present to a certain extent without any pathological disturbances. Free fat is present in every case, the amount varying in proportion to that which was contained in the food taken, since only a certain quantity of fatty matter can be digested and absorbed from the intestine. Occasionally its presence may be recognized macroscopically by a peculiar silvery appearance of the fæces. Microscopically it appears sometimes in the form of minute drops (in the fæces of infants fed on milk), or more frequently crystallized in the form of tufts or as solitary sharp needles. The greater portion, however, of the fat which is contained in the stools escapes detection by the eye, and can be discovered only by a chemical analysis, being present as an oleate, or as free oleic acid, or in the form of soap.

The percentage of water in the fæces is with a meat diet about sixty to sixty-five per cent., with a mixed diet about seventy-five per cent., and with a purely vegetable diet as high as eighty-five per cent.

The reaction of the normal fæces is neutral or weakly alkaline, but in the case of acid fermentation after the ingestion of carbohydrates in excess, it is acid.

Bile pigment and mucus are never present in the normal stools, at least not in appreciable quantities.

Pathological Stools.

Very liquid as well as firm, stony-hard dejections are pathological. The first form the *diarrhœal dejections*, which are caused by the too rapid passage of the contents of the small intestine through the large intestine, there being then not sufficient time for the normal consolidation of the stools through loss of water and complete digestion. Under these circumstances, therefore, the characteristic contents of the small intestine will be found in the evacuations. The percentage of water in the latter may be as high as eighty or ninety. The stools contain appreciable quantities of soluble albumin, at times also peptone (v. Jaksch), and bile pigments which can be detected by the well-known Gmelin reaction (addition of nitric acid, to which has been added a small portion of nitrous acid).* Starch granules are colored blue by iodine.

Mucus can either be recognized macroscopically in the form of

* The best way is to dilute the fæces if necessary with a little water to the consistence of a thin broth, filter, and, after drying the filtrate, pour a drop of the nitrosonitric acid mixture on it. At the margin of the drop the well-known green color will be seen. If a small quantity of the diarrhœal matter be mixed in a beaker with a concentrated aqueous solution of corrosive sublimate, the whole mass will become green in the presence of larger quantities of bile pigment, otherwise solitary particles only of the fæces become discolored. Urobilin produces a red coloration.

slimy, filamentous, vitreous, or jelly-like masses, or in bands or grains (sago grains), or may appear under the microscope as white, glistening, glassy, homogeneous, coherent large drops or islets, which contain in their interior crystals, epithelium, and bacteria. Mistaking these for vegetable food remnants, which may also appear in the form of granules, may be obviated by noting their microchemical reaction with iodine in solution of potassium iodide, which colors these substances blue.

In the diarrhœal stools we also frequently find abundant remnants of food, consisting of undigested muscular fibres, vegetable cells, starch granules, and fat crystals, and also, according to Leo, ferments which are capable of digesting albumin. The odor is usually very penetrating. This is particularly noticeable in children whose stools do not have any very marked odor in health.

In contrast to the diarrhœal stools the dejections in *constipation* are extraordinarily firm, at times so hard and dry that they fall noisily into the chamber as though they were stone. They are apt to have a rounded form, from their long detention in the large intestine, and vary in size from that of a hazelnut to that of an apple, the so-called sheep's dung. They have a dark color, and are occasionally covered with mucus and streaked with fresh blood. The latter comes either from the rupture of hemorrhoidal tumors, or from the tearing of the mucous membrane of the sphincter by the passage of the hard masses. If we introduce a finger into the anus in severe constipation, we find the ampulla filled as if with stones, and frequently experience the greatest difficulty in removing these masses. There are cases on record in which the removal of the indurated masses was possible only after an incision through the sphincter.

Microscopically we find a copious detritus of a brown or black color, usually numerous colorless or slightly tinged triple phosphates (phosphate of ammonium and magnesium crystallizing in the form of a coffin lid), or more sparse crystals of neutral phosphate of lime. Seldom do we meet with the rhomboidal plates of cholesterin, which are recognized by the fact that they are colored a reddish-brown to violet by dilute sulphuric acid (1:5), and become blue or green on the further addition of a solution of iodine. Needle-shaped crystals of fat, single and in the form of tufts, are frequently met with. Bile pigment cannot be detected. Undigested remnants of food are only sparsely present, owing to the long detention of the fæcal matter in the bowel. Epithelium from the mucous membrane, pus cells, and blood corpuscles, unless they come from the passage of the fæcal mass through the anus (in which case they are simply adherent to the external surface of the scybala and are but little changed), are greatly

changed, fatty, degenerated, shrunken, and hardly recognizable. The micro-organisms are numerous, in lively motion, but have no specific significance.

In addition to these typical peculiarities of the two great groups of unhealthy stools, we find under peculiar conditions certain changes of a positive and negative nature.

Fat in the Stools, Steatorrhœa.—An unusually large proportion of fat in the fæces is met with in cases of occlusion of the gall duct or in diseases of the absorbent apparatus of the intestine (mucous membrane, lymph vessels, glands of the mesentery). An acute enteritis, particularly catarrh of the small intestine in children, atrophy of the mucous membrane, amyloid degeneration, or tuberculosis, may be the cause of fatty stools. The presence of fat may in these cases be recognized macroscopically by the asbestos-like, glistening, tallowy character of the stool. Or the latter is studded with fat in the form of small whitish spots, or oil floats on the surface of diarrhœal stools. Microscopically it is seen in the form of abundant needles and tufts, glistening drops, or lumps. The slender, finely curved needles of the fatty acid crystals are readily distinguished from the shorter and thicker formations of similar shape which represent the fatty soaps, and which in contradistinction to the fatty acids are not soluble in ether.

Colorless, acholic stools are found in cases of occlusion of the bile ducts. They nearly always contain a large proportion of fat.

Certain pale, clayey stools which are observed when there is no occlusion of the bile duct, hence without the presence of jaundice, owe their color to the large proportion of fat they contain. If these stools are thoroughly treated with ether, they gradually assume a brownish color. There are cases, however, in which the proportion of fat is low, and still, in spite of the absence of icterus, an *acholic* stool is passed. The best explanation of this seems to be that, in place of bilirubin, a colorless product of its decomposition, the leuco-urobilin of Nencki, is formed in the intestine.

Abnormal color of the stool is caused by certain drugs. A yellow color is produced by rhubarb, senna, santonin, and gamboge; a black discoloration follows the administration of bismuth, iron, or manganese. The green color produced by calomel is due, as Hoppe-Seyler has shown, to the presence of unchanged biliverdin, not, as was formerly supposed, to the formation of sulphate of mercury. Wassilieff has shown that calomel by its antiseptic qualities protects a portion of the bile pigment from the effects of putrefaction, and from conversion into biliprasin and urobilin. The green color of the stools of little children is caused, according to Lesage, by a bacillus which produces a green pigment.

Blood may adhere to the fæces externally, either in streaks or in larger spots, in which case no difficulty is experienced in recognizing it. Or it may be intimately mixed with the stools and communicate to them, according to its amount and the decomposition to which it has been subjected during its sojourn in the intestine, a reddish or a blackish-brown tar-like appearance. Or lastly, it may be passed without any fæcal admixture in large clotted lumps or in a fluid state. If this be the case, it has either come from the lower portion of the intestine or it has been carried quickly downwards by a greatly increased peristalsis. In this case the proof of the presence of blood is not difficult. One look through the microscope or through the spectroscope is sufficient.

It is quite different when only a small quantity of blood is intimately mixed with the fæces, or when it has been decomposed by long detention in the intestine. The blood corpuscles are then as a rule so changed that they cannot be detected with certainty. The spectroscopic diagnosis is also impossible, because the hæmoglobin has been decomposed, or else there is not enough of it in the sample to give the hæmoglobin bands in the precipitate or in the filtered portion of the fæces. The familiar test of Schoenbein-Almen with tincture of guaiacum and turpentine or peroxide of hydrogen is by no means to be relied on, because other matter may be present in the fæces which might act as oxidizing agents on the tincture of guaiacum and produce a blue coloration. Teichmann's hæmin test with common salt and glacial acetic acid is reliable when positive results are obtained, that is, when the characteristic hæmin crystals are formed; this, however, is the case only when there is an acid reaction of the intestinal contents, otherwise irregular forms of crystals are obtained whose interpretation is often difficult. At the same time this test requires a certain amount of skill and practice.

The method recommended by Fr. Müller and Weber is the one which gives the best results. A portion of the fæces (rubbed up with water if necessary, and filtered) is mixed with a few cubic centimetres of glacial acetic acid and shaken up with ether. In case the ether should not settle down clear, a few drops of absolute alcohol are to be added. A brownish-red tint of the ether layer indicates the presence of acetate of hæmatin, *i.e.*, of blood. The guaiacol test may also be made with the ether extract. It may not be superfluous to warn the observer against mistaking blood which may have been swallowed with the food, or which has come from the respiratory tract, the œsophagus, or the stomach, for that coming from the intestinal mucous membrane.

Pus is only exceptionally recognizable macroscopically when there

is a discharge of large masses coming from or passing through the lower portion of the intestine. In cases of ulcers of the small intestine we occasionally, but very seldom, find small masses of a grayish-white color, which are made up of a mixture of pus cells and mucus. These confirm, according to Nothnagel, the diagnosis of ulcers of the intestine. Usually the leucocytes are changed to such a degree by the action of the intestinal secretions, putrefaction, and fatty degeneration that their identification may be difficult or impossible unless they are present in large aggregations.

Concretions.—There are occasionally found in the stools gall stones, fæcal calculi (coproliths), intestinal concretions (enteroliths), and foreign bodies.

The coproliths are much more frequently found in the intestine at autopsy than they are in the stools. They are caused by the stagnation of the intestinal contents in certain parts where there is even normally a retardation of the fæcal stream, that is in the cæcum or the appendix, in the sacculations of the colon, and in the rectum. They are of stony hardness, generally oblong, sausage-shaped, and may present concentric rings, like the trunk of a tree on section. They may reach an enormous size, occasioning occlusion of the gut, or even being taken for neoplasms.

Enteroliths are concretions which usually consist of a mixture of inorganic salts (lime, magnesia) and organic matter (portions of bone, hair, needles, seeds of fruits, eggs of parasites). They are lighter in color and smaller than the coproliths. To these also belong the rarely found stones of the pancreas. They are of a soft, crumbling consistence, rough surface, occasionally facetted, readily soluble in chloroform (?), and, according to Minnick, develop an aromatic odor when heated.

Foreign Bodies.—To these belong everything that is swallowed and passed again *per vias naturales*, such as pieces of bone, money, needles, marbles, artificial teeth, lead soldiers, etc. It is astonishing what tolerance the gastrointestinal canal has for such things, of which the most curious stories are related. Hair balls and concretions of shellac (after drinking furniture polish) also belong to this category.

For the detection of smaller concretions—larger ones are easily recognized—the stool should be passed through a sieve. I generally add some ether previously in order to overcome the bad odor somewhat.

INFLAMMATION OF THE INTESTINAL MUCOUS MEMBRANE.

Catarrh of the intestine, or enteritis, may be acute or chronic. It may occur either as a primary, independent disease—idiopathic enteritis—or as a secondary affection, a part or a sequel of numerous other morbid processes which run their course in the intestine or its vicinity, or of infectious and other chronic diseases—secondary symptomatic enteritis.

Etiology.

Acute intestinal catarrh is caused by either chemical or mechanical irritations which are brought to bear on the intestine. The chemical irritants consist of organic or inorganic matter; the latter is always introduced from without, the former is either introduced from without or is formed within the canal as a consequence of faulty intestinal digestion. Among the inorganic irritants are the metallic poisons, arsenic, corrosive sublimate, tartar emetic, and the caustic alkalies; among the organic are the drastic cathartics, colocynth, and croton oil, carbolic acid, and lastly and mainly, alimentary substances which have been ingested in too large quantities or in a spoiled condition, such as tainted meat, fish, sausage, oysters, mussels, fruit, milk, bad water, sour beer, etc. These food substances, in so far as no direct pathogenic infection may be present, act by undergoing in the intestinal canal abnormal disintegration and fermentation, the products of which irritate the mucous membrane and produce an inflammatory condition. It is hardly necessary to mention that, according to our present views, we have in these cases always to do with a bacterial cause, whether it be through an excessive growth of the common organisms of fermentation, the entrance of pathogenic germs, or the transformation of normal bacteria into those of a pathogenic nature. However, it has not been possible up to the present, leaving aside the comma bacillus of Prior-Finkler, to discover a specific microbe of enteritis.

Catching cold may also be mentioned as a cause of catarrh of the intestines by producing a disturbance of intestinal digestion, that is to say, of intestinal function, either directly or by means of reflex nervous influences, the further consequence of which is inflammation of the mucous membrane. It need hardly be mentioned, and is really self-evident, that for the originating of these processes and the facility with which they spread, individual peculiarities as well as climatic

and accidental conditions are responsible. Children, particularly infants, are much more readily attacked than adults, whose intestinal mucous membrane is less susceptible, having become, as it were, hardened. In the summer and in hot climates the food products are more readily spoiled, and there is also a greater inclination to consume foods which contain a great deal of water and which ferment readily, such as fruits and beverages of various kinds.

Among the mechanical causes we must include enteroliths, gall stones, and also parasites of the intestinal canal; these may, however, with equal right be looked upon as causes of *secondary catarrh*. In this category may belong also the acute duodenitis following burns of the skin, which is probably caused, as shown by Samuel Hunter, by certain toxins formed in consequence of the burn, which are separated by the bile, and cause an irritation of the mucous membrane of the duodenum. On the other hand, those forms indisputably belong to the symptomatic or secondary catarrhs which occur with diseases of the oral cavity and of the stomach, and also with the acute infectious diseases, in chronic cachexias, heart and kidney diseases, general and intestinal tuberculosis, diabetes, etc. Secondary catarrhs are also caused by all the manifold occurrences in or about the intestinal canal which lead to sudden disturbances of the circulation of a local or general nature, such as strangulation, volvulus, invagination, peritonitis, thrombosis, embolism, etc. Finally secondary intestinal catarrhs accompany all neoplasms or ulcers of the mucous membrane of the intestine. Under all these last-named conditions, however, the intestinal catarrh possesses only a secondary interest, as its name well implies, and usually occupies a very subordinate position among the other symptoms of the primary complaint.

Chronic intestinal catarrh either follows an acute primary catarrh, particularly if the injurious causes are frequently repeated, or else the catarrhal inflammation assumes a chronic character from the outset, and is excited by small but frequently repeated irritations, or is developed as a result of all those causes which induce the symptomatic catarrh.

Pathological Anatomy.

It is only in rare cases that the inflammation involves the entire length of the intestine, and ordinarily it is confined to one portion. A pathologico-anatomical division of the individual regions can therefore be made, and we may speak of a duodenitis, jejunitis, ileitis, colitis, etc. Nature, however, does not usually confine herself closely to the divisions made by anatomists for their better guidance, and

quite frequently the inflammatory process passes from one of these divisions of the intestine to another, or attacks at the same time portions which are not contiguous, as, for example, the lower portion of the ileum and the mucous membrane of the rectum. This frequently happens in secondary catarrh, and the reason is that the injurious causal factors, for example tuberculous ulcers, may have their seat in different portions of the intestinal tract. In its gross appearances the inflammatory process cannot be distinguished from that affecting other mucous membranes.

There occurs first of all a hyperæmia which is either diffuse or is spread about in spots. The mucous membrane appears swollen, succulent, more or less intensely colored from a dark red to purplish, of velvety surface, and covered with tenacious mucus. In isolated points, particularly those corresponding to Lieberkühn's follicles and Peyer's patches, there appear at first small prominences, from the size of a pinhead to that of a lentil, or transparent nodules of a grayish color, which rupture later and occasion deep ulcerative lesions, the follicular ulcers. Besides this, there are found shallow losses of substance, the so-called catarrhal ulcers, which are caused by the loss in places of the protecting epithelial covering of the mucous membrane. Through extension of the inflammation in width and depth, large, irregular losses of substance with undermined edges are produced from the original round erosions. In their neighborhood polypoid growths may arise in consequence of the inflammatory irritation, when the process has run a protracted course. In severe cases there may be, on the prominences of the rugæ, a circumscribed necrosis which may take on a diphtheritic character.

On microscopical examination, also, we find changes similar to those occurring in catarrhal inflammation of other mucous membranes. The mucous membrane is permeated with a small-celled infiltration, the epithelium is cloudy, fatty degenerated, and in part desquamated. A copious accumulation of round cells is found, particularly in the follicles which rupture in consequence. If the disease has progressed so far as to the formation of follicular or catarrhal ulcers, and then erosions of the smaller vessels take place, hemorrhages will be found within the cellular tissue or on the free surface. The submucous tissue is usually somewhat hyperplastic, otherwise it is unchanged. The muscular and serous coats are affected only in chronic inflammation.

In chronic catarrh there will be found elongation and distention of the tubules of the glands, whose contents are either expelled or give rise, from strangulation of the neck of the gland and retention of its secretion, to the formation of cysts. The blood-vessels are greatly

distended and tortuous. The effused blood pigment, which has been converted into hæmatin, gives to the mucous membrane a slate-colored appearance.

Finally, the true parenchyma of the glands disappears altogether and nothing remains but a layer of connective tissue or an even accumulation of round cells; we then have atrophy of the mucous membrane.

This atrophy of the mucous membrane, which has been particularly studied by Klebs, Leube, Nothnagel, Jürgens, and Ewald, and which is analogous to the similar change in the stomach, is, like the antecedent catarrh, uniformly diffused over the entire surface only in isolated cases. In the great majority of cases diseased and normal parts alternate, or the process is confined to a certain portion of the intestine. Most usually the ascending colon and the cæcum with the parts in its immediate neighborhood are implicated, more rarely the upper portion of the ileum and the jejunum. The statement of Nothnagel, that the wall of the intestine is unchanged throughout its whole thickness, I am not prepared to accept. In my experience there is found rather frequently a distinct hyperplasia of the submucous tissue, while the muscular coat is variously affected, being in some places hypertrophic, in others not noticeably changed.

The follicular and catarrhal ulcers gradually extend downwards, as far as the serous tissue. Adhesions with neighboring intestinal coils, chronic peritonitis, perforations into the neighboring organs, the stomach, gall bladder, vagina, and bladder, and into the great vessels of the abdomen, have been observed. In case these ulcers heal, cicatricial contractions are produced, which, according to their extent and seat, may or may not occasion intestinal stenosis.

Symptoms.

Acute Intestinal Catarrh.

The affection usually begins suddenly, after the action of the causal injury, with diarrhœa which is preceded by unpleasant sensations and colicky pains. These become milder or disappear after the first defecation, return however in a short time, and again cease after the next stool, and so on. Diarrhœa, however, is not always present, and may be absent in disease of the upper portion of the intestine or even of the entire extent of the small intestine, so long as the large intestine properly performs its functions and carries out the solidification of the intestinal contents.

The diarrhœa is occasioned first by the increased peristalsis; secondly by the fact that the intestine, on account of its changed condi-

tion, loses in great part the power of absorption. Along with this, the inflamed mucous membrane secretes a more or less watery material mixed with mucus, and thus increases the liquid condition of the intestinal contents. The number of stools varies from two or three to twenty or more in the twenty-four hours. Defecation is accompanied by much tenesmus, and the desire frequently comes on so suddenly and urgently that the patient is hardly able to reach the water-closet. With the first or second stool, any firm fæcal still present masses are voided, then follow thin, gruel-like, frothy, and later watery evacuations, which in time become lighter in color and contain larger amounts of mucus. The odor, which in the beginning is characteristically penetrating and bad, becomes insipid and acid in time; the reaction is slightly acid or neutral, rarely alkaline. Mucus and bile pigment are present, the first always, the latter frequently, particularly if the inflammation extends high up into the small intestine. In infants and small children the dejections, which are normally of a bright-yellow color, are grass-green, or become so on standing in the air a short time, from oxidation of the bile pigment. Under the microscope are found masses of epithelium, more or less altered leucocytes, in the beginning remnants of food, consisting in part of unchanged or partially digested muscle and vegetable fibres, and in part of a granular or lumpy yellowish-brown detritus, some triple phosphates, and lastly innumerable bacteria. Among the latter the bacterium coli is most prominent; but in addition there are found cocci and other short rod-shaped bacteria, to the presence of which, however, we are unable to attribute any particular specific action. A copious development of gas is evidenced by rumbling noises within the abdomen. The air is in part passed at stool, and when passed at other times it is apt to take with it some of the liquid contents of the intestine (so-called moist flatus). The abdomen is generally distended by the meteorism and is slightly painful on pressure. As a result of the upward displacement of the diaphragm, dyspnœa, palpitation, and distress in the præcordium may occur.

The *subjective symptoms* are chiefly pressure and fulness of the abdomen, colicky pains preceding an evacuation, but rarely, and then only in catarrh of the large intestine, tenesmus following it. More commonly a temporary sensation of comfort exists after a stool. A feeling of general debility and malaise quickly comes on. As a rule there is no fever, although occasionally the disease is ushered in with chilly feelings or even a distinct chill, and the temperature may rise to 39° C. (102° F.). In little children or in decrepit persons there may be convulsions or even delirium. Enlargement of the spleen, as reported by Fischl, has never been observed by me. Such an occur-

rence is more than problematical, and the danger of mistaking the affection for typhoid fever, which is feared by some authors in view of this enlarged spleen, can hardly exist if a careful examination is made. When the catarrh has lasted some time and invaded extensive tracts of the intestine, the urine is diminished by reason of the great loss of fluid and even temporary anuria may occur. The urine is concentrated, dark in color, rich in urates, and contains indican and occasionally also albumin and casts (Fischl). One of the consequences of the loss of fluid is the intense thirst of which these patients complain.

The appetite is always lost, and nausea is present. Only in cases of catarrh limited to the large intestine may the appetite of the patient remain normal. In catarrh of the rectum, a burning, itching, or severe pain in the anus with frequent contractions of the sphincter (tenesmus) may be a very distressing symptom.

Chronic Catarrh.

Inasmuch as chronic catarrh belongs chiefly among the secondary intestinal diseases, the symptoms of the primary disease are generally predominant, although it may be that the disturbances occasioned by the catarrh remain for a long time the only signs pointing to the presence of disease. The dejections vary greatly in appearance in the course of a chronic catarrh. Constipation, diarrhœa, or the two alternately may be noted. This variation is explained when we recollect the different conditions of the mucous membranes during the course of the chronic catarrh. It is certain that following the primary irritation there must occur a paretic condition, that the originally increased secretion gradually diminishes, the increased peristalsis becomes less, the excitability of the intestinal nerves and the reflexes diminish. The contents of the intestine have more time to become inspissated through absorption, and are less diluted by the intestinal secretions. In this the form of bacterial infection may also be of importance, and the symptoms will vary according to the nature and the extent of the bacterial growth, and of the resulting toxins. Chronic intestinal catarrh with constipation is much more frequent than that with a continuous diarrhœa. Fæcal accumulations may then be felt within the abdomen, which sometimes cannot be removed in spite of vigorous and repeated purgation and may give rise to the suspicion of a neoplasm.

Should the catarrh be seated chiefly in the lower portion of the large intestine or the rectum, the stools will be generally fully formed. The scybala are enveloped in mucus or blood; and sometimes with violent tenesmus and bearing-down pains there will be voided only

large quantities of pure mucus. A common result of this tenesmus is the production of hemorrhoidal tumors, and bleeding is not infrequent.

Chronic inflammation of the rectum, *proctitis*, has a tendency to provoke an inflammation of the neighboring parts, a *periproctitis*, which nearly always results in abscess formation. By the bursting of the abscess an external rectal fistula, or an internal, that is, a rectovesical or rectovaginal, fistula is formed. Occasionally the evacuations will take on a typical periodic character, and occur only, for example, in the morning or evening or at a certain time in the night.

The composition of the stools does not present anything peculiar. Pus is found only when an abscess, resulting, for example, from proctitis or periproctitis, is present. Blood also is absent, unless there be complications present occasioned by the primary disease. The lower down the seat of the catarrh the less will be the amount of undigested food contained in the stools. The ever-present mucus either covers the fæces, if they are solid and formed, or is mixed with them in the form of flakes, streaks, or granules similar to sago grains or frog spawn.

The subjective symptoms are similar to those of acute intestinal catarrh, though the general condition is usually disturbed to a greater extent than in the latter affection. Indolence, apathy, dulness, hypochondriasis, headache, vertigo, palpitation, and an irregular pulse are symptoms which are due in part to the absorption of poisonous products from the intestinal canal (autointoxication). It is evident that nutrition must suffer in these cases more and more, and special investigations in regard to metabolism are not necessary to prove that the appropriation of nourishment is seriously interfered with. Thus emaciation is marked, and the loss of weight even in uncomplicated catarrh may be so excessive, under certain conditions, as to suggest malignant disease. In children these chronic catarrhs, complicated with diarrhœa, which appear usually about the period of the first dentition, may cause pædatrophia or marasmus, which usually leads to death unless we can succeed by proper treatment and nourishment in preventing the affection from running into intestinal atrophy. In these cases there is extreme emaciation with the senile facies, while the abdomen is greatly distended. Occasionally there is also a chronic peritonitis present which leads to a free serous exudation in the abdominal cavity.

Under certain conditions there may be an enormous distention and dilatation of the intestines. Walker and Griffiths have related the case of a boy eleven years of age whose colon was dilated to such a degree that its circumference in its descending portion measured 61

cm. The greatest circumference of the abdomen was 123 cm. The boy had suffered from birth with an enormously distended abdomen, but had been in fairly good health except for slight disturbances of the intestinal functions. He died rather suddenly, having previously become greatly emaciated, and having complained of dyspnœa and abdominal pains, which were quite severe at times and then again only slight. The cause of death was considered to be pressure of the distended abdomen on the enfeebled heart. A microscopical examination of the transverse colon revealed a nearly total loss of the glandular element of the mucous membrane, which was in a great measure replaced by fibrous connective tissue, while there was in places a marked infiltration of round cells. The muscularis mucosæ as well as the longitudinal and circular fibres of the muscular coat were greatly thickened. The authors believe that there was primarily a chronic colitis, and that the enormous distention of the colon was the result of a stagnation of the ingesta and formation of gases. I have myself observed a similar case, presenting an enormous dilatation of the colon, with great increase in the circumference of the abdomen, in a boy twelve years of age. In this case the diagnosis of dilatation of the large intestine was made during life, from the fact that without any difficulty whatever two and a half litres of water could be passed into the large intestine. Varying percussion phenomena could then be obtained, *i.e.*, dulness in the lateral walls and absence of dulness, according to the position occupied by the patient on one side or the other, a phenomenon which in conjunction with the greatly enlarged abdomen might easily, on careless examination, have been mistaken for ascites. An autopsy was unfortunately not permitted.

It has been already stated above that the sequel of a chronic catarrh may be complete destruction (atrophy) of the mucous membrane. Should this involve a large extent of the intestinal mucous membrane it will cause the gravest disturbances of nutrition. The visible symptoms of this are a chronic diarrhœa, which resists all medication, anæmia, and debility, such as might result from any great loss of fluid; there is no pain in the abdomen, and no tumor can be detected. In some cases there may be an appreciable distention of the abdomen due to the formation of gas, in others this may be entirely absent. The condition of the stools may also vary; sometimes they may have only a slight, stale odor; at other times they have an exceedingly disagreeable, nauseating stench. If, as frequently happens, an atrophy of the stomach coexists with the atrophy of the intestinal mucous membrane, we have the distinct picture of a grave pernicious anæmia. I have recently observed six cases of this kind, and on careful examination have found in all six a greatly diseased

condition of both the stomach and the intestine throughout their entire length, the mucous membrane being in great part converted into connective tissue. In a small portion were discovered the changes of a greatly advanced catarrh, and only at isolated points were the glandular elements so far preserved as to permit one to believe that their functional activity had been left intact.

The significance of these conditions in the production of the symptoms of pernicious anæmia has already been pointed out by Osler, Nothnagel, and others.

The so-called membranous enteritis and colitis will be considered in the section on nervous disturbances of the intestines.

Diagnosis and Localization of the Catarrh.

It has been said above that a strict localization of an intestinal catarrh is possible only when either the small or the large intestine is alone affected.

Catarrh of the duodenum is recognized by the presence of jaundice, since a catarrhal icterus is caused by the duodenal catarrh. In the absence of icterus, the presence of marked tenderness in the right hypochondrium, following upon a catarrh of the stomach, and the occurrence of stools particularly rich in mucus, will point to a catarrh of the duodenum. The true relation between burns of the skin and duodenal catarrh has already been mentioned. Catarrh of the small intestine may be differentiated from that of the large intestine, as a rule, by the presence of indican in the urine or by the Burgundy-red reaction of Rosenbach.* In catarrh of the large intestine and rectum, especially in proctitis, tenesmus and severe colicky pains in the left iliac fossa are characteristic. Itching and a burning sensation at the anus, the passage of mucus, either pure or streaked with blood, and great pain during digital examination, are of still further diagnostic import.

In chronic catarrh also, especially in that form which is accompanied by constipation, the passage of mucus forms the most important diagnostic symptom. Mucus, alone or mixed with fæcal matter, may also be present in the so-called membranous enteritis or colitis.

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* Rosenbach's reaction is obtained by boiling the urine in a test tube, while adding nitric acid drop by drop. Some specimens of urine will then gradually assume a Burgundy to a peony-red color, and retain this even on further boiling. Other specimens, although also becoming red at first, will on further boiling suddenly assume a light-yellow color. Only in the first case have we to deal with a specific coloring material which may be extracted with ether, and which is to be regarded as due to a disturbance of metabolic processes taking place in the small intestine.[16]

This form of disease, however, the cause of which is to be referred to a special neurosis, results in actual catarrhal disease of the mucous membrane, which differs from the forms we are now considering only in that the special catarrhal symptoms remain in the background, and the nervous element predominates. We can thus readily distinguish this affection, which we shall discuss more fully in the section on intestinal neuroses, from true catarrhal disease of the intestine.

For the diagnosis of a primary intestinal catarrh, the previously mentioned symptoms are usually sufficient. As, however, very many diseases of the intestine are complicated with secondary catarrh, it is of great importance to determine whether, in a given case, we have to do with a primary or with a secondary catarrh. For this diagnosis a painstaking examination is often necessary, yet sometimes the presence of a secondary or symptomatic catarrh is almost self-evident. Not infrequently the disturbance of nutrition results in such a great loss of weight, especially in persons who had a tendency to embonpoint, that the suspicion of malignant disease may be awakened, and it then becomes especially necessary to ascertain with the greatest care all the circumstances of the case in order to arrive at a correct diagnosis of the actual disease.

Prognosis.

The prognosis of primary intestinal catarrh becomes unfavorable only when the disease continues for a long time, for then there is danger that the catarrh may go on to atrophy or that the extension of intestinal ulcers may favor the production of untoward complications. In children and old people every intestinal catarrh is to be considered a serious disease.

In secondary catarrh the prognosis is that of the primary affection. It is not always easy, however, to distinguish a primary from a secondary catarrh, and hence we should give a guarded prognosis in all those cases in which there is any doubt as to the nature of the catarrhal process.

Treatment.

In slight cases *acute intestinal catarrh* will pass away without any treatment, if the cause ceases to operate and the organism removes the irritating substances from the intestinal canal. The diarrhœal discharges are salutary as serving to clear out the diseased intestine. Absolute abstention from all food, with perhaps a few drops of muriatic acid, is all that is necessary. A gradual return to ordinary diet, first thin broths, then mucilaginous broths and weak bouillon to

which an egg may later be added, tea with milk, some red wine, pure or mixed with boiled water or with some natural table water, such as Apollinaris or Selters, may hasten the cure. Very cold drinks, ice water, ice, raw fruit, farinaceous foods, vegetables, tough or fat meats, smoked meats, potatoes, and fresh bread, are to be avoided for some time after the affection has subsided.

When pain in the abdomen is severe, warm or even hot applications, or a Priessnitz compress, perhaps opium internally (ten to fifteen drops of laudanum several times daily), or in the form of suppositories, in the dose of 0.01–0.05 (gr. ⅙–i.) of the extract, may be prescribed. Should the symptoms not abate within the first forty-eight to sixty hours, that is if the diarrhœa is not sufficient to remove the noxious material from the intestine, then it will be necessary to render artificial aid. Calomel in doses of 0.2–0.5 (gr. iij.–viij.) for adults or one-tenth this amount for children, and castor oil in teaspoonful to tablespoonful doses, pure or in emulsion, have proved themselves in my experience to be the best purgatives in these cases, and I regularly employ one or the other of them. In obstinate cases we must not hesitate to employ disinfecting and astringent remedies internally or by enema if necessary, according to the seat of the disease. I prefer a powder composed of resorcin, bismuth salicylate, and tannigen in the following proportions:

℞ Resorcin, ... 5.0 (gr. lxxv.)
Bismuth salicylate,
Tannigen, ... āā 15.0 (ʒ ss.)
White sugar,
Sodium carbonate, ... āā 7.5 (ʒ ij.)
M. ft. pulv. S. A small even teaspoonful to be taken every two hours.

Tannin, nitrate of silver, rhatany, and decoction of columbo have given me little satisfaction, and the much praised naphthalin has produced bad after-effects (strangury). Ipecacuanha, if necessary with small doses of opium, and the decoction of hæmatoxylon (lignum campechianum) act sometimes very favorably in subacute catarrh. The local treatment of an acutely inflamed intestinal segment consists in irrigations of astringent or disinfecting solutions. Nitrate of silver, gr. iss.–viij. to Oij., tannic acid, gr. viij.–xv. to Oij., or boric acid in the same strength can be employed. I use the following formula by preference and with good results:

℞ Chloral hydrate, ... 3.0–5.0 (gr. xlv.–lxxv.)
Tannic acid, ... 1.5 (gr. xxiv.)
Lime water, ... ad 500.0 (O i.)
M. S. One-quarter to one-third of this quantity is to be mixed with twelve ounces of warm water or thin starch water, and of this five or six ounces or more may be injected into the bowel, and should be retained as long as possible.

The violent tenesmus may be relieved by suppositories of cocoa butter with opium 0.05–0.1 (gr. ¾–1½) and extract of belladonna 0.01–0.02 (gr. ⅙–⅓). It should, however, be remembered that some individuals have an idiosyncrasy in regard to belladonna, and suffer even after the smallest dose with slight symptoms of intoxication, such as dryness of the throat, difficulty in swallowing, and disturbance of vision.

In *chronic intestinal catarrh*, regulation of the diet holds the first place in the treatment. Regular, frequently repeated, and not too abundant meals, slow eating, and thorough mastication are just as important measures as they usually are neglected. As a general rule, the patient should refrain from indulging in any stimulating or easily fermenting food or drink. If an inclination to diarrhœa be present, the following articles are especially to be avoided: carbonated waters, lemonade, acid or not fully fermented beer, champagne, sweet wines, fruit, cabbage, salads, sweet farinaceous foods, very fat meat, and all smoked meats. It is injurious also to take any food either very cold or very hot. Careful attention to the diet should be continued for some time after the cessation of the catarrh. Repeated relapses are frequently the result of disobeying this injunction. The drinking-water should always be boiled before using, whenever possible.

Chronic Intestinal Catarrh.—The measures recommended for the treatment of acute catarrh may be used in the chronic form, but they should be supplemented by mineral waters, and by measures which may be required for the relief of the frequently existing constipation. As regards the treatment of the latter, the reader is referred to the section concerning it, and mention will only be made here of the fact that drastic cathartics always produce evil results. Not only is the inflamed mucous membrane of the intestine thereby made worse, but their effect, as is well known, becomes lessened in time so that the doses will have to be increased, and at last no effect whatever can be obtained from them. Of mineral springs, the saline-alkaline waters of Carlsbad, Marienbad, Vichy, Tarasp, Kissingen, and Homburg have from olden times had a great and well-earned reputation. The indication for the use of the individual springs is not always an easy matter to determine, and is regulated chiefly by the individual necessities of each patient. An experience of many years is necessary to a correct judgment in these cases. This can be obtained, of course, only by one who is personally acquainted with the places and the various therapeutic methods (*Heilfactoren*) there in use, and who has the opportunity to see the patients again after the course of treatment in order to be able to control the result. In general it may be stated that the hot baths of Carlsbad

are indicated in those forms of catarrh which are complicated with diarrhœa, and that for those cases in which constipation is present, the cold waters of Marienbad, Homburg, and Kissingen are to be recommended. The springs of Vichy (Grand-Grille, 41° C.; Celestine, 140° C.) take an intermediate position. Franzensbad and Elster are to be taken in consideration in the case of anæmic individuals; Tarasp waters are indicated when there is at the same time general debility, neurasthenia, or defective metabolism.

The treatment of the *intestinal catarrh of infants* and young children requires special consideration. Spoiled food is here in every case the cause of the disease. Therefore it is well, in the acute intestinal catarrhs of children, to withhold food of every kind and permit the patients to take only boiled water (Meinert, Heubner[17]). In chronic intestinal catarrh the food should at least be changed, and we should take the greatest care to see that germ-free milk be used. In nursing children the catarrh as a rule is checked by these simple measures. In bottle-fed children the process is not usually stopped so quickly. Here we may use thin farinaceous soups made from the different artificial foods, of which, according to Heubner, that sold under the name of Mellin's food is the best. In Germany a preparation called Hygienia, also Hartenstein's leguminose, Nestlé's milk food, and others, are very popular. In somewhat older children, the white of egg beaten up in water, thin broth made from the so-called white meats (poultry or veal) may be given in teaspoonful doses. These different substitutes for milk, are frequently, and especially at the beginning, vomited or rapidly passed undigested through the intestine. Under these circumstances lavage of the stomach has been of inestimable value. Formerly it was the rule to begin the treatment of an acute catarrh in children by a dose of calomel, and there is no doubt that following it the stools quickly take on a better condition and decrease in number. Within a few hours there will now follow a number of grayish-green stools, and as soon as these appear the calomel is to be discontinued. Instead of the calomel, a dose of castor oil (one-half to one tablespoonful) may be poured directly into the stomach through the tube, after irrigation of this organ.

After a thorough evacuation has been obtained in this way I prescribe a solution of muriatic acid in slight cases, or tannigen in severe cases. The latter is an acetyl combination of tannic acid, which is split up in the alkaline contents of the intestine so that tannic acid is liberated. In this manner the disturbance of the digestion of albumin in the stomach, which is caused by the formation of albuminate of tannin, is avoided. Tannigen may be given in doses of 0.1 gm. (gr.

iss.) to very young children and of 0.2 gm. (gr. iij.) to older children, from four to six times a day. The result, according to my own experience and that of Kunkel, Biedert, Escherich, Hoch, and others, is nearly without exception a prompt one, and the drug is certainly more reliable than rhatany, catechu, hæmatoxylon, and other astringents. The metallic astringents, nitrate of silver, acetate of lead, and alum, have been rarely used by me, and in these rare cases did not have any other effect than to cause still more gastric disturbance. Opium I use only in cases of very obstinate vomiting, severe spasms, or pain. With the administration of carbonate or tribasic phosphate of lime, recommended by Rendnitz, I have no personal experience. Irrigations of the bowel are made with solutions of common salt 0.6 per cent., tannic acid 0.5–1 per cent., salicylic acid or borax 0.5–1.0–2.0 per cent., and frequently repeated during the day. My preference here is for lime water with a slight addition of chloral, according to the formula above given.

In concluding this section on the therapy of gastroenteritis in children, we may recapitulate the following points:

1. *Diet.*—In cases of uncontrollable vomiting all food by the mouth will for the time have to be interdicted, and only ice water in quantities of a tea- to a dessertspoonful should be given. On the following day a solution of the white of egg in water may be administered in small quantities (two to three teaspoonfuls) every half-hour. As improvement takes place gruels may be substituted, and later on milk may be added to these.

In less severe cases feeding by the mouth may be continued. Cow's milk which has been given up to the time of illness should, however, be replaced by solution of egg albumin and gruels. When the child becomes exausted, strong bouillon, in tea- to tablespoonful doses, or Tokay wine diluted with equal parts of water, may be given every three hours. When the vomiting persists, enemata of peptone (5 gm. dried peptone to 50 c.c. of lukewarm water, three to five times a day) are to be given. Children at the breast are allowed to continue to nurse, although they should be deprived of the breast for a day if they vomit, and should take only solution of egg albumin during this time.

2. *Medicines.*—In the beginning of the illness, especially when diarrhœa prevails, calomel (0.01 gm.) may be given to children twelve months old. After a few calomel stools have occurred (after about six doses of calomel) the following should be given:

℞ Bismuth subnitrate, ... 0.1–0.2 (gr. iss.–iij.)
Powdered gum arabic. . . . . 0.5 (gr. viij.)
M. S. One such powder every two hours.

If the diarrhœa, however, has already lasted a number of days, tannigen in doses of 0.1–0.2, administered every two hours, may give very good results; it is, however, to be borne in mind that this remedy should be continued for some time after its favorable action has been obtained. In case of failure with these remedies, an infusion of ipecacuanha (0.2 to 100) with tincture of opium (three drops) may be given in the dose of a teaspoonful to a dessertspoonful every two hours. When the stools are of a green color a teaspoonful of a two-per-cent. lactic-acid solution may be given every hour or two. Should vomiting persist and no good result follow the calomel, creosote may be prescribed after the following formula:

℞ Creosote, ... gtt. ij.–v.
Syrup of althea, ... ʒ ss.
Distilled water, ... ʒ i.
M.

A teaspoonful of this solution every two hours may be tried, and is usually successful.

Lavage of the stomach is of extraordinary benefit, is surprisingly well borne by children and even infants, and has no bad after-effects whatever. Warm water may be used as the irrigating fluid, and in atony of the stomach an addition of common salt is made, the irrigations being continued until the water returns clear. The acute symptoms frequently subside after one, or at most a few irrigations. In endemic gastroenteritis the results are less favorable, but even in these cases great benefit may be expected from lavage of the stomach. Leo obtained a cure in forty per cent. of his cases treated in this way, and at the Polyclinic and in the children's ward of the Augusta Hospital in Berlin this method has been in use for some time with excellent results.

INTESTINAL ULCERS.

As the typhoid and tuberculous ulcers of the intestine will be treated of under the head of the respective diseases, and the catarrhal and follicular ulcerations have already been mentioned above, we shall here consider only the so-called round duodenal ulcer or ulcus duodeni pepticum.

The acid secretion of the stomach may under favorable circumstances produce a corrosion of the mucous membrane above as well as below the stomach itself, that is to say, in the lower portion of the œsophagus or in the upper portion of the duodenum.

The conditions for the formation and the development of these

ulcerations are the same everywhere, and whatever is said of ulcer of the stomach may also be applied to ulcer of the duodenum.

Etiology.

The formation of an ulcer, *i.e.*, the peptic digestion of a limited portion of the mucous membrane, can occur only when the acid secretion of the stomach attacks a mucous membrane which has been disturbed in its nutrition, that is at a point where a circumscribed disturbance of the circulation has manifested itself, be this occasioned by a direct disease of the vessels (embolism, thrombosis, atheroma), or by indirect influences (inflammation, hyperæmia, compression, effusion of blood, corrosion, or trauma). To be sure, the part taken by these causal factors is frequently even more difficult of proof in the intestine than in the stomach. An exception to this seems to be presented by the duodenal ulcers which make their appearance after extensive burns of the skin. The view of Hunter[1*] mentioned above, that we have to do in this case with an irritation of toxic material eliminated by the bile, and a consequent corrosion of the mucosa, is opposed by the belief of other authors that a thrombosis of the duodenal veins is caused by the liberation of a fibrin ferment.

The duodenal ulcer most frequently occurs in persons between thirty and sixty years of age, but may also be found in younger persons, not infrequently even in the newly born. In contrast to gastric ulcer it is more often met with in men than in women, in the proportion of three to one. As the action of the gastric juice is necessary to the production of the ulcers, it can be understood that these are met with especially in the upper part of the duodenum, between the pylorus and the papilla Vateri, and usually close to the pylorus. Only exceptionally is it found below the entrance of the ductus choledochus.

Pathological Anatomy.

The ulcerations are from the size of a pea to that of a shilling, and have their seat by preference on the inner or posterior wall. The loss of substance involves the mucosa and submucosa down to the muscularis, and sometimes the latter as well down to the serosa. The edges are sharp, the destruction is more extensive superficially than in the deeper parts. The base is covered with greenish or blackish sloughs, or shows the smooth tissue of the muscularis or serosa. Under the microscope the mucous membrane is seen to be healthy close up to the borders of the ulcer. Below the base of the ulcer we find circumscribed hemorrhages, small-celled infiltration, and in older

ulcerations increase of interstitial and fibrous tissue. If the process continues, adhesions with neighboring organs, the formation of an abscess, abnormal communications with adjacent viscera, perforation into the abdominal cavity, or erosion of the great vessels may occur. Among the complications or sequelæ may be suppuration creeping along the sheaths of the vessels and muscles upon the back or into the pleural cavity, or forming subphrenic abscesses; perforation into other intestinal coils or the gall-bladder, corrosion of large vessels of the wall of the intestines or even of the vena porta or of the aorta (Stich).

The ulcer may heal smoothly, without producing any cicatricial contractions, or a duodenal stenosis and consecutive dilatation of the stomach may be caused by the cicatricial contractions, or a compression of the gall-bladder and icterus may result.

In the same manner as from the base of an ulcer of the stomach, a carcinoma may be developed from the duodenal ulcer. Such cases are of course rare, but examples of the kind have been reported by Eichhorst and Ewald.

Symptomatology.

The ulcus duodenale may run its whole course without any symptoms whatever, and may be found accidentally at the autopsy, or its presence may be revealed only by the occurrence of one of the previously mentioned complications, namely, sudden and fatal hemorrhage, peritonitis due to perforation, symptoms of stenosis, dilatation of the stomach, or icterus, for which no other cause can be found.

In many cases the duodenal ulcer causes symptoms which cannot be distinguished in any way from those of an ulcer at the pylorus, or it may simulate gall-stone colic so closely as to make it impossible to come to a positive differential diagnosis. In such cases there is a more or less intense pain over the right lower border of the liver, but not at the parasternal line, coming on a certain time, usually from two to three hours after meals, but by no means always at the same time, and after a while gradually passing away. The pains radiate in the epigastrium and towards the sacrum. A spot is usually present near the location of the gall-bladder, or it may be farther towards the right and externally to it, which is tender on pressure. A tumor is hardly ever felt. The nutrition of the patient is generally not much affected, the appetite is not disturbed, the appearance is good, the tongue is clean, red, and moist, and there is usually more or less constipation. An examination of the contents of the stomach, after Ewald's test breakfast, shows that the amount of free hydrochloric acid is not increased, contrasting thus with ulcer of the stomach.

Indeed, in only a very few cases will characteristic symptoms be found. Among these the following may be mentioned: The pains make their appearance only late, in the third or fourth hour after meals, and are not increased by small amounts of food or alcoholic beverages, but are occasionally even diminished by them; they may, of course, entirely disappear if the patient takes no solid food or even liquids for several days, being nourished entirely by enemata; the seat of the pain is to the right of the parasternal line, and tenderness exists at this point even in the intervals of actual pain. The most important diagnostic sign, however, is hemorrhage. The blood either flows only into the lower part of the intestine and is discharged from the rectum (melæna), or it flows back into the stomach at the same time and is vomited (hæmatemesis). The occurrence of hæmatemesis alone is extraordinarily rare, and is probably an evidence of stenosis already formed.

Melæna and hæmatemesis have for their basis the corrosion of some larger vessel and the hemorrhage resulting therefrom. If the hemorrhage continues for some time but in such small amount that the blood is decomposed in the intestine and is passed in the fæces without any great change, profound anæmia may result, but the cause of it will remain problematical in the absence of a minute chemical examination of the dejecta. On the other hand, the hæmatemesis may possess a nearly typical character, inasmuch as remnants of food will be vomited at first, and only later distinctly recognized blood.

The hemorrhage, as in the case of ulcer of the stomach, is either fatal at once or will recur at longer or shorter intervals, finally ceasing or perhaps leading to a fatal issue.

The rarity of cases of this sort in which a diagnosis can be made during life will justify the relation of the following history of a patient observed by me:

A man, aged 34 years, was received into the Augusta Hospital on May 4th, 1892. The patient denied having had any of the diseases of childhood or any venereal disease. He was temperate in his habits and had always been healthy up to three years ago; since then he had been employed in a gun factory, polishing the stocks of guns. After having worked here for a few weeks, he experienced intense pains in the upper part of the abdomen, to the right of the median line. These pains were felt especially in the morning on rising, and also regularly four to six hours after meals; two to three hours after meals no pain was ever felt. The patient had a voracious appetite which could be appeased only for a few hours by food. At the end of this time he had nausea which was often followed by vomiting. The vomited matter was said to have consisted of a watery liquid without any admixture of food, and this, if the emesis continued, was followed by a bilious fluid. Liquid food was retained better than

solids. This condition lasted two weeks, after which the patient did not complain for nine months. Then the previous trouble began again. The patient suffered with cramps of the stomach and with vomiting of the same nature as before, the rejected matter consisting at first of a watery fluid, then directly following it a bitter, thick, brown liquid, of the consistence of syrup, often resembling cow-dung, in quantities of half a pint to a quart or more; and finally pure blood was vomited, sometimes of a bright red color, sometimes blackish-red, sometimes thin, sometimes thick, in quantities of a few teacupfuls. The patient had not noticed whether or not the stools were black. At this time the man was confined to his bed for eight weeks, and was greatly reduced on account of the nearly incessant vomiting of blood for five days. Following this he remained well for a year and a half. In October, 1891, the vomiting again returned, and took the same course as described above. The patient said that he could have collected the three different liquids vomited into three different glasses, and that without looking at them he could distinguish them by their taste, they being first insipid, then bitter, and finally sweetish. As long as bile was present this vomiting was very distressing; the blood came up without any effort. The pain, which was described by the patient as burning, came on almost constantly within five hours after each meal, and especially after anything of a particularly indigestible character had been eaten. The pain always preceded the vomiting. In November the patient was admitted to the Polyclinic of the Augusta Hospital. He reported that his stools were black like stewed prunes, even when he had not vomited any blood; and that frequently three to four days after he had had an attack of hæmatemesis the stools would also be of a black color, but before the vomiting of blood there usually would be constipation. From October up to the date of his reception he had had attacks of hæmatemesis every three or four weeks. The pain was constantly present. The patient had become much reduced; once he had been able, in the athletic club, to lift two hundred pounds easily with one hand, but now it was a difficult matter for him to lift ten pounds. In two years he had lost thirty pounds in weight. With frequent interruptions he had been able to work. The appetite was good even during the time when he suffered great pain, but was poor when the attacks of vomiting recurred daily. The patient did not complain of eructations, water brash, or of a bitter taste in the mouth. Along with the paroxysms of pain, which hardly lasted half an hour, there was a feeling of nausea and a burning sensation to the right of the linea alba. The stools, as a rule, were normal. The patient attributed his trouble to the fact that for three years he had been obliged in his work to press the handle of an auger against the scrobiculus cordis. While the man was in the hospital, a typical paroxysm of pain with vomiting was observed. Examination did not reveal any symptoms of tabes. After this there was a period of temporary improvement; the patient felt well, his pains left him, and he daily gained in weight and strength, complaining only occasionally of a queer sensation in the right hypochondrium. In the beginning of September he experienced another severe attack, following a slight indisposition for a few days previously.

Without any especial cause violent pains came on in the right side, which increased more and more until vomiting occurred. The latter was of the character already described; at first mucus, then, with great retching, bile, and lastly blood was vomited. Vomiting continued for three days and then ceased, while the pains, especially after meals, continued about two weeks longer; eventually these also disappeared, and the patient was gradually regaining his strength when another attack occurred on November 17th. For a few days preceding this there had been anorexia, constipation, and malaise, and the patient anticipated the return of his old affection, although he was unable to assign any reason whatever for its recurrence. At this time it was said that blood was vomited in large quantities, the patient estimating the amount to have been at least two litres. On the second day the vomiting ceased, while the pains were still very great. The patient lay continuously on his back, every movement increasing the pain. He was quite exhausted and miserable.

On a superficial study of the symptoms in this case, it was impossible at once to make a differential diagnosis; a closer study, however, of the individual symptoms, and a consideration of the other possible diseases, seemed to establish without doubt the diagnosis of ulcer of the duodenum.

As regards gall-stone colic, there are undoubtedly a number of symptoms common to the two affections; among these may be mentioned the appearance of the pain in attacks, in the intervals of which the patient is nearly absolutely free from any discomfort, the exclusive seat of the pain in the right hypochondriac region, a peculiar tenderness in the region of the gall-bladder, and lastly the attack of vomiting at the height of the paroxysm. Nevertheless the picture of the disease would be different in the case of gall stones. It seems unlikely in the first place that attacks of gall-stone colic, which usually lasts a few hours or at most a few days, should extend over weeks as they did in the case above related, and furthermore we ought to find other symptoms pertaining to the liver. It is certainly not so very rare to meet with gall-stone colic in which no icterus is present, when, for example, the stone does not fully close the ductus choledochus so that the bile may pass by it into the intestine, or when the calculus is not in the ductus choledochus but is arrested in the hepatic duct. In either case, however, the stone would, by its long retention, cause other symptoms; in the case of arrest in the ductus choledochus the bile would gradually be dammed up behind the stone and would be retained in the bile ducts, the liver would become enlarged, and icterus would be the result; in the second case the gall-bladder would become sympathetically involved, dropsy of the gall-bladder would result, and this could be recognized as a tense, palpable tumor at the border of the liver.

But in addition to the absence of these symptoms there were others present in the above narrated case which directly militated against a presumption of gall-stone colic. These were the undeniable connection between the taking of food and the occurrence of the pain, the hæmatemesis, and the bloody stools.

These symptoms would point with certainty to the presence of an ulcerative process, which was irritated by the chyme and which easily caused hemorrhage; but they might be present in the case of a malignant new growth in the small intestine. In their first stage of development, ulcer and carcinoma of the duodenum can be as little distinguished from each other as can the same processes occurring in the stomach; but in its further course the affection can nearly always be positively diagnosed. The course of a carcinoma is rapid and resistless; its average duration is about one year, its maximum three years. The appetite of the patient is generally diminished or entirely lost, the pains are continuous and increase to paroxysms, hemorrhage is usually not alarming, vomiting occurs frequently, and the patient rapidly loses ground and soon presents the characteristic appearance of cancerous cachexia. This morbid picture of a carcinoma does not agree with that related above. The affection has now existed three years and a half, and though the patient has during this period lost much ground, the decline has not been continuous; but there have been periods in which he gained in strength, felt well, and did not suffer from his affection. Neither does he present the earthy color of one suffering from a carcinoma, and the emaciation and pallor are probably not the result of the ulcerative process *per se*, but are due to the great loss of blood which may be even more severe than the patient thinks, as the blood may be present only in the stools. The most important among the symptoms of carcinoma is, however, the presence of a palpable tumor, which is hard, irregular in outline, and immovable. It may become appreciable to palpation early in some cases, later in others, but it could hardly fail to be present in a case with so long a history as the one we have been studying. Another common result of an advanced carcinoma, which usually involves the entire circumference of the intestinal wall, is stenosis of the intestine. The upper portion of the duodenum would then become distended first, and later there would be the symptoms of stenosis of the pylorus, namely, dilatation of the stomach, vomiting after each meal, constipation, and retraction of the abdomen. None of these symptoms, however, was present in the above case, and weighing all the evidence, there was much more that argued against the presence of a carcinoma than that could be found in favor of such a diagnosis.

We may lastly suspect the presence of the so-called gastric crises

of locomotor ataxia. But the absence of all symptoms pointing to a disease of the central nervous system would exclude this at once.

The affection which it is most difficult, and sometimes impossible, to differentiate from duodenal ulcer is ulcer of the stomach. This could be excluded, however, in the case under consideration by means of two symptoms which stood out very distinctly. The first was the pain which was felt always in the right hypochondrium, and did not pass to the left of the median line. Then on palpation there was found, somewhat below the arch of the ribs and to the inner side of the mammillary line, therefore in the course of the horizontal portion of the duodenum, a circumscribed spot which was extremely tender to pressure; we were justified in assuming that this was the localization of the ulcer. The second symptom was the time of occurrence of the pain. While the pain in gastric ulcer makes itself felt with, or only a short time after, the ingestion of food, that accompanying duodenal ulcer appears only some hours after meals at the time when the food is passed into the intestines by the stomach and comes in contact with the ulcerated surface. This patient above mentioned always maintained with great positiveness that the pain did not come on until about five hours after a meal.

To establish the diagnosis conclusively an examination of the gastric juice was made. About one hour after Ewald's test breakfast, consisting of one roll and a cup of tea, the stomach tube was passed, and by aspiration 150 c.c. of well-digested chyme was obtained. The mass gave an acid reaction to litmus paper. In the filtered solution free hydrochloric acid was distinctly found by Günsburg's reagent. Lactic acid was not found with Uffelmann's reagent. The total acidity was determined by trituration with decinormal soda solution to be 40. Digestion of albumin was perfectly normal, even without the addition of any muriatic acid. Testing for peptone by means of caustic soda and sulphate of copper gave a positive result. The gastric juice therefore presented no abnormalities. Of special note was the fact that the amount of hydrochloric acid was low, for even though hyperchlorhydria is not present in all cases of gastric ulcer, a diminished amount of free acid is certainly the exception.

An interesting symptom in the above case was presented by the character of the vomited matters, which consisted of three totally different parts, distinguished by an insipid, a bitter, and a sweeetish taste. An explanation of this can easily be given. When the food passed from the stomach into the intestine, the surface of the ulcer was brought in contact with it, and was thereby irritated; then by the passage of more and more matter the pain constantly increased, and in the end became so great that vomiting was occasioned by reflex

action. This at first brought up the food which was present in the stomach as an insipid mass; the pain, however, caused an antiperistalsis of the upper portion of the intestine, and bile was thus carried into the stomach, whence it was vomited as the bitter mass; finally, the strong contractions of the muscular fibres of the intestine, stretching the ulcerated surface, caused the bursting of a vessel, and the blood passed into the stomach in the same way as the bile, and when ejected had a sweetish taste.

Diagnosis.

I have reported the foregoing case in detail because it disposes at one and the same time of the diagnosis and differential diagnosis of ulcer of the duodenum. The diagnosis of the complications and sequelæ of duodenal ulcer may usually be made from their characteristic symptoms, but the fact that these are complications or sequelæ of an existing or a healed ulcer can be determined only by a consideration and careful weighing of the symptoms above mentioned. As this is possible only in exceptional cases, the final cause of such complications will generally be discovered only on the post-mortem table.

Prognosis.

The prognosis of duodenal ulcers is unfavorable, for they can be influenced but little or not at all by medicines, and their tendency is less to heal with smooth cicatrices than it is to lead to contractions and stenosis, or to go on to deep ulceration with all its consequences.

Treatment.

Therapeutically we are nearly powerless in the presence of ulcer of the duodenum. The avoidance of dietetic indiscretions is of the first importance. The proper management of these cases and their medicinal treatment (especially the so-called Leube cure) are the same as those for ulcer of the stomach, and have been treated of in detail in the article on Diseases of the Stomach in another part of this work (see Vol. VII., p. 232). Unfortunately, however, because of the hidden and inaccessible position of duodenal ulcer, it is with much more difficulty influenced by medicinal measures.

TYPHLITIS AND PERITYPHLITIS (APPENDICITIS).

Introduction.

The history of typhlitis and perityphlitis reaches back, leaving aside a few reports of cases of earlier date, into the thirties of the present century, when the first monographs on the subject were pub-

lished by Dance and Menière in France, and by Bamberger, Volz, and Albers in Germany. In no chapter of the diseases of the intestines has there arisen so much discussion or has there been so great a change of views as in that of the inflammatory conditions in and about the cæcum. The importance attributed by Albers to typhlitis in its narrower sense, that is, to the inflammatory processes of the mucous membrane, in the causation of perityphlitic inflammations, has undergone considerable limitations, as a result of anatomical researches and of the experience gained in the numerous operations of recent times. Among those who have contributed to our knowledge of this affection, we shall mention merely the names of Bardeleben, Kraus, Henle (1861), Treves (1885), Tuffier, Fitz (1887), Maurin (1890), Ferguson (1891), Senn, Murphy, McBurney, Roux, Delorme, Lenander (1893), Sahli, Kümmel, Schede (1894), Sonnenburg (1889–1895), G. R. Fowler (1894), Renvers (1895), without, however, in the least exhausting the list of those who deserve to be quoted in this connection.

Anatomy.

The Relation of the Peritoneum to the Cæcum and the Appendix.— The older anatomists and physicians were of the opinion that the peritoneum only partly covered the cæcum, enveloping about three-fourths of its circumference and leaving free the portion lying behind and in front of the psoas muscle. So also the appendix was supposed not to be completely enveloped by the peritoneum, whose duplicature was thought to form the mesentery or mesenteriolum of the appendix, but that the lesser segment of its periphery lay outside the peritoneal cavity.

Accordingly, a sharp distinction was made between the inflammatory processes which arose within the cavity surrounded by the peritoneum, the *perityphlitic* inflammation, and those occurring extra cavum peritonei, the *paratyphlitic* inflammations.

It has, however, been demonstrated that the cæcum, in the greater number of cases, is perfectly enveloped by the peritoneum. When Treves[19] says: "The cæcum is always entirely covered by the peritoneum, and will never be found attached by areolar tissue to the iliac fascia," and when Maurin writes: "la main peut faire la tour du cæcum, comme elle fait la tour de la pointe du cœur dans le péricarde," this is certainly not always and without exception true, for Tuffier has seen the peritoneum absent from the upper third of the posterior surface of the cæcum in 120 cadavers, and the intestine rested directly upon the cellular tissue of the pelvic wall. Ferguson found, in 200 autopsies, 77 cases in which the posterior wall of the

cæcum, together with the appendix, was situated behind the peritoneum. Fowler gives an illustration, in his monograph, of the position of the cæcum according to Tillaux, in which it is represented as resting upon the retroperitoneal connective tissue, with its posterior segment free from any peritoneal covering. Sonnenburg, in his monograph, narrates a few remarkable cases of unusual situation of the appendix, in which the latter was met with partly outside of and partly enveloped by the peritoneum. Lenander quotes the following statement of Sydow, of Gothenburg, based upon an examination of 586 cadavers: "The general rule is, therefore, that the processus vermiformis arises from the inner side of the cæcum and lies free in the abdominal cavity, although covered by a peritoneal investment which forms its mesentery. In 44 cases the appendix, however, was attached to the posterior portion of the cæcum, in 23 cases to its outer wall. It was found 49 times far up in the fossa iliaca; 61 times it dipped down more or less into the pelvic cavity. It was found in the peritoneum lining the iliac fossa, partially covered by it in 19 cases, and wholly covered in 20 cases. In 11 other instances it was entirely lost in the retroperitoneal connective tissue, behind the cæcum."

According to this, therefore, the total or partial extraperitoneal position of the cæcum and its appendix, although occasionally present, notwithstanding the views of Treves and Maurin, is to be looked upon only as an exception.

The Position of the Cæcum and the Appendix in the Abdominal Cavity.—Although normally the cæcum rests on the psoas muscle in the ileocæcal fossa, and the point of entrance of the ileum must be looked for below the venter of the ilium, with the tip of the cæcum a little to the inner side of the middle of Poupart's ligament, it may be found in other and very strange locations. This displacement may be due partly to anomalies of formation, for example, the congenital absence of the ascending portion of the large intestine, partly to the mobility of its mesentery, partly to the presence of old adhesions, contracting and pulling upon the intestines. In this way the cæcum and ascending colon may be drawn over as far as the median line, even to the left of it, or the cæcum may be found lying close to the border of the liver, or even behind it in the direction of the transverse colon. Or, again, the cæcum may sink down into the true pelvis (here it was found by Treves in eighteen out of one hundred cases) and may rest on the bladder, the uterus, or the sigmoid flexure. Curschmann also draws attention to the presence of flexures and curves of the cæcum, of such character that the extremity of the cæcum may point upwards towards the diaphragm, covering a corresponding portion of the as-

cending colon. Of the possible occurrence of all these anomalies we find abundant evidence both in the older and recent authors; among the latter we may mention Curschmann and Fitz, but any one who has had somewhat extensive dissecting material at his disposal will be able to recollect such anomalies in his own experience.

It goes without saying that the vermiform appendix follows these abnormalities of positions, and hence may be met with in very diverse parts of the abdominal cavity under certain circumstances. However, even when the cæcum remains in its normal position, the appendix may be anomalous in various ways. In the first place, as is well known, it differs extraordinarily in its length. The normal size of 7–8 cm. (3 inches) is occasionally increased to 10–12 cm. (4–5 inches), and to find an appendix measuring 14–16 cm. (5½–6½ inches) at an autopsy is not very unusual. It passes at a right angle from the intestine to the rim of the true pelvis, and projects more or less beyond it into the pelvis; or it extends for a greater or lesser distance behind the ascending colon so as to reach the lower end of the kidney with its apex, or takes its course perpendicularly downwards towards Poupart's ligament. The following positions may therefore be recognized:

1. Behind the ileum, with the apex directed towards the spleen, either free or wound around the ileum;
2. Behind the cæcum, resting on the psoas muscle, with the apex towards the wall of the pelvis;
3. In connection with the bladder;
4. Adherent to the ovary or broad ligament;
5. Adherent to the umbilicus, the gall-bladder, or the kidney;
6. Situated to the left of the median line, towards the sigmoid flexure.

M. Lafforgen found as a result of the examination of 200 cadavers of persons of both sexes and all ages, that the appendix was directed downwards in 41.5 per cent. of the cases, laterally and externally in 26 per cent., laterally and internally in 17 per cent., upwards in 13 per cent. Osler found in one case the appendix, together with the cæcum, situated in the inguinal canal; here it twisted on itself, returned to the abdominal cavity, and ended in an abscess cavity which was situated on the right side of the promontory of the sacrum. According to Lockwood and Rolleston,[20] the appendix was free 94 times in 104 cases, 7 times it was free but obliterated or stenosed, and 4 times dilated. Frequently the appendix is twisted on its own axis like a corkscrew on account of the shortness of its mesentery. In the great majority of cases it is slightly curved with its concavity turned towards the cæcum, and occasionally it is sharply flexed like

a hook. The reason of this is that the mesentery or mesocolon of the appendix arises from the left or lower fold of the mesentery of the ileum, and is attached along the whole appendix only in the fœtal period or exceptionally in later life; but in the adult it is attached to a certain portion only of the appendix, that is to say, it is too short.

It may be remarked that the mucous membrane of the appendix has the same histological formation as the mucous membrane of the ileum, and thus constitutes a secreting surface; but it is distinguished by its richness in follicles. It has for this reason been compared to the pharyngeal tonsil, and an explanation of the tendency of the appendix to inflammatory disease has been deduced therefrom. Below the mucous membrane a submucosa is found, and under this a layer of muscular tissue which is covered by the serosa. Generally the appendix is a little wider at its blind extremity than where it enters the cæcum. In the latter portion there is a duplication of the mucous membrane called Gerlach's fold, which opposes a certain impediment to the exit of the contents which have found their way into the appendix. It is very evident that the evacuation of the contents of the appendix into the intestine can be accomplished only slowly even under normal conditions on account of the short mesenteriolum, but that it will be impeded to the utmost when flexures or distortions or inflammatory processes are present.

The appendix, nevertheless, possesses a typical peristaltic movement. Parker Syms[21] reports an observation in which a long appendix removed during operation continued to make circular movements, like an earthworm, and eventually expressed some fæcal matter.

The cæcum and appendix constitute the field in which the clinical picture of typhlitis and perityphlitis is formed. Taking these anatomical conditions as a basis, the following table of the inflammatory processes originating from the ileocæcum and its appendix may be presented:

1. Typhlitis; 2. Perityphlitis or periappendicitis; (*a*) of the cæcum, (*b*) of the appendix, either intraperitoneal or extraperitoneal (paratyphlitis).

Fowler[22] includes both forms under the heading of paraappendicitis, and differentiates an endoappendicitis (inflammation of the mucosa) from a peritoneal appendicitis (extension to the muscularis and serosa). In their clinical aspect these forms are of either the simple catarrhal or suppurative (perforative) variety, so that we may distinguish an appendicitis aut perityphlitis simplex vel catarrhalis, and an appendicitis suppurativa vel perforativa, with their sequelæ.

This division cannot, however, be adhered to in all strictness. If

it is difficult on the post-mortem table, notwithstanding careful examination, to find the starting-point of a localized, circumscribed, or diffuse peritonitis, it is even more so during the lifetime of the patient.

The clinical symptoms of typhlitis and appendicitis, as well as perityphlitis, are occasionally so similar as easily to give rise to mistakes. In such cases we can only make use of the experience which we have gained on the operating-table or in the dead-house. Nevertheless, I cannot agree with Treves, when he says of the above or a similar classification: "This classification has long since been shown to be unsound." In the first place, the assertion of Treves, that the cæcum and appendix, without exception, lie for their whole extent within the peritoneum, that a paratyphlitis therefore is anatomically and clinically impossible, is not correct, as may be deduced from what has preceded and as I can assert on the strength of my own observations. This condition is, however, non-essential or only of secondary importance. All perityphlitic abscesses, as Sonnenburg well says, as long as they do not cause a diffuse peritonitis, are to be looked upon as extraperitoneal or paratyphlitic, because they are separated by firm adhesions from the peritoneal cavity. But we will leave this academic question, which practically only comes into consideration as regards the greater facility with which the paratyphlitic abscess makes its way towards the surface or the interior. Undoubtedly it is absolutely necessary, for the comprehension of the processes under consideration, to classify them in such a way as to represent as nearly as may be the true condition present. This can best be done by means of the foregoing schedule, which we shall make the basis of the following study, with so much greater reason, as we are convinced that it is founded upon actual anatomical conditions, although the clinical picture of the diseases which we shall consider does not always correspond with them. If we were to base our classification upon the clinical symptoms alone, following Rotter[23] we might distinguish two large groups, as follows: (1) Perityphlitis diffusa, *i.e.*, those cases which are complicated with a diffuse peritonitis, and (2) perityphlitis circumscripta, which embraces appendicitis simplex, appendicitis cum perforatione, and appendicitis retroperitonealis.

It seems clear that these two groups may nearly always be separated from each other without any difficulty. The thorough physician will, however, hardly be satisfied with the all-embracing term perityphlitis circumscripta, but will always endeavor to discover the real nature of the affection with which he has to deal.

Typhlitis.

Typhlitis, in the true sense of the word (*i.e.*, an inflammation of the inner wall of the cæcum), is anatomically nothing else than a colitis, distinguished by the peculiar position of the cæcum. It is, however, of special interest by reason of its relation to perityphlitis, *i.e.*, the inflammatory processes occurring in the parts surrounding the cæcum, in so far as it may be the cause of the latter and might be mistaken for it. Strictly speaking, inflammation of the appendix should also come under the heading of typhlitis, the appendix being nothing else, according to its history of development and its anatomy, than the rudiment of a former cæcum. The peculiar and determining rôle which is played by appendicitis in the inflammatory processes occurring in the ileocæcal fossa makes it seem more suitable, however, as well as convenient to accord to appendicitis an independent position and to discuss it separately.

The importance of typhlitis in relation to the disturbances occurring in the neighborhood of the ileocæcum has, up to recent times, been greatly overestimated. The common belief until lately was, on the whole, that the mucous membrane of the cæcum, becoming irritated by the pressure of accumulating masses of fæces, took on an inflammatory action—typhlitis stercoralis—and that the inflammation thence spread through the wall of the intestine to the parts surrounding the cæcum, *i.e.*, became a perityphlitis. According to this opinion the appendix was only in so far entitled to an etiological consideration as an obvious perforation of its wall was found to be the cause of the perityphlitis. This view has latterly, by reason of experience obtained in the numerous operations performed on the living subject, been completely reversed, so that some authors, as for example Sahli, deny altogether that typhlitis has any causal relation to perityphlitis, and regard the appendix as alone responsible for the origin of perityphlitic disease. This, to my mind, is like throwing out the child with its bath. Does a typhlitis stercoralis exist, and in what relation does it stand to perityphlitis? In general there is not much foundation for the theory of an accumulation of hardened fæces in the cæcum, for, as a rule, in the cæcum there is found even in long-standing constipation, soft or semi-solid or liquid matter containing firmer fæcal masses, which, however, by no means fill the lumen of the intestine. Those cases in which, at the autopsy, the cæcum is found tensely packed with hard masses are exceptional, and frequently the evidences of recent or older inflammatory or ulcerative processes of the mucous membrane are not found even in these

cases. When the right iliac fossa is palpated in the living subject, especially in those cases in which pain is complained of in that region, and also in such conditions of chronic constipation, we may convince ourselves that the contents of the cæcum are liquid. If now we depress quickly and forcibly the tips of the fingers of the hand resting with its palmar surface on this region of the abdomen, a very characteristic swashing sound will be heard, as might emanate from an intestine filled partly with air and partly with fluid. Whenever a more or less large, hard, or tense, elastic, sausage-shaped tumor can be felt under the finger, we may be sure that we have to deal either with some acute inflammatory process outside of the cæcum, or with some kind of mechanical force, as the presence of enteroliths, strangulation, external compression, or the like, which obstructs the passage of the contents of the intestine. The fæcal obstruction is not therefore the cause, but rather the result of a disease in or about the intestine.

Although we do not wish to deny the possibility of the existence of a typhlitis stercoralis and of a perityphlitis resulting from it—Lenander, for example, has operated for a recurrent perityphlitis on a patient in whom there was no appendix—it must certainly be encountered in an extraordinarily small number of cases. Thus it has happened, as has already been said, that among recent authors Sahli denies the occurrence of a typhlitis stercoralis altogether, while Talamon, Curschmann, Fitz, Treves, and others have proved that the inflammation of the cæcum, in cases of perityphlitis, does not occur from within outwards, but rather the reverse, the course of the inflammatory process being from without inwards. Sonnenburg in one hundred and thirty operations has never met with conditions which could be interpreted as a typhlitis, and calls especial attention to the fact that he operated for symptoms which in general are looked upon as characteristic of typhlitis. It is obvious, however, that we should not be justified in denying altogether the occurrence of typhlitis on the strength of such observations. The surgeon has no chance to see cases of typhlitis which, as is well known, rapidly go on to recovery. The cases seen by him are those of perityphlitis or of appendicitis, the symptoms of which are the same as those of typhlitis, and which can be differentiated from uncomplicated inflammation of the cæcum only by the fact that it is not readily recovered from, and for that reason comes under the notice of the surgeon. Clinically these affections have the greatest similarity and often are not to be distinguished from each other. The description of the "typhlite simple stercorale," which is given by Jeanselme,[24] may be applied to appendicitis as well as to typhlitis.

If we therefore assent to the question of the occurrence of perityphlitis, *sensu proprio*, that is in its origin from the cæcum, it should be remarked that typhlitis proper and the perityphlitis arising from it become the starting-point of the process only in a very small number of cases, so that the causal element, as far as practice is concerned, is confined essentially to the diseases of the appendix. We shall therefore be able to discuss the perityphlitic processes together, no matter whether they originate in the cæcum or in the appendix, and shall merely precede this study by a few remarks on typhlitis proper; but here the fact at once presents itself that typhlitis and appendicitis are so nearly related to each other that the two processes frequently cannot be sharply separated.

Etiology and Pathological Anatomy.

The origin of typhlitis is attributed to mechanical and chemical irritation, due to improper food or to trauma in the vicinity of the cæcum. Among these etiological factors are hardened, inspissated fæces, gall stones, true enteroliths, and foreign bodies. Inflammation of the mucous membrane may also occur in infectious diseases, complicated with the formation of ulcers, as in typhoid fever, tuberculosis of the intestine, syphilis, dysentery, diphtheria, carcinoma, and actinomycosis. It should, however, be mentioned that in the latter processes there is an extension of the disease to the neighboring parts in such a manner that the symptoms of the primary affection occupy the foreground in the picture of the disease, and therefore a true perityphlitis can hardly be observed clinically, since the condition is dominated by the original disease. Thus Hoffmann discovered forty-seven cases of ulceration of the cæcum and appendix in two hundred and thirty-three autopsies of typhoid-fever patients, a number which seems to me, according to my more limited experience, to be altogether too small.

Moty[25] has twice found dysenteric ulcerations in the cæcum, near the entrance of the appendix, which had caused the symptoms of an appendicitis. Extensive tuberculous ulcers with consecutive adhesive peritonitis are more frequently met with. They may extend from the cæcum and its appendix as far as the valve of Bauhin, and possess externally some similarity even to cancerous tumors.[26] The walls of the intestine are then infiltrated by the neoplasm, whose true nature can be determined only by microscopical examination. A number of cases of recurrent appendicitis, to which I can add a similar one to be described below, are, according to Richelot and Benoit, of a tuberculous nature.

Treves mentions a case of sudden perforation of the cæcum by a tuberculous ulcer; in another case an ulcerating epithelioma was found to be the cause of the perityphlitic symptoms. Actinomycosis leads to long-continued suppuration,[27] and the characteristic grains of actinomycosis can be recognized in the pus. In these cases, which are very rare, the error of taking it for a genuine typhlitis or perityphlitis is especially easy, and can, in fact, be avoided only by a close examination of the pus, obtained by puncture at operation or through spontaneous rupture. Fitz found in two hundred and fifty-seven cases of perforating appendicitis and two hundred and nine cases of so-called typhlitis a perforation of the cæcum in only three cases. In one case a needle was found, in another a fish bone, and in the third only a "strangulation" of the intestine. But, as in the case of the above-mentioned injuries, it might be difficult to bring the proof of their mechanical injury, and it might be that the fæcal concretions, or gall stones, etc., were so large as to cause, by mechanical pressure, disturbances of the circulation with consequent erosion. Small foreign particles which may be carried to the lower portion of the intestine, such as undigested husks, seeds, fish bones, small pieces of bone, etc., are always encased (as can occasionally be seen at autopsy) in a thick coating of mucus, and can hardly as such exert any irritating influence on the mucous membrane. In the greater number of cases it is probably due to chemical or bacterial products which may in these cases be the more suspected as the patients generally give a rather long history of the disease, in which constipation, alternating constipation and diarrhœa, or insufficient emptying of the rectum is complained of. These conditions may possibly gain some support by the subsequent finding of foreign bodies.

It is only rarely possible to study the changes in the cæcum, since typhlitis as a rule runs a favorable course. A case published by Mariage was that of a boy, ten years of age, who had presented the symptoms of typhlitis stercoralis. The affection was subsiding when the boy was attacked with a diphtheritic angina, from which he died. The wall of the cæcum was found thickened, the mucosa was red and swollen, but was neither ulcerated nor perforated. In some places the cæcum was adherent to the small intestine, and in these adhesions small circumscribed abscesses were found, the largest of which, of the size of a hazelnut, was situated on the inner side of the cæcum. The appendix was free and absolutely healthy.[28] From these and similar findings it may be seen that the anatomical changes are identical with those found in catarrh of other portions of the intestine. In time the formation of follicular and catarrhal ulcers with circumscribed adhesive peritonitis will take place here also, and eventually suppura-

tive inflammation of the peritoneum or of the retrocæcal connective tissue will follow.

Symptoms.

Typhlitis proper usually comes on slowly and lingeringly, and as long as it is confined to the mucous membrane does not present any characteristic symptoms. A dull pain in the ileocæcal region, increased by movement, cough, or pressure, which may also occur in paroxysms and which radiates in various directions towards the umbilicus, the right hypochondrium, or the hypogastric region, is combined with dyspeptic symptoms. Eructations, nausea, rarely vomiting of food particles or bile, and above all constipation are present. Ileus or symptoms of the latter, which the text-books say may occur, have never been observed by me in a case of simple typhlitis. The abdomen is usually somewhat distended, pressure in the ileocæcal region provokes tenderness or actual pain. If the tension of the abdominal muscles is not too great, a sausage-shaped tumor may be felt in the right iliac fossa running from above downwards and parallel to the inner edge of the ileum; the finger passed over this in an oblique direction enters the true pelvis at its inner side, and at its outer side impinges against the centre of the ileum. As a rule this tumor will be, as already mentioned in speaking of its etiological significance, by no means hard or firm like inspissated fæcal matter, but rather gives the impression of a pasty mass.

A marked elevation of temperature is usually not present, although I find repeatedly recorded in my case book temperatures as high as 38.8°–39° C. (101.8°–102.2° F.). The temperature of the skin over the tumor feels warmer to the hand than that of the vicinity. The urine is diminished, high colored, and occasionally contains traces of albumin and indican.

Prognosis.

The termination of a simple typhlitis is in recovery. As long as the inflammation confines itself to the cæcum, it retains its tendency to spontaneous recovery. The accumulated fæcal masses are removed by the returning peristalsis or through purgatives, and the *status quo ante* is restored.

Treatment.

So long as the doctrine prevailed that perityphlitis was a sequel of typhlitis stercoralis, the treatment of the latter naturally was directed towards the removal of the accumulated fæcal masses. But the question whether typhlitis or perityphlitis in a broader sense was

present in a given case was of the greatest importance. For as little as the physician was inclined in the latter case to cause injury by purgatives and the increase of peristalsis produced by them, so much the more necessary was it in the former case to clear the intestine of the materia peccans. At present, the importance of typhlitis has certainly been greatly diminished, and the question therefore, whether or not purgatives are indicated, has lost very much of its former significance. Still a number of cases do occur, and this makes it desirable to be able in the very beginning to lay out some positive plan of treatment. In my experience the following procedure has given the best results: If the complaint has not come on suddenly but gradually, and has been preceded for some time by irregular action of the bowels, a circumscribed, sausage-shaped tumor being felt in the right iliac fossa, or if a resistance is felt which is painful on pressure, but there exists no large, diffuse, or circumscribed exudate with distinct dulness; and finally if no fever or at the most only a slight evening rise without any essential acceleration of the pulse rate is present—if, in one word, the symptoms do not point to an acute attack with profound involvement of the general condition, although the pains and the subjective symptoms of the patient may be very intense, I do not hesitate to cause an evacuation of the intestine. An enema of from 300 to 500 c.c. (℥ x.–xvi.) of water or oil is given, and in addition a large dose of castor oil (at least 25–30 gm. or ℥ i.) per os if the former is not sufficient to produce a large evacuation and disappearance of the fæcal tumor. The oil is given best in the form of an emulsion. Other purgatives, for example senna or rhubarb or one of the salines, are to be avoided if possible, on account of their strong effect on peristalsis and of their uncertainty. Should a copious evacuation follow these measures, the disturbance will at once cease, the tumor and pain will disappear, and the *status quo ante* will be re-established. If this is not the case, but the symptoms continue and the purgative remains without result, there are two possibilities. Either the intestinal musculature is too weak and the dose of the purgative was too small, or the inflammatory process is too extensive and intense to be so rapidly influenced, that is to say, the affection is not a simple typhlitis stercoralis, but a perityphlitis. In the latter case, an increase or an extension of the process, elevation of temperature, and increased frequency of the pulse, extension in area of dulness and of the palpable tumor, and an increase in the general symptoms will be easily determined, and then the treatment suitable for perityphlitis may be continued. In the first case, we must not hesitate to prescribe a second enema, and another dose of castor oil, calomel, or Carlsbad salt, which will have the desired effect.

The only question is, whether we may not do more harm than good, in case a perityphlitis should be present from the beginning instead of a typhlitis, by the use of the aperients. According to my experience there is no such result to be feared. A perityphlitic inflammation of any severity, whether it originate in the cæcum or in the appendix, especially a so-called perforating perityphlitis, can always be recognized on careful examination, and the treatment can be instituted accordingly. This would leave only those cases in which no typhlitis, but an appendicitis or a very mild perityphlitis is present. Under these circumstances, the danger which might possibly arise from an increased peristalsis occasioned by the use of evacuant remedies is obviously more imaginary than real. In truth, the removal of the dammed-up intestinal contents, the relief of the pressure, which the filled intestinal coils exert on their surroundings, the riddance of the products of decomposition which are formed *in loco*, can only be conducive to an improvement in the general condition of the patient. As a fact, I do not find a single case in all my histories in which any bad result followed these measures, and I do not remember one bad case of this kind in my consulting practice. Of course, in such a case it must always remain doubtful whether or not a perityphlitis was present. If it were present, however, this is a proof that the employment of mild purgatives in these slight cases of perityphlitis may be practised without any misgivings.

Appendicitis, Periappendicitis, Perityphlitis.

In discussing the processes which until recently have been included under the term perityphlitis, we know that we are dealing with an inflammation of the appendix and its sequelæ. This has been undoubtedly proved, as already mentioned above, by the minute study of these conditions made possible by the early operations in the living subject. Leaving aside, therefore, the few cases in which the perityphlitis arises from a simple typhlitis, and which are practically not under consideration, we shall discuss only the different varieties of appendicitis. Every appendicitis, whether it be simply catarrhal or suppurative and perforating, must be, in the nature of the case, in itself a "perityphlitis" not only in the sense that the appendix lies in the neighborhood of the cæcum, but also because appendicitis is almost without exception combined with perityphlitic inflammation of the peritoneum. It seems that the proneness of inflammatory processes originating in the cæcum to extend over the neighborhood is so great, and the resistance offered by the wall of the appendix to the source of infection (bacterial invasion) is so weak, that those

cases in which the inflammation confines itself, in the strict sense of the word, to the vermiform appendix, at least in so far as it is expressed by clinical symptoms, are greatly in the minority.

Etiology and Pathological Anatomy.

At the present day there is a fair degree of unanimity concerning the causes which lead to inflammatory disease of the appendix. We have in the first place to deal with the irritating influence which the arrested intestinal contents bring to bear on the latter. As soon as a retention of its contents through any external cause, as a longer retention of the intestinal contents in the cæcum, bending of the appendix, twisting (Treves), or muscular relaxation of its wall is caused, the most favorable conditions are present for its decomposition and the development of bacterial infection. In this manner dietetic excesses, violent exercise after meals, the ingestion of irritating mechanical or chemical substances, and finally and most important, a continued arrest of fæcal matter and the congestive relaxation of the muscular tissue of the intestine combined with it, may prove a cause for the retention of fæcal matter in the appendix. The intestinal contents which have passed into the processus vermiformis and the secretion of the mucous membrane of the appendix form an excellent medium for the development of the bacteria there contained. The appendix itself is swollen—in a state of erection, as the French say—and its walls are thickened; its mucous membrane is reddened and turgid; a sticky, mucous or purulent, grayish-white or brownish substance is found in it, and the higher the internal pressure in the appendix rises, the more congested and paralyzed its walls become in consequence, the less will its contents be able to return into the cæcum, and the more probably will the inflammatory process invade its walls. In this way are formed small particles of the size of a pea to that of a bean, sometimes of the shape of oats or barley grains, which consist almost wholly of fæcal matter. The latter are by no means of stony hardness, but have the consistence of potters' clay in recent cases. In older cases they are harder and drier, but can generally be easily crushed between the fingers. There have also been found concretions with a nucleus in the shape of gall stones or intestinal calculi (of magnesia or lime), eggs of ascarides, fish bones, small bones, bristles, hairs, and seeds of fruit. The significance, however, of these fæcal concretions and foreign bodies for the origin of the appendicitis has been greatly overestimated. The latter particularly have been found more rarely the more painstakingly they have been searched for. Langhald [39] met with them in only a very small number out of

one hundred and twelve cases of perforation of the processus vermiformis. Fürbringer[30] never found the "famed cherry pit" in his seventeen years of hospital service. Osler[31] found eight grains of shot at one time and five apple seeds at another. Fowler, with his rich experience, reports only two cases, in one of which a gall stone was found, and in the other a lime concretion.

I myself found a grape seed only in one case, in all others only inspissated fæces and mucus, or no solid contents whatever. Treves[19] estimates fæcal concretions as occurring in nearly one-half, foreign bodies in nearly one-eighth of all cases, an estimate for the last category which is undoubtedly too high. Only in the rarest cases is it possible to demonstrate the exertion of a direct pressure of these formations on the wall of the appendix, and the causation thereby of pressure gangrene. Usually the significance of these bodies for the origin of appendicitis is only indirect, by forming an obstruction to the evacuation of the contents of the appendix into the cæcum, and in that way favoring inflammation and bacterial growth. In favor of this is also the extraordinary but frequently reported fact (Talamon and others), that the obstructing foreign body usually is found somewhat above the seat of perforation. We do not deny, however, that such "stones" may occasionally be the direct cause of catarrhal ulceration or provoke a greater loss of substance in previously existing catarrhal ulcers. As a rule, however, in cases in which the appendix is still present and a solid body is found in it, the mucous membrane beneath the foreign body or in its immediate neighborhood is found to be swollen, succulent, of a deep red or very dark bluish-red color, yet smooth and not eroded. This is the more readily conceivable, as the peristaltic movements and contractions of the appendix are exceedingly slight, so that any kind of strong or regular friction of the wall with these formations cannot occur. Should the process pass through the wall of the appendix to the serosa, and the inflammation spread thence to the neighborhood, a local circumscribed peritonitis with adhesions of the neighboring intestinal coils will be the result, in which case it may be that the appendix will be enclosed in them, as in a sac.

We have to deal with bacterial invasion of the intestinal wall and of the serosa of the peritoneum, in which are concerned chiefly the bacterium coli and the streptococcus pyogenes and aureus; occasionally also a streptococcus citreus or a diplococcus is found. This has been positively proven by the researches of Tavel, Lanz, and others, and there remains to be determined only what part the individual varieties of bacteria take as regards their injurious effect. It was originally believed that the bacterium coli, whose occasional con-

version from a harmless intestinal parasite to a pathogenic microorganism is unquestionable, was responsible in the first instance; but recently the streptococci have been put forward as the real culprits, so that a mixed infection at least is present. This question, however, is more of interest bacteriologically than clinically. The main fact is that we have in the greater number of cases to deal with a progressive course, with an invasion of bacteria, for whose development the proper medium is present in the cæcum or appendix, while at the same time the walls of these organs are so changed that they are unable to oppose the resistance of healthy tissue to the entrance of the microbes. The further course of the disease depends wholly on the more or less intense virulence of the micrococci.

It is self-evident that this appendicitis will present different symptoms, according as it is acute or chronic in its course, and that it will show a variety of gradations according to the manner in which the processus vermiformis itself and its surroundings react towards the source of inflammation, and according to the particular form of microorganism which is present.

If in the course of time an occlusion of its proximal end should take place, either from cicatricial stenosis following catarrhal ulceration, from impaction of a fæcal concretion, from a bend in the appendix, or maybe from simple tumefaction, the necessary result, as long as any secretion from the wall still exists, will be the formation of a cyst, which will be filled with either seromucous or purulent contents. A cyst of this kind may exceed a walnut in size. Its purulent contents may be sterile in a bacteriological sense, or they may contain the germs of a septic infection. The further course of the disease will depend much upon this, and either an infiltration or a rupture of the cyst wall will take place.

We have thus far considered only those cases in which the inflammation in and around the appendix has developed in a slow and insidious manner. In such cases the peritoneal irritation does not go beyond the stage of an inflammatory serofibrinous exudation, and between the fibrinous layers of the intestines there is not present an appreciable amount of pus. The affection presents an altogether different aspect when there is a sudden rupture of the appendix with extrusion of its contents into the surrounding tissue; this always happens as the result of a necrotic process in its wall, but is by no means always connected with a true perforation. The contents of the appendix may escape either into the free abdominal cavity or into a pocket formed by a previous adhesive peritonitis. In the latter event the process may here come to a standstill, or it may advance farther and lead to rupture through the anterior abdominal wall, or, accord-

ing to the position of the appendix, the pus may take the most devious routes, going to the kidney or liver or passing up through the diaphragm into the pleura or to the lung, or even invading the pericardium, the bladder, Douglas' cul-de-sac, the left iliac fossa, the pelvis, or other parts. It may cause pylephlebitis, abscess of the liver, thrombosis, or erosion of the vessels of the abdomen with its sequelæ, œdema of the right leg from compression of the iliac vein, or erosion of the internal iliac or circumflex artery with fatal hemorrhage (Osler). In 57 cases of Bull, the pus burst through the anterior abdominal wall 28 times, 15 times it found its way into the cæcum, 8 times into the peritoneal cavity, and twice each into the thorax, the rectum, and the bladder. The rupture may, however, also occur in the retrocæcal tissue, thus giving rise to extraperitoneal diffusion. This is the form denominated paratyphlitis, which extends by preference upwards towards the kidney or downwards towards the inguinal region. In all cases, however, the intensity of the symptoms and the extent to which the organs of the peritoneal cavity, and, as dependent on them, the whole organism will be sympathetically affected, will depend on the virulence of the masses which escape from the appendix.

Symptoms and Prognosis.

Appendicitis, as a general rule, is a disease of youth. According to Fitz over fifty per cent. of the cases, according to Treves about thirty-six per cent. are under twenty, sixty per cent. between sixteen and thirty years of age. Although the disease is rare before the third year of life, one case has been reported in which it occurred in the seventh week.

Statistics, in so far as they dispose of great numbers, indicate a preponderance of the male sex. Contrary reports are dependent on the peculiar form of material handled by the observer.

Fowler distinguishes an acute, subacute, chronic, and relapsing form. As chronic latent appendicitis he characterizes those affections which remain in a stationary condition, with occasional efflorescences; as recurrent, those forms of the disease in which the individual attacks are separated by a period of perfect freedom. It seems plain that this distinction is with difficulty carried out in practice.

Relapses are frequent, and occur in from eleven (Fitz) to fifteen per cent. of cases. Generally a recurrence of the attack takes place several times. Treves reports a case in which recurrence took place nineteen times; Fowler was told by one of his patients that the latter had had more than fifty attacks in eight years. It should be

mentioned, however, that recurrences, in the true sense of the word, do not take place when an abscess has formed and pus has been evacuated. The disturbances which then result, and which may have the appearance of relapses, are due to the conditions present in the abscess cavity, but not to a fresh perityphlitic attack. This is in the nature of the case, as the appendix is here already destroyed.

The *mortality* of perityphlitis varies, according to different reports, between 10 and 20 per cent. The reason of these variations is, that the material used as the basis of these statistics is a variable one, derived partly from surgical and partly from medical practice. Porter[32] finds a mortality of 17.23 per cent. in 448 cases; Fitz estimates the mortality in cases treated internally at about 11 per cent. On the other hand Renvers,[33] Sahli, Rotter, Sonnenburg, and others find that from 80 to 90 per cent. of all cases recover without operative interference. It will therefore not be much out of the way to estimate the mortality of perityphlitis, where a judicious internal and operative treatment is pursued, at less than 10 per cent.; in other words, perityphlitis is a disease which, if treated properly, presents a comparatively favorable prognosis. Especially dangerous are only the very acute forms, which lead at once to grave symptoms. According to Fitz, no less than 68 per cent. die within the first eight days, and two-thirds of this number die between the fourth and eighth day inclusive.

One glance at the preceding etiological and pathologico-anatomical considerations will show that the clinical symptoms of appendicitis may take on quité diverse forms.

In simple catarrhal appendicitis the inflammation runs its course without any symptoms, and need hardly be accompanied by particular pain. The latter is felt only when a stronger irritation causes greater peristaltic movements of the vermiform appendix or the adjacent portion of the intestine. It then appears in the form of frequently repeated colicky pains in the ileocæcal region, occasionally also in the region of the umbilicus, or in the epigastrium (colica appendicularis). These pains may be dull, boring, or stabbing, occur at irregular intervals, occasionally in conjunction with constipation, and may take on the character of typical relapses, which are separated by long intervals of perfect well-being. Their nature is so little characteristic that they may not be referred to the intestine at all, but may be taken by superficial observers for nervous or hysterical pains, especially in the case of women. A skilfully made examination, however, will enable us to feel the swollen appendix, and to move it about, if sufficiently deep pressure can be made, on its bony bed, or to push it upwards or to one side after insufflation of air into the intestine.

Three years ago I was consulted by a lady from Sweden, who had spent the winter in Nice, on account of a supposed hysterical affection. Her debility here increased to such an extent that she had to be transported to her home under the care of a physician. The patient was seen by me in consultation while in Berlin on her way home. One constantly reiterated complaint of this patient, besides many others which were indeed hysterical, was that of a pain in the right iliac fossa radiating towards the liver. Similar attacks had been experienced two years before and subsequently, characterized by sudden pain with diarrhœa and vomiting, but the physicians in attendance were not able to find sufficient grounds for the diagnosis of perityphlitis. A swelling of the liver and a tumor in the vicinity of this organ were said to have existed. I could with positiveness palpate the swollen appendix of the size of the little finger, and induced Professor Sonnenburg to perform a resection of the appendix, which was recorded by him in his work[34] under Case V. It was found that the previously palpated cord was the indurated, rigid, unperforated vermiform appendix. The peritoneal cavity around it was entirely obliterated by numerous adhesions. In the appendix itself, which showed a greatly thickened muscular structure, submucosa, and mucous membrane, and turbid sanguineopurulent contents, two shallow ulcers were found, but no fæcal concretions. The patient was perfectly cured and enjoys the best of health.

In this case we had surely to deal with a recurring appendicitis, the recognition of which was only possible because it was carefully searched for, thanks to the latest discoveries in this field, and the *corpus delicti* was also discovered, thanks to the excellently developed technique.

These conditions, after once the attention has been called to them, are not at all rare. I have twice had occasion during the past two years to make the diagnosis of chronic recurrent appendicitis under conditions in which the disease presented only the symptoms of a chronic enteritis, although the seat of greatest intensity of the pain was in the right iliac region. In every case the swollen appendix was plainly appreciable to the touch. Twice resection of the appendix was done by my colleague Lindner, in the first case with excellent result; in the second case, convalescence was greatly prolonged by the occurrence of hectic fever. Microscopical examination of the extirpated, greatly thickened appendix showed tuberculous nodules in it, and gradually a general debility was developed due to a latent tuberculosis, while at the same time the local intestinal symptoms entirely disappeared.

A second group of cases is characterized by the appearance of a larger or smaller crescentic-shaped area of dulness and a tumor-like resistance situated at the border of the ilium. These cases are marked by more pronounced symptoms of peritoneal irritation, such

as circumscribed or diffuse pain in the abdomen, distention of the latter, dyspeptic phenomena, perhaps vomiting of food particles, bile, or medicine which has been taken, sometimes stercoraceous vomiting, constipation, and a slight rise of temperature. Irregularity of the bowels, usually constipation or more rarely diarrhœa, precedes the attack. The urine is scanty, high colored, frequently but by no means always containing indican, and gives the Burgundy-red reaction (see page 128). The local symptoms proper appear in an acute or subacute form, without being particularly violent. These are the cases which are most frequently seen by the general practitioner. They were formerly simply classified with the cases which will next be studied as perforating typhlitis, although they differ very materially from the latter by not setting in, as a general rule, with such violence, and by not being accompanied by a continued high temperature and increased pulse rate. The tumor and pain are found in a greatly preponderating number of cases to the right of the median line, usually in the ileocæcal region. Cases of a left-sided position of the appendix are so rare that they are always reported as medical curiosities. Fowler reports three cases. I myself have never seen any. Nevertheless the possibility of their occurrence should not be forgotten, so that we may protect ourselves against diagnostic errors. The tumor may consist only of adherent intestinal coils, or of a serous or serofibrinous exudation, free from bacteria, of the presence of which exudate we may obtain conclusive evidence by exploratory puncture. It seems incomprehensible to me how some authors, for example Sahli, can dispute the existence of such an exudate, for years ago I convinced myself of its presence by puncture in the living subject, and I have occasionally also found it at autopsy. Sonnenburg very recently reported a case which is absolutely classical in which a turbid, serous exudate was found during the operation. As a rule, the process is confined to a purely adhesive inflammation, and one is astonished to find at the operation, in place of the expected exudate occupying the area mapped out by the dulness, nothing more than tense intestinal coils filled with air and a little fæcal matter. Under these circumstances the adhesions of the intestinal coils to each other must obviously influence the percussion sound in a similar manner as the mute influences the vibrations of a violin string, and the œdematous infiltration of the tissue and the plastic exudation must have a particular influence on the resistance.

The course of these cases is generally a favorable one. According to Renvers, Sonnenburg, and Sahli, who have seen a very great number of cases and whose experience has been very similar, about ninety-one or ninety-two per cent. of all cases recover without an

operation. The exudation may, however, take on a purulent character by direct propagation of the infective process through contiguity or by way of the lymphatics, and go on to a progressive purulent adhesive peritonitis. We shall refer in the proper place to the influence which the treatment has upon the result.

By far the most frequent cause for the formation of large perityphlitic accumulations is furnished by perforative typhlitis or appendicitis. Here we meet with the classical form of the so-called perforating perityphlitis. It should, however, be again mentioned here that we do not by any means always have to deal with a true perforation, *i.e.*, a necrosis and ulceration, meaning thereby an actual loss of substance, for the sudden emigration of virulent bacteria from the appendix to the peritoneum is in itself capable of producing the acute symptoms to be described below. There are numerous cases on record in which, notwithstanding the typical array of symptoms, a perforation was not to be found. In the midst of health, or after some insignificant disturbance of intestinal digestion, following some trauma or the swallowing of a foreign body, such as a splinter of bone, fish bone, seeds of fruit, and the like, a violent pain may be experienced in the abdomen, with simultaneous enlargement and tension. At first the pain may be diffuse, but soon it will become localized in the right side, which will become tender on pressure and resistant. The temperature rises to 39° C. (102.2° F.) and over—in one of my cases 40.6° C. (105° F.) was reached. This is not, however, always and necessarily an accompanying symptom of perforation. In rare cases there may be rise of temperature and no acceleration of the pulse rate, or both pulse rate and temperature may even be lowered, as the expression of severe collapse. The pulse becomes small and frequent, the skin is dry, or, if symptoms of collapse make their appearance rapidly, is covered with cold sweat. As symptoms of extreme peritoneal irritation, and of the pressure exerted by the exudation, there may occur pain on urinating (*urina spastica*), pain in the testicles, and various paræsthesiæ, chilly sensations, formication, etc., of the lower extremities. The patients are, however, usually so much occupied by the abdominal pain that they do not pay a great deal of attention to other sensations. Much more distressing is the obstinate hiccough, which is not infrequently present. As a rule, there is constipation occurring as a result of the paresis or compression of the intestinal coils by the exudation; I find, however, in my own histories as well as in the reports of cases by other writers, the presence of diarrhœa occasionally noted. The patient is unable to move and generally lies with somewhat flexed limbs and the respiration is quite shallow. He avoids speaking

aloud, sneezing, or coughing, that is, every movement which might increase the pain. The anxious facial expression, the deeply sunken eyes, and the weak voice complete the picture of a grave disease. After the patient has recovered from the first shock, remissions in the fever generally take place, the pulse becomes stronger, the pain less, and there is a gradual amelioration of his condition. Or else there is either the picture of a general peritonitis under nearly continuously high fever, and the accentuation of all other symptoms with eventually collapse and death, or that of a septic infection which takes its course, accompanied by repeated chills, slight icterus, repeated accessions of pain, or perhaps a false euphoria. This difference in the symptoms is dependent on the special character of the perforation, the condition of the peritoneum, the composition of the pus, and other factors which have been already referred to above. Those cases in which a perforation and suppuration of the appendix enclosed in a hernial sac occurs are exceedingly rare. On the other hand there have frequently been observed erosions of the larger vessels followed by fatal hemorrhage, or extensive formation of thrombi. A pylephlebitis, with metastatic abscesses of the liver, which has its origin in the network of the veins of the cæcum and the appendix, may also arise.

In the chronic form, and when a slight improvement sets in, the case may drag along slowly or purulent inflammation may extend from the site of perforation, or the diseased part may become encapsulated. Clinically the spread of the inflammatory process will be indicated by a renewed rise of temperature, renewed and increasing pains, which may extend in various directions, chills, jaundice, symptoms of subphrenic abscess, of pleural effusion, of inflammation of the lungs or pericardium. In every case, however, the formation of a tumor and a corresponding dulness will be found, which is due, at first at least, only in very slight measure to liquid purulent effusion, but rather, as above stated, to the œdematous infiltration of the tissues occurring at the same time, and to the soft fibrous or purulent exudate on the latter. In rare cases a distinct crepitation is present over the tumor.

A *spontaneous cure*, or better recovery without operative interference, may occur in one of three ways. In the first the abscess becomes encapsulated, the pus loses its virulence and is absorbed, the tumor disappears, fever and pain gradually cease, and the patient is in reality or seemingly cured. In this case danger to life is for the present removed, it is true, but frequently the patient suffers severe and lasting injuries from the result of the abscess and the adhesions of the intestines among each other. The occurrence of a sudden bursting

of the abscess hangs like the sword of Damocles over the head of the patient as long as there is still pus present. These are the cases in which in seemingly perfect health a fatal peritonitis may occur, for which no cause can be found at first, if the previous history is imperfect. The following is the report of a case of this nature:

H. v. K——, 29 years of age, was admitted to hospital presenting the symptoms of a severe general peritonitis. The patient was so weak that the previous history could be obtained only with much difficulty. He stated that he suddenly felt an intense pain in the abdomen below the navel while practising gymnastic exercises a week before, and at once felt exceedingly ill. He had never had hemorrhage from the stomach or bowels, nor gastralgia. His bowels were always constipated, so that he always had been obliged to have recourse to purgatives or enemata. He did not remember any previous attacks of inflammation of the bowels, and only recollected indistinctly having had pains in the abdomen for a few days, about nine months ago. Although the possibility of a perforating ulcer of the stomach seemed to be excluded by the previous history, and there were no particular symptoms pointing to the presence of an intestinal ulcer, the question of an operation was discussed. The patient, however, collapsed too quickly, and was dead within forty-eight hours. The autopsy disclosed a diffuse peritonitis, with a large quantity of pus in the abdominal cavity, and an encapsulated, ruptured perityphlitic abscess, which obviously dated from some time back. No traces of the appendix could be found in the thickly indurated wall of the cæcum.

A second method of cure is by the more or less early rupture of the abscess into neighboring hollow organs. There are cases recorded of rupture into the cæcum, the colon, the small intestine, the bladder, the vagina, and the pelvis of the kidney. A number of authors even go so far as to assert that this termination is the usual one. In opposition to this it may be remarked that the only proof of this, namely, the appearance of pus in the dejections or in the urine, can with difficulty be established, and in the absence of this evidence the only other foundation for such an assumption would be the post-mortem findings. Such observations have indeed been made, but they are exceptional. A third possibility is that the abscess may find its way externally, and either rupture spontaneously or be opened with a simple incision. This may occur in different places, as the pus, from the position of the patient on the back, is liable to take its course backwards and downwards, appearing in the lumbar region, and perhaps forming a subphrenic abscess. In this the pus may rupture through the diaphragm and make its appearance in the pleural cavity, and even in the lung. H. Grawitz has published such a case which came under my observation a number of years ago. On

the other hand, the pus may break through below Poupart's ligament, or burrow still deeper in the thigh. The rupture in these cases of acute perforation, as they may seem, may occur without the appearance of any previous symptoms of disease, or there may have been certain symptoms which would lead a careful observer to suspect the threatening danger, or at least would direct his attention to the presence of some disease of the ileocæcum. A careful and searching review of the previous history, directed to this point, may bring to light a number of facts which point to antecedent attacks of appendicular colic and may lead to a suspicion of the probable existence of disease for a long period. The fact, however, that in a number of cases all data which might point to a previous disease are absent is the surest proof that the appendicitis as such may run its course without any symptom whatever, for a sudden perforation without any antecedent disease is obviously an impossibility.

Appendicitis and Rheumatism.

In recent times attention has been drawn, from various directions, to the relation between articular rheumatism and appendicitis. Yeo,[25] for example, describes the case of a young girl who had previously suffered from articular rheumatism. She was suddenly taken ill with the symptoms of a perityphlitis with high fever, and on the fourth day pains of the right knee, shoulder, wrist, and elbow made their appearance, while at the same time a systolic murmur was to be heard at the apex of the heart. These symptoms rapidly passed away on the administration of salicylic acid, but a few days later pain and tension reappeared in the right iliac fossa, and on palpating here a gurgling could be felt. This attack was also promptly controlled by salicylic acid. Yeo named this affection, after the analogy of those rare cases of rheumatic peritonitis, a rheumatic perityphlitis. It should, however, be noted that no tumor could be demonstrated in this case, and therefore the diagnosis of perityphlitis does not seem at all clear. In two other cases, reported from Brazil, in which an appendicitis was complicated with polyarthritis, we miss any direct causal relation, and there seems to be no reason why both affections might not have accidentally existed at the same time. The same applies to similar cases which Sutherland[26] has gathered from literature, and on the strength of which he comes to the conclusion that under certain circumstances appendicitis and rheumatism may be dependent on a third unknown poison, which may produce inflammation. Spillmann and Ganzinotti[27] collected fifteen cases of this kind, all of which, however, as Treves justly remarks, will not bear strict criticism. It cannot, of course, be denied that appendi-

citis occasionally follows a cold. If every cold be therefore referred to some rheumatic or infectious cause we may correctly speak of an infectious cause of appendicitis, one due to the sudden action of an external agent, and not to the bacteria, which has been referred to and discussed before. If any relation exists in reality between appendicitis and polyarthritis, it would be in the highest degree surprising that this has not been discovered before, in view of the extraordinarily frequent occurrence of the latter. The same is true of the view lately expressed by Goluboff,[11] who, because of the more frequent occurrence of appendicitis at certain seasons of the year, looks upon it as an epidemic affection, at least as an infectious disease *sui generis*, which is exclusively confined to the appendix in the same way as angina follicularis is to the tonsils and dysentery to the intestines. In this case also there is up to the present an entire absence of practical proof, and the question is as yet a purely theoretical one.

Diagnosis.

Easy as it may be to recognize a perityphlitis running its course with the classical symptoms above detailed, *i.e.*, a simple or perforative appendicitis, equally difficult or even impossible may the diagnosis become when the ileocæcum is in an abnormal position, or if the patient comes under observation at a time or in a condition in which the picture of the disease is indistinct or obscured by complications. Two facts are here of determining importance, and should always be most carefully looked for: (1) The previous history of the patient; and (2) the presence of dulness or of a tumor in the ileocæcal region, its circumference, and its position in relation to neighboring organs. In no doubtful case, and never in children, should we fail to make an examination by way of the rectum or the vagina. We do not possess one single reliable symptom by which we can estimate the gravity of the process, *i.e.*, its probable course. American physicians have laid stress on the disproportion between a moderately high temperature and a very frequent and small pulse as an ominous sign. This fully agrees with my own observations. In three cases, in which perforation occurred under our own eyes, the temperature varied between 38° and 38.8° C. (100.4° and 101.8° F.), rising to 39.2° C. (102.5° F.) in one case, while the pulse rate was from 112 to 132. Two of these cases were operated on, the third one died before operation. In each case there was an extensive purulent exudate and a necrotic, *i.e.*, gangrenous, appendix was present. But even when the disease runs its course with mild and not particularly severe symptoms, we are not secure against the occurrence of dis-

agreeable complications, such as may arise from a perforation, extension of the exudate, or the like. One of the cases above mentioned had a normal temperature for twenty-three days, and was seemingly a very mild form of appendicitis simplex, when suddenly the temperature rose to 39.6° C. (103.3° F.), perforation occurred, and within fourteen days the patient died with symptoms of general peritonitis. Every case of perityphlitis should therefore be looked upon as a very grave disease, whose progress is absolutely uncertain. This not only applies to the attack itself, but in the same measure to what the patient has to expect in the future. We have already repeatedly called attention to the tendency of recurrence.

The numerous complications, and the particular symptoms which may be occasioned by the propagation of pus, can generally be easily recognized as such and as a result of the primary disease focus. The escape of pus into the free peritoneal cavity, with the resulting general peritonitis, is sufficiently characterized by its well-known symptoms. On the other hand, we may meet with cases in which the slow burrowing of pus downwards and between the intestinal coils causes no marked symptoms whatever. In these cases the persistence of the fever, which is occasionally interrupted by apyretic intervals, and the increasing debility of the patient, will point to the fact that the process has not died out, but like lava under the ashes is still glowing. We have to differentiate the affection from several other conditions.

Renal colic, especially in its beginning, or when the local symptoms are indefinite and examination of the urine gives negative results, the patient being uncertain in the localization of the pain, may give rise to mistakes. The liability to error is increased by the fact that the radiation of the pain towards the os pubis, scrotum, and testicles, the tenesmus, and the dysuria may be the same in both conditions. Very soon the diagnosis will, however, be made clear by the characteristic seat and extension of the appendicitis. Fowler gives the following differential diagnosis (p. 168), which I accept, although with some reservation as regards the dogmatism which is inherent in all such schematic tables.

It may be more difficult to recognize the true nature of a suppurating appendicitis when it has extended outside of the right iliac fossa. To this class belong perinephritic phlegmons, hæmatoceles, and salpingitis or pyosalpinx in women (Richelot), cholelithiasis or hepatic abscess, suppuration in a hernial sac of the scrotum (Thurmann, Monks, Jalaquier) into which the cæcum together with the appendix have slipped.

Chronic appendicitis, and especially tuberculous typhlitis, with

thickening of the intestinal walls may be mistaken for a cancerous growth.

Appendicitis.	Hepatic colic.	Renal colic.
Pain around the umbilicus and in the epigastric region, not radiating from these points, fixed painful point in the iliac fossa.	Pain in the epigastric region, radiating to shoulder and angle of scapula, arising from the gall-bladder as the fixed point.	Pain radiating to inguinal region and testicle, occasionally to the rectum, when at stool, also tenesmus.
Greatest tenderness in the right iliac fossa, particularly at McBurney's point.*	Great tenderness below the arch of the ribs, slight over gall-bladder.	Greatest tenderness behind over the pelvis of the kidney, in front the maximum point of tenderness is over Poupart's ligament.
Vomiting may be present, but is usually not continuous.	Vomiting frequent and not to be suppressed.	Vomiting is not a frequent nor prominent symptom.
The bladder and testicles are very rarely symptomatically tender or painful.	Bladder and testicles give no symptoms.	Bladder irritable, dysuria and tenesmus of the bladder; occasionally hæmaturia; testicle retracted.

Recurrent perityphlitis occasionally simulates a tuberculous peritonitis, and we are the less able to distinguish these two affections from each other as the appendicitis may in truth be tuberculous.

A purulent collection may itself cause absolute constipation, or this condition may be produced by large doses of opium. In both cases, particularly if the previous history is unknown and the quantity of opium taken cannot be estimated, the possibility of an internal strangulation or an invagination must be taken into consideration. The absence of blood in the stools, and the relatively mild character of the constipation, will in these cases make a diagnosis possible. If, however, no relief is obtained by injections or the administration of purgatives, if the abdominal walls remain tense, vomiting appears, and the pulse becomes small and frequent, we are apt to have

* McBurney's point is found in the following way: A line is drawn from the umbilicus to the anterior superior spine of the ilium on the right side, intersecting a second vertical line drawn parallel to the external border of the right rectus abdominis muscle, and this point of intersection is usually the seat of the greatest tenderness. Should it be difficult to find the external border of the rectus abdominis exactly, McBurney's point may be determined by placing the tip of the thumb on the spine of the pubic bone, and the tip of the middle finger on the umbilicus. Now the index finger is extended at a right angle and touches the abdomen at McBurney's point (half-way between the points mentioned). Although the base of the appendix may not always correspond exactly to this point, nevertheless the presence of a localized tenderness within these narrow limits in conjunction with other symptoms is of diagnostic value.

a very uncomfortable and anxious time of uncertainty in diagnosis, which may continue for forty-eight hours or more.

In children, also, the differential diagnosis of appendicitis from other affections will be very difficult, as these little patients are not able to give an exact description of the pain or a satisfactory account of the beginning and the course of the disease. If the inflammation becomes chronic in these cases, and involves the psoas muscle, a flexion of the thigh at the hip may occur, which is liable to simulate a coxalgia (Gibney).

In all febrile cases in which a typical dulness can be demonstrated, the question arises as to whether pus is present or not, and this cannot always be solved by the results of exploratory puncture. It stands to reason that a puncture in these cases can only be decisive when a positive result is obtained. A great deal has been said for and against this measure. In my opinion, which is in perfect accord with that of a number of clinicians of large experience, puncture, if properly performed, is an innocuous and in itself permissible but unreliable measure, and as a general rule it is superfluous. A needle 6–8 cm. ($2\frac{3}{5}$–$3\frac{1}{5}$ in.) long, and not too fine should be employed, and it, as well as the field of operation, must be thoroughly disinfected. I insert it for its entire length at the point of greatest dulness, preferably close to the edge of the ilium, and as soon as it has entered I pull the piston back as far as it will come so as to obtain a very strong aspirating force. If the accumulation has been reached, the pus will well up into the syringe. Should this not be the case, the needle is slowly withdrawn, millimetre by millimetre, and in this way any collection of pus which may happen to lie on this route will be struck. I have never seen an infection of the intestinal coils or of the abdominal wall result from this procedure, although in former times I have had recourse to exploratory puncture in numerous instances. But even in case this should happen, it would seem to me to be relatively a small matter as compared to the grave process already existing. There is, however, another objection which has been raised to exploratory puncture; for since, according to the belief now prevailing, pus is always present though possibly in small quantity, we may well ask whether it is ever necessary to demonstrate its presence. According to my opinion, this is unnecessary in the greater number of cases.

Occasionally, however, an ocular demonstration to the patient or his relatives of the presence of pus may be necessary in order to induce him or them to consent to an operation, although in most cases this will be superfluous if the physician possesses authority and the confidence of his patients. It is hardly necessary to mention that

when there is any doubt as to the nature of the fluid which has been obtained in this way, it should if possible be examined microscopically.

The diagnosis of a relapse must naturally be based on the evidence of a preceding attack. The previous history, the recurrent pain in the right iliac region, spontaneous pain or tenderness on pressure, the discovery of a thickened appendix or a tumor, the generally present irregularity of the bowels, and the presence of indican in the urine make the diagnosis of these cases certain. It should never be forgotten that the vermiform appendix may have an anomalous position and may lie in any part of the abdominal cavity, even in the left hypochondrium (Lenander); however, as Penzoldt justly remarks, we should not on account of these exceptions lose sight of the rule.

Treatment.

"Perityphlitis belongs to the surgeon," has been until lately an assertion defended with emphasis by many surgeons, but which has never received the assent of the general practitioner, and never will. According to the experience of general practice, and the statistical results of Sahli, Renvers, Guttmann, Leyden, Fürbringer, Hollander, Rotter, and the majority of French physicians, from ninety to ninety-one per cent. of all cases of perityphlitis, taken in the widest sense, recover without any operation. It would therefore smack of insanity to subject every case of perityphlitis to the uncertainties of an operation. The question as to when the surgeon's knife is required we shall discuss later, for the present we will discuss the main outlines of the medicinal treatment.

The idea of *preventing perityphlitis* by a special diet or the avoidance of certain articles of food, or even by a prophylactic extirpation of the vermiform appendix, which has been proposed in all good faith, is utopian and is not entitled to any serious consideration. The wisdom of not indulging in certain harmful practices, as the swallowing of fruit seeds, bones, fish bones, and the like, or of avoiding trauma, is self-evident, and the command not to swallow the seeds of fruit should in particular be forcibly impressed on children. But no one swallows bones or fish bones purposely, and children or even adults cannot be prevented from swallowing a seed occasionally, or from receiving a blow in the region of the lower abdomen during play, gymnastic exercise, cycling, and other active sports. The well-meant warning against such eventualities belongs to the same class as the injunction "not to become angry," which may be easily laid down by the physician, but can be obeyed by the patient only with great

difficulty, if at all. More likely is it that we may do some good by regulating the diet, instructing our patients to abstain as far as possible from articles of food which give rise to a large residue, for example, vegetables and other substances which contain much cellular tissue (asparagus, beans, coarse bread), and from those which tend to induce constipation, by enjoining slow eating and thorough mastication, and by counselling them to rest a while after meals whenever this is possible. The greatest weight should, however, be laid on the regulation of the bowels, especially in those cases in which a certain disposition to constipation exists, whether this be an acquired habit or a familial peculiarity (Sahli). That fæcal accumulation very frequently forms one of the etiological factors of a perityphlitis cannot be doubted, however little one may estimate the importance of the local fæcal tumor. Therefore, certainly no harm can come and probably some benefit may be gained from the observance of a rule which is in itself salutary, and what is of greatest importance, it is possible to follow.

The *treatment of the attack proper*, consists, in the first place, in the enforcement of the most absolute quiet, both general and of the intestines. The pain as well as the general malaise compel the patient to seek his bed, to assume a position in which the abdominal muscles are as much as possible relaxed, and to avoid all violent movements of the abdominal parietes, such as in coughing, sneezing, deep breathing, and the like. The stools and urine should be passed into a bedpan, the utmost gentleness should be observed by the attendants in making the bed, giving enemata, etc., and this precaution should be insisted upon far into convalescence, even in cases which run their course without the occurrence of fever.

The diet should be bland, at first consisting only of small quantities of liquid nourishment. The amount of food taken will usually for the first few days be very small, even without the physician's interference, because of the anorexia present. It will be best to allow nothing but some boiled water or ice pellets during the first twenty-four to thirty-six hours. The objections to this course on the part of unreasonable relations or of the patient himself, I always meet with the absolutely true remark that I have never yet seen one of these patients die from starvation. It should always be borne in mind, however, that a certain amount of strength must be preserved in case of an operation becoming necessary. Some cold or lukewarm milk, small quantities of oatmeal, farina soup, or a tablespoonful of bouillon made from white meat, will be sufficient to maintain a fair amount of strength. The administration from the beginning of nourishing enemata, as proposed by Sahli, is a theoretical notion which could

seem practicable only when measured by the autocratic regulations of a hospital director, but which is partly unnecessary and partly directly injurious in general practice, on account of the manipulations to which the patient must be subjected. In proportion as the fever and the pain abate and the existing exudation remains stationary, the danger of a perforation disappears, and the diet may gradually, but only very gradually, be made more nourishing. Bouillon with egg, raw or very soft-boiled eggs, milk and wine soups, very tender soft meats, the crumb of wheaten bread, or better toast, later mashed potatoes, rice, groats, marrons, etc., may be administered in small quantities. All meats with coarse fibre, smoked articles, vegetables, all kinds of raw fruit and preserves, fermented drinks and those containing quantities of carbonic acid gas, are to be avoided for a long time after recovery. The beverages which may be allowed are tea, red wine, milk with perhaps a little coffee or cognac added, or cocoa. The advisability of occasionally allowing to patients who may be especially debilitated stronger stimulants, such as heavy wines, punch or champagne, in spite of their secondary effects, is a matter of course.

The first indication for *medicinal treatment* is the immobilization of the intestine by opiates. At the same time nothing would be more erroneous than the indiscriminate administration of opium according to a fixed rule and in large doses. The treatment of this disease by opium has been condemned by many authorities, especially by surgeons, mainly on the ground that both patient and physician may thereby be misled into a false sense of security, and the actual danger may be thus overlooked by them. This danger is, I believe, exaggerated, for any half-way careful observation of the patient, even in the absence or in spite of the statements of the latter, will keep us informed concerning the course of the disease process, and the occurrence or approach of dangerous symptoms which raise the question of an operation cannot then be overlooked, even when the patient is under the influence of moderate doses of opium. Of course, it is understood that the administration of this drug should be kept within the bounds of common sense, and not pushed to the verge of opium poisoning.

A few days ago there came under my care a school-boy, 16 years old, who had received, during the first four days of an attack of perityphlitis, so much opium from his physician before coming to the hospital, that he had during the following seven days—that is to say, for eleven days altogether—absolutely no passage of fæces or even of flatus, nor did the bowels react to high irrigations of castor oil, castor oil by the mouth, or lastly to croton oil. The enormously distended abdomen, through which the inflated individual intestinal

coils could be observed, and the entire absence of any intestinal motion, made me think very seriously of a possible intussusception or intestinal strangulation. In view, however, of the absence of any fæcal odor from the mouth or of stercoraceous vomiting, although the patient frequently did vomit, and the fact that the pulse was strong and only 84, and that no localized pain or tumor could be discovered, I did not abandon my diagnosis of simple obstipation, although the temperature eventually rose as high as 38.3° C. (102° F.). I finally ordered the administration of 0.5 gr. of calomel, and at the end of the seventh day after the patient came into the hospital some flatus was passed; in a short time there were four passages from the bowels and the diagnosis was fully cleared up.

The best criterion of when and how much opium should be given is the pain which is caused either by the motion of the intestine and consequently of the appendix, or by perforation with its sequelæ. It is well to begin with a full dose, say for adults twenty to twenty-five drops of the tincture, or 0.1–0.15 gm. (1½–2½ gr.) of the extract, for children up to ten years of age as many drops of laudanum as the child is old in years, to be repeated later, according to the pain, three to four times a day, in one-half and even less of the initial dose. This must, however, depend altogether on the individual indications present in each case. As soon as the pain has ceased the opium is to be stopped, and a purely local treatment, or one directed against the existing dyspepsia, should be instituted. In this way we are often able to arrest the vomiting and the hiccough which is occasionally present, and the patient experiences a feeling of euphoria, which seems to me to be very desirable in spite of the previously mentioned objections.

In what form the opium is administered, whether as tincture or as extract, by the rectum in solution or in suppository, by the mouth, or hypodermically, is quite irrelevant, and depends on individual conditions.

The application of cold in the form of ice-cold compresses, a large ice-bag, or Leiter's coils, or the application of warm compresses, with sedative infusion of camomile, valerian, etc., or of hot poultices, will serve as adjuncts to the opium treatment.

Whether the one or the other should be used depends to a great extent on the subjective sensation of the patient, for we are not in possession of any experimental or clinical data which would enable us to decide off-hand. The general custom is to begin with the cold applications, because cold is believed to have an antiphlogistic action, yet we frequently observe initial inflammations subdued by warm applications. Inunction with blue ointment so frequently used formerly, which I have seen regularly applied even by Traube, seems

now to have been quite generally abandoned. The application of leeches to the ileocæcal region has also never been productive of any real good in my hands.

An important question is when and how the intestines are to be evacuated? The least harmful way of ridding the lower portion of the intestine of accumulated fæcal masses is undoubtedly by means of an irrigation of cold or lukewarm water or camomile infusion. This should not consist of more than 500–750 c.c. (a pint to a pint and a half) of water, and should be given under slight pressure, care being taken to spare the patient as much as possible in passing the rectal tube. The acute stage of the disease should always be over before an attempt is made to move the bowels, four, five, or even six days of obstinate constipation being allowed to pass without any scruple. As long as symptoms of advancing disease and of peritoneal irritation of a general nature are present, it behooves us to be particularly careful. Frequently, however, these small injections are of no use. The intestines are so greatly paralyzed by the previous administration of opium, or the fæces are so hard and firm that a movement cannot so easily be obtained. In these cases I have administered, without doing any harm and with good results, high irrigations of one and a half and two quarts of water to which I have added 30–40 gm. (an ounce or more) of castor oil mixed with the yolk of an egg, so as to make a sort of emulsion. The internal administration of purgatives should be as far as possible avoided, but when such is necessary castor oil, above all others, is to be recommended, given either in one large dose of 30 gm. (one ounce) to an adult, or in the form of an emulsion (50:200) in doses of one ounce every two hours. Perhaps medium doses of calomel or of Carlsbad salts may be given, but only after all acute symptoms, including pain on pressure, have disappeared, and the increasing distention of the intestines with gas and solid matter imperatively demands a movement. The exhibition of senna or jalap, either internally or by enema, which has been recommended, or of the stronger vegetable purgatives and drastic cathartics, is to be avoided altogether on account of the irritating qualities of these drugs.

When has the time for operative interference arrived? The answer to this question is dependent in no small measure on individual factors, with the result that much uncertainty and doubt often arise. It is necessary therefore to adopt certain rules, according to which, as far as possible, the decision may be dependent. I resign the patient to the surgeon's charge under the following circumstances:

1. As soon as a perforation has occurred, followed by a general peritonitis. In such a case laparotomy should be done as quickly as

possible. Statistics have shown that the greatest chances for life are within the first twenty-four to thirty-six hours after rupture. Later operations are not very successful.

2. When, in the course of the disease, with acute symptoms of advancing inflammation there is formed a purulent collection in such a location that the operation would amount to nothing more than the opening of an abscess, as for example in the right iliac fossa, in the lumbar region, in the subphrenic region, in Douglas' cul-de-sac, in the perirectal connective tissue, etc. An operation of this kind will of course become necessary only in a later stage of the disease, probably about the fourth or fifth day. It need not, however, be necessary for the abscess to become apparent by pointing or fluctuation. A febrile movement of some duration, and other symptoms indicating constitutional disturbance or septic infection, are to be our guide in leading us to ask for the assistance of the surgeon, even though the pus is deeply situated and not easy of access. In these cases I lay particular stress, as is also done by American practitioners, on the disproportion between the pulse and temperature, of which mention has already been made. A temperature in the neighborhood of 38° C. (100.4° F.) and a pulse above 110 or 120, is always of evil omen, and for me is a sign that operative interference is indicated. The danger under these circumstances, that there may be a loosening of the adhesions between the intestinal coils with consequent effusion of pus into the abdominal cavity, is of hardly any weight with an able surgeon. I have never seen such an occurrence in any of the numerous operations for perityphlitis which I have witnessed for a number of years. It seems self-evident that the appendix, or as much of it as may yet be present at least, should be removed at this time, yet authors differ on this point. Renvers, for example, decides this question only very conditionally, because the vermiform appendix, as far as it is diseased, will be thrown off, if a broad incision and drainage of the pus cavity are effected. Casparsohn[39] thinks that in one case the fatal result was due to the fact that the strength of the patient was not sufficient to withstand the operation, which was unduly prolonged by the search after the appendix. This point, however, is one which should be left for the surgeon himself to decide.

3. Another indication for operative interference is, to my mind, furnished by those cases of recurrent perityphlitis in which the frequency and increasing severity of the attacks not only offer a continual menace to the patient, but also interfere with the earning of his living. I have lost three cases of recurrent perityphlitis which I believe might have been saved by the timely removal of the appendix. While it is often in acute cases a toss up whether or not to operate,

the necessity of an operation in recurrent perityphlitis is governed by absolutely no rules whatever. Shall an operation be performed after the second attack, or shall a third or a fourth be waited for? Here it will be necessary to take into account all the accompanying circumstances, among which are the possible hereditary disposition of the patient and the fact as to whether the attacks are increasing or decreasing in violence. As a general rule, these prophylactic operations will usually have to be done during an interval between the attacks, and most patients will give their consent only after having passed through a number of them.

4. Finally an operation is also indicated in those cases of chronic appendicular colic which do not present the classical symptoms of perityphlitis, but run their course with indefinite and obscure symptoms, and can be properly diagnosed only when the swollen appendix is appreciable on palpation.

In all other cases I advise against an operation, for I believe that to operate on a case of typhlitis of medium severity the symptoms of which do not relegate it to any of the above-mentioned categories, simply because there is a possibility of a fatal ending or of a recurrence, is unjustifiable surgical interference, which could only be approved if the operation were as easy and safe as the pulling of a carious tooth. Statistics also uphold this view. Among nine hundred and thirty cases of operation which Renvers has collected from the literature, from fifteen to twenty per cent. only were cases of perityphlitis simplex, and seventy-five per cent. were cases of perityphlitis purulenta. I express here my personal convictions, according to which I have acted for a number of years, without as yet ever having had any reason to regret it. Frequently, however, have I seen cases go on smoothly to recovery which my operation-loving assistants were much inclined to give over to the surgeon. It will be seen from the foregoing that in my opinion the particular necessity of making an exploratory puncture and demonstrating the presence of pus can arise only in the smallest number of cases in which there may be the possibility of confusion between an abscess and some other tumor.

In all other cases in which an operation may become necessary serofibrinous or purulent exudate will undoubtedly be present. To find the particular site of the latter is the affair of the surgeon, who will be able to discover it, after making his incision with even greater certainty than before, and with perfect freedom from danger by means of an exploratory puncture.

For obvious reasons it is better, when the symptoms are not alarmingly urgent (foudroyant), to have the patient operated on in a public or private hospital, and for the purpose of transportation an ambu-

lance or stretcher and trained carriers will be necessary. Nevertheless I have frequently had patients operated upon in their own home without any great difficulties or detriment, for strict asepsis can be attained and maintained here as well.

From 448 cases taken from the literature of the subject Porter obtains the following data: Removal of the appendix during the attack in 151 cases, of which were cured, 122 = 80.3 per cent.; died, 29 = 19.7 per cent. Removal of the appendix between the attacks in 14 cases, of which were cured, 13 = 92.86 per cent.; died, 1 = 7.14 per cent. There were treated by incision and drainage 188 cases, of which were cured, 154 = 81.82 per cent.; died, 13 = 13.68 per cent. These figures may be compared with what we have said above (page 159) on the prognosis of appendicitis (perityphlitis).

In my opinion, the mortality figures are too high, in all the divisions of Porter's *résumé*, for the chances of the operation have, since the time these cases were collected, greatly improved, on account of better technique and of a more general knowledge of the indications for operation, especially on the part of the family practitioner, who now brings his patients to the surgeon earlier. As a proof we may cite the successful results which are reported in the latest communications of surgeons, such as Bull, Treves, Sonnenburg, and others.

I am of the firm opinion that, wherever the general practitioner and the surgeon go hand-in-hand, and the first is guided by the rules above quoted, the mortality of perityphlitis will not exceed ten per cent., *i.e.*, the deaths will be confined to those extreme and foudroyant cases which are from the beginning as good as lost, and can only in exceptional cases be saved by surgical intervention.

On the other hand, these severe complications which are caused by the boundless and unchecked advance of the inflammation, *i.e.*, of the abscess, and which we have already considered, should not, speaking generally, become developed at all, but should and can be avoided by a careful surveillance of the progress of the disease and timely surgical intervention. It is a particular satisfaction to me that the principles expressed above are also coming to be recognized by surgeons.

Professor Rotter, surgeon to the St. Hedwig Krankenhaus at Berlin, has published his experiences based on two hundred and thirteen cases, in a recent memoir.[49] His statistics are of especial value because (1) they include all cases brought into the hospital, whether treated in the medical or in the surgical wards; (2) in all cases of peritonitis which died during this time in the hospital the condition of the vermiform appendix, that is to say, its etiological relation to the peritonitis, was determined; and (3) the material is

quite uniform, and all very mild cases are excluded, since such are not brought to the hospital. Rotter divides the cases for the sake of simplicity into perityphlitis diffusa and perityphlitis circumscripta. The first includes all cases of general diffuse peritonitis. The latter embraces appendicitis simplex, appendicitis perforativa with an encysted exudate, and appendicitis retroperitonealis. The total mortality for the years 1893, 1894, 1895 amounted to 19 = 8.9 per cent. There were 21 cases, with 14 deaths = 60 per cent., or 6.5 per cent. of the total mortality, of diffuse perityphlitis. Of these 21 cases all except 3 were at once operated upon. Of the 14 cases which died, 6 had been operated on between the third and sixth day of the disease; all of them however died, and for the most part within the first twenty-four hours after the operation. The others were operated on between the sixth and twenty-fifth day of the disease. In most cases the disease had commenced mildly, and had assumed a malignant course later, on account of the improper behavior of the patient.

Of the 192 cases of circumscribed peritonitis, 156 (82 per cent.) made a spontaneous recovery, 33 were operated on, 3 died before operation—a mortality of 2.5 per cent. Twenty cases were operated on immediately, and these all recovered with the exception of one, a woman seventy years of age, who died from marasmus. Three cases of death, which ought not to be included, were 1 from a pneumonia and 2 from pelvic abscesses which had not been diagnosed. Of the remaining 169 cases, 13 were operated on, 8 suffering from circumscribed abscess and 4 from diffuse peritonitis—all recovering; 1 patient died who was suffering from suppurative peritonitis and who was seen too late for operative interference to be of any avail. Therefore only 17 per cent. of the cases of circumscribed perityphlitis were operated on. Although two-thirds of all these cases—or, more exactly, 153 cases in which positive facts were obtained from the history of the disease—were admitted in the first three days of the disease, the necessity of an operation only appeared, at the earliest, on the sixth day. If we take into consideration that among those who died there were a number whose condition obviously was made worse by improper treatment outside of the hospital, and the operation had to be done when it was too late, we may say, that with proper expert treatment the mortality would have been reduced to 5—at the very highest 7 per cent.

Altogether 84 per cent. of all cases which were brought to the hospital in the first six days of the disease recovered spontaneously, and the whole number of spontaneous recoveries amounted to 74 per cent. If we include, however, only the cases of circumscribed perityphlitis, we shall have as high as 90 per cent. of spontaneous recoveries.

These are as good results as can possibly be obtained. They surpass the statistics of Sahli, which showed a mortality of 9.5 per cent., and nearly equal those of the Moabit Hospital in Berlin, as regards the cases of diffuse peritonitis after appendicitis; as in the former 6.5 per cent., and in the latter 5.6 per cent. of all the cases recovered after the operation. Nevertheless the total mortality in the Moabit Hospital (149 cases in three and a half years) was 14 per cent. This may partly be due to the fact that the material was bad, that is to say, the patients were received in a more or less desperate and advanced stage of the disease. At all events there was no less operating in the Moabit (Professor Sonnenburg) than in St. Hedwig's Hospital, for in the former we find out of 149 cases, 94 operations performed—63 per cent.; in the latter out of 213 cases 54 operations—25.3 per cent. This difference in the number of operations is in a great measure due to the fact that, as already mentioned, the material in the Moabit Hospital was obviously on the whole of a worse kind. However, this can only be assumed for a limited number of cases, and for the balance the difference in the number of operations is to be explained by the fact that Sonnenburg, at Moabit, operated in many cases, in which Rotter, at St. Hedwig's, successfully pursued an expectant treatment. From this we may draw two conclusions, namely, (1) that it is unnecessary always to operate at the earliest possible moment, and (2) that in cases of periappendicular abscess spontaneous resolution occurs in an appreciable number of cases. For as Sonnenburg found pus in all the cases operated on by him, and as Rotter saw recovery follow in cases in which the disease ran an exactly similar course under conservative treatment, it cannot be doubted that a resorption of the pus, which must also have been present in Rotter's cases, took place. In what manner this is accomplished, whether by autodrainage into the cæcum through the perforated vermiform process, by direct perforation into the gut, or by resorption on part of the peritoneum, must remain problematical.

As to the indication for an operation in perityphlitis, Rotter holds the same opinion as that which I have already expressed.

As regards the question of recurrence, Rotter also gives some noteworthy and valuable data. Among his cases there had been a previous attack in only from twenty-one to twenty-four cases. These were generally seen during a second attack, rarely in a later one. The disease recurred most frequently within a year after the first attack, but few cases were seen in which recurrence took place during the second year, and later than that they were very rare. Besides this, autopsies in cases of recurrent perityphlitis have shown that these frequently recurrent cases are not cases of perforating appen-

dicitis. Under these circumstances it seems to me that these relapses lose most of the terror with which they have been clothed in recent times, and consequently it is not justifiable to operate solely for the purpose of avoiding a possible recurrence and to extirpate the vermiform appendix on this indication only.

NEOPLASMS.

Carcinoma.

Cancer is the most frequent of all neoplasms of the intestine, although it is less common than cancer of the stomach. It is usually primary, and but rarely invades the intestine by extension from a neighboring organ. Sex exerts no influence on the occurrence of intestinal cancer, male sufferers being only slightly more numerous than female, the proportion being about the same as that of men to women in general.

The *age* at which the greater number of cases occur is between forty and sixty-five years. However, cancer of the intestine has been repeatedly met with at a much earlier age, and even in children. I have myself seen a carcinoma of the duodenum which had developed from the cicatrix of an old ulcer in a girl sixteen years of age. According to Maydl, the cases which occur before thirty years of age amount to one-seventh of the whole number of intestinal carcinomas.

Location.

Carcinoma occurs by preference in certain parts in which, owing to the anatomical position of the intestinal coils, a longer stagnation of intestinal contents takes place, and so-called decubital or stercoral ulcers are easily developed. The analogy with cancerous formations in gastric ulcer, which has been pointed out by Nothnagel, is evident. From a compilation of the statistics relating to the seat of intestinal carcinoma, which I have made from those of Maydl, Bryant, Leichtenstern, Müller, and others, we find the following figures: The rectum was involved 874 times, the large intestine 148 times (of these 12 were in the colon transversum), the cæcum including the appendix 64 times, the ileum 26 times, the duodenum 19 times, the jejunum 17 times. From this we see that the large intestine is much more liable to carcinoma than is the small intestine, and that of the different portions of the latter the duodenum and jejunum are most frequently attacked.

Metastases of intestinal carcinoma most frequently extend to the lymphatic glands of the peritoneum, next to the liver, the perito-

neum, the lungs, and the uterus. According to Hauser, a difference is found according to the variety of the carcinomatous disease. Carcinoma gelatinosum only rarely causes a metastasis to internal organs, while scirrhus has a special tendency to the formation of metastases, which frequently greatly surpass the primary growth in size.

Pathological Anatomy.

All the various forms of carcinomata are found represented in the intestine. Most frequently we meet with cylindrical-celled cancer (adenocarcinoma and colloid cancer), less commonly with the pavement-celled carcinoma (epithelioma cancroid). Colloid cancers have a predilection for the rectum, the epitheliomata for the anus.

As regards its histology and development, cancer of the rectum differs in no way from carcinomatous tumors in general; we need only mention the fact that the tendency towards an ulcerative destruction and polypoid excrescences in intestinal carcinoma is particularly marked, because of the continuous irritation produced by the intestinal contents.

Carcinoma of the intestine, especially its scirrhous form, grows by preference in an annular manner, and thereby causes a narrowing of the intestinal lumen, which may progress to its complete closure. As a consequence an accumulation of the intestinal contents will occur above the stenosis, leading in the course of time to great dilatation, catarrhal inflammation, the formation of shallow ulcers, and hypertrophy of the muscular tissue. Occasionally, on horizontal section of the colon above the strictured portion, the gut has been found to be larger than the stomach of the same person. The constriction may, however, be in a measure relieved by suppurative destruction of the tumor, and a seeming improvement may thus temporarily occur. Below the constricted portion the intestinal coils will be found empty and contracted.

Not infrequently hemorrhage results from erosion of the vessels, and the blood then becomes mingled with the intestinal contents, or appears more or less unchanged in the dejections, according to the seat of the carcinoma and the amount of the hemorrhage. Usually one tumor only is present, very rarely more. The appearance differs according to the variety, *i.e.*, the histological character of the neoplasm; it may occur as scar-like, cartilaginous, annular indurations, as smooth nodes the size of the fist, polypoid formations, or cauliflower-like growths, disintegrated by suppuration, in the sigmoid flexure and rectum.

In the event of progressive growth we may find a hard, tubular

infiltration of the intestinal wall, which is then converted into a rigid tube the length of a finger or more.

Upon still further progress the mesenteric glands will become involved by way of the lymphatics, and will then be the seat of a tumefaction and cheesy degeneration. They may attain the size of a hazelnut, an apple, or a man's fist. Not infrequently there occurs an infection of the peritoneum, the latter being studded with cancer nodules. Ascites and hemorrhage into the abdominal cavity may also occur. In one case under my care I evacuated at three different times an effusion which contained so much blood that it could not be distinguished from venous blood, and at first I feared that I had opened a large vein. When the neoplasm has invaded the muscular and serous coats from the mucous and submucous layers, adhesions with neighboring organs, an extension of the carcinoma to them, and perforations are likely to result. In this manner gastrocolic, vesicorectal, rectovaginal, and even recto-uterine fistulæ may be formed, or the cancer may grow towards and through the abdominal wall and cause an external intestinal fistula. Cancer of the duodenum may involve the gall-ducts or the gall-bladder.

Symptoms.

The period of time at which the initial subjective or objective symptoms occur does not generally correspond to the beginning of the neoplasm, and the latter may have existed proportionately a long time without causing the slightest symptoms to awaken the suspicion of so grave a disease. Thus it may happen that the scene is opened with the symptoms of an acute obstruction of the bowel, or that a swelling is discovered quite by accident in the intestine of a patient who is being examined for general dyspeptic or other symptoms, and of the existence of which the patient did not have the slightest suspicion.

These abrupt symptoms of obstruction arise when the strictured part becomes suddenly blocked by hard scybala or fruit seeds, worms, gall-stones or intestinal calculi, or is occluded by the growth itself. As a general rule, the beginning is not quite so sudden, and the symptoms gradually develop in various ways, according to the seat of the neoplasm, its growth, and its character.

It may be said that the mechanical element which is created by the formation of the tumor, and the occlusion of the lumen of the intestine, is the real characteristic symptom. In the beginning there is nothing peculiar about the movements of the bowel; loose mucous passages alternate with periods of normal defecation or of constipa-

tion. Then suddenly symptoms of complete intestinal obstruction appear, and throw a new and startling light on the seemingly innocent "intestinal catarrh." But after a drastic cathartic, or maybe spontaneously, they may disappear, and the danger, especially if no tumor can be discovered (which is very often the case in carcinoma of the sigmoid flexure), seems past for the present. The disease, however, progresses, the symptoms of obstruction may return again, and again subside, and so on until at last the obstruction remains permanent. The general symptoms of stricture or occlusion of the intestine will be treated more in detail in another section.

These symptoms of stenosis are accompanied by the malnutrition resulting from disturbed digestion and the cancerous cachexia. The latter may, however, be postponed for a long time in cases of intestinal carcinoma, and particularly in those cases in which its seat is the sigmoid flexure, and a large percentage of patients do not succumb to the cancerous cachexia but to the intestinal obstruction. For this reason the duration of the disease under favorable circumstances may be comparatively long, and the general condition may be very little influenced.

Carcinoma of the *duodenum* reacts particularly on the stomach. Anorexia, nausea, vomiting, and pain (cardialgia) are the usual symptoms. In case the tumor narrows the lumen of the intestine, gastric dilatation with its characteristic symptoms is produced, and should the constriction be situated below the bile duct, the contents of the stomach will be found mixed with bile or with the contents of the small intestine. A differential diagnosis between carcinoma of the pylorus and that of the intestine may, under such circumstances, be altogether impossible, for in a carcinoma of the pylorus, when the walls are so rigid as to prevent closure of the ostium duodenale, the bile may be regurgitated into the stomach. Should the neoplasm be located at Vater's ampulla, icterus will result, which will be of varied intensity according to the increase or decrease of the tumor, that is, its growth or reduction by ulceration.

The tumors occurring in the upper portion of the *jejunum* behave in the same manner as those of the duodenum. Riegel has reported a case in which enormous dilatation of the duodenum and stomach resulted.

Carcinoma of the *cæcum* or of the *large intestine* is characterized by constitutional symptoms, the formation of a tumor, disturbances of digestion and of the intestinal functions, and symptoms of stricture.

Cancer of the *rectum* is characterized by tenesmus, spasm, and later by paralysis of the sphincters, the passage of blood and pus, and radiating pains towards the back, the pelvis, and the thighs.

All neoplasms may give rise to febrile symptoms through suppurative destruction and the absorption of pyogenic matter into the blood, or they may cause pyæmic and septic symptoms as a result of this absorption.

According to the size and site of the neoplasm, it may cause disturbances in neighboring organs by compression or dragging, radiating pains along the nerves, and disturbances of circulation, especially venous stasis. In this way œdema of the lower extremities may occur, and it is somewhat surprising that this œdema not infrequently disappears just before death, after having lasted weeks and months. Obviously the explanation is to be found in the gradual weakening of the circulation.

The Stools.—It is the nature of this disease that the condition of the stool should be a variable one. We find solid or liquid dejections with more or less admixture of undigested food, blood, pus, detritus, and mucus, and in deep-seated and disintegrating cancer particles of the tumor will be easily recognized. The diagnostic significance of the latter is so great that the dejections should be carefully and frequently examined in order to detect them if present. As a characteristic sign of the presence of intestinal stricture, the flat, ribbon-shaped, or nudel-like dejections may be mentioned. At the same time all textbooks say justly that no absolute reliance can be placed upon this appearance, since stools of the same shape may be passed in certain neuroses, or even in the absence of any intestinal affection, in spasmodic contractions of the intestinal coils, or in cases of starvation. I have quite frequently seen such ribbon-like dejections after the employment of nutrient enemata. At other times hard, round, blackish-brown or black masses which resemble sheep's dung, so shaped by their long sojourn in the large intestine, are voided. The laity usually describe such stools as looking "burned." There is also nothing characteristic in the odor of the fæces. In suppuration and putrefaction of the cancerous mass the fæces as well as the flatus may have a very fetid odor.

The Tumor.—In accordance with the mobility of the intestinal coils the neoplasms situated in them will also possess, as soon as they attain a certain growth, an active and a passive mobility; active, in so far as they will by their weight drag the intestine downwards; passive, in so far as they may be displaced by outside pressure. The latter is only possible in cases in which the site of the tumor is not in those parts in which the intestine is adherent, that is to say, the splenic flexure of the colon, the horizontal portion of the duodenum, and the rectum; the sigmoid flexure, the transverse colon, and the coils of the small intestines, on account

of the length of their mesentery, allow of a considerable displacement.

The tumor is, as a general rule, globularly egg-shaped or sausage-shaped, or, if of long duration, irregular. It is often tender on pressure, or else may be perfectly indolent. As a consequence of the mobility above referred to, and of the varying degree of distention of the intestine with gas or solid matter, it can be understood that the tumefaction may at different times be felt plainly or not at all, and may not only change its position, following inflation of the stomach or the intestines, but may at times disappear, or, on the other hand, become palpable only under these circumstances. Inflation of the intestines should therefore never be neglected in any doubtful case. In making palpation, it is frequently of advantage, especially in deep-seated tumors, to aid the pressure of the fingers of one hand by pressure on them with the other. The deep-seated tumors of the rectum may be directly exposed to view by the introduction of a speculum, with possibly the assistance of a small incandescent lamp passed into the rectum. Under all circumstances a digital examination should be made per rectum and in women per vaginam, or in the case this is not sufficient, the whole hand should be introduced according to Simon's method, the patient being deeply anæsthetized. An examination under ether is also necessary in those cases in which the abdominal muscles are so tense that a palpation of the deeper portion becomes impossible. In rare cases it may happen that a tumor is simulated by the local contraction of the transversus or obliquus abdominis muscle, which may mislead even an experienced examiner. Lately I have seen two such cases, one in a young man and the other in a young woman, in which both the attending physician and myself received the impression of a flat and even apparently movable tumor situated in the left hypochondrium. The youthful age of the patients and the absence of other symptoms, which would naturally be associated with the presence of such a tumor, influenced us to examine the patients under deep anæsthesia and to employ electrical transillumination of the stomach (Einhorn's gastrodiaphany). We found that the supposed tumor was produced by a tonic contraction of the muscles, and the seeming change of position on deep inspiration was obviously caused by the raising of the abdominal wall. Not to mistake a fæcal accumulation for an intestinal cancer or other neoplasm belongs to the A B C of diagnosis. A thorough evacuation of the intestine should therefore always precede an examination.

The tumefaction of the *peripheral lymphatic glands* does not seem to be particularly marked in intestinal cancer. It is well to remember

the discovery of Dietrich, that fully ninety-two per cent. of healthy persons have inguinal glands varying in size from a pea to a bean.

Further symptoms occurring in the course of the growth of the tumor are prolapse during defecation in cases of cancer of the rectum situated low down, and involvement of the bladder, the vagina, the peritoneum, or the stomach, in the course of which the various kinds of fistulæ may be formed.

Course.

The course of cancer of the intestine is a comparatively slow one. The disease may extend over a period of years if strictures are not formed early, and periods of perfect comfort may alternate with others of great distress.

I attended from time to time for two years a man suffering from cancer of the colon, situated high up, who enjoyed such good general health, and only occasionally complained of slight discomfort, that a consultant who was called in my absence took exception to my diagnosis. Very soon after, general decline and death occurred, and the autopsy confirmed my diagnosis.

The longest duration—three to four years—is generally found in cancer of the rectum. Usually the end comes quite rapidly and the patient succumbs either to an occlusion of the intestine or to debility, fever, and slight coma. The latter is designated coma carcinomatosum, and is thought to be the result of an autointoxication by the products of a decomposition of the intestinal contents or by the toxins of the carcinoma. I have succeeded in such a case in isolating a body, belonging to the group of diamines, from the urine.[41]

As a result of the disturbed circulation of the blood, caused by the cachexia and marasmus, œdema, especially œdema of the ankles, and thrombosis of the veins of the lower extremities occur. It is hardly necessary to mention that a successful operation, even though it be simply the formation of an artificial anus above the tumor, may be of decided influence in the prolongation of life. In this, more than in any other part of the organism, can be seen how much the growth of a tumor is retarded by excluding the source of irritation (in this case the intestinal contents).

Diagnosis.

The recognition of intestinal cancer is based on the presence of a tumor and the general cancerous cachexia. Strictures, or at least symptoms of occlusion, may result from other causes, and hence their

occurrence is not convincing, however valuable it may be as corroborative evidence.

The difficulty lies in the palpation of the tumor and in its differentiation from other tumors, that is, in defining the borders of tumors situated on other organs. A number of points which might be considered here have already been mentioned above (page 109).

The following conditions are to be thought of as possible and liable to give rise to error unless they can be definitely excluded: (1) Cancer of the pylorus; (2) cancer of the gall-bladder; (3) cancer of the pancreas; (4) cancer or ecchinococcus of the omentum; (5) retroperitoneal neoplasms arising from the wall of the pelvis; (6) tumors of the uterus and its adnexa; (7) gall-stones or fæcal concretions in the intestine; (8) perityphlitic exudations; (9) tumors of the kidney or (very rarely) of the spleen; (10) all those processes which lead to cicatricial stenosis of the intestine, such as tuberculosis, dysentery, syphilis, and typhoid or other intestinal ulcers. The dilated portion of the intestine above such cicatricial contraction may be taken for a tumor, and the condition may lead to a chronic cachexia, which, however, has no characteristic specific symptoms.

It is impossible in this place to discuss the differential diagnosis of all these affections from cancer of the stomach, for such a discussion would require a separate treatise. It will usually be possible to arrive at a positive diagnosis after a thorough examination, bearing always in mind the possible sources of error mentioned above, and taking account of the general symptoms of cancerous disease. In those cases in which no definite tumor can be made out, it will frequently be impossible to make a positive diagnosis of cancer of the intestine during life, and at best it can only be suspected, the diagnosis being made by exclusion. Even here, however, a true conception of the case will be reached as soon as marked symptoms of stenosis appear, especially if regard be had to the history of the case which will allow of the exclusion of tuberculosis, syphilis, dysentery, and typhoid fever.

The complications above referred to are easily recognized on account of their characteristic symptoms. Cancer of the rectum which has invaded the bladder, the vagina, or the uterus will present the symptoms of a false passage as soon as a fistula is established. A cancerous fistula between the stomach and the colon is characterized by the so-called lientery, *i.e.*, the rapid passage of large quantities of undigested food from the stomach to the large intestine. Upon inflating the intestine through the rectum the stomach is first filled directly, while the small intestine remains empty. Water put into the stomach, on the other hand, rapidly passes into the rectum.

In a case reported by Goodridge, however, in spite of the presence of a gastric fistula, there was never any lientery. It is possible that valvular duplications of the mucous membrane were here present.

Treatment.

Internal medication is even more hopeless in cancer of the intestine than it is in cancer of the stomach. The accompanying catarrh of the intestine may of course be influenced through proper measures (see page 131) or even temporarily cured. It will, however, obstinately recur, as its cause cannot be removed.

The treatment of deep-seated strictures with rectal bougies is outside of the domain of internal medication, but we must not neglect to draw attention to the fact that the too frequent and forced introduction of instruments often accomplishes just the contrary of what is expected. The irritation unavoidably connected with it stimulates the tumor to more rapid growth, and this, of course, leads to an increase of the stenosis. Not infrequently ulceration of the tumor is caused by it.

An *operation* constitutes the only successful method of treatment; it should, of course, be undertaken as early as possible, and the ideal operation would be the extirpation of the tumor with, if necessary, a resection of the diseased portion of intestine. Unfortunately, however, the conditions are favorable to this in only the smallest number of cases. Generally a tumor is recognized only when at the same time symptoms of obstruction have appeared, and by this time the tumor has usually reached an advanced state of growth and has passed beyond the limits of the intestine by metastasis. Even this, however, does not contraindicate an operation, provided the growth be accessible. The conditions are much more unfavorable when numerous adhesions with neighboring organs have already been formed or when no tumor can be discovered. In these cases there is nothing left but to make an artificial anus. What course to pursue will in most cases be decided on by the surgeon only at the time of operation, and upon this decision rests the prognosis as regards the duration of life. We would, however, again say that a marked improvement, and with it a prolongation of life and a perceptible improvement in strength, may follow a colotomy. A few years ago a man, fifty-six years of age, was treated by me in the hospital, in whom, on account of the presence of an undoubted cancer in the splenic flexure, a colotomy was performed. The man was so comfortable after the operation that he felt vigorous enough to get married in spite of the open fistula.

Sarcoma and Lymphosarcoma.

Intestinal growths of this kind are rare. Fleisher states that only fourteen cases have been reported in literature up to the present time, of which one occurred in a child four years old. The site of the sarcoma, and especially of a lymphosarcoma, is by preference in the small intestine; as a general rule, the course of tumors of this nature is more rapid than is that of carcinoma. They are mostly primary, rarely secondary affections, and take their origin from the mucosa (Balzer and Madelung), *i.e.*, from the lymphatic system of the intestine, the solitary and agminated glands. The tendency of these tumors to metastasis is remarkable. Much more remarkable, however, is the fact that there may be an extensive infiltration of the intestinal canal for quite a distance, and yet there will be no contraction but rather a sac-like dilatation of the diseased portion. Bessel-Hagen reports the case of a seven-year-old boy in whom, during the course of an extensive sarcomatous infiltration of a portion of the jejunum, there had formed an enormous aneurysm-like dilatation of the size of a large man's fist.

The constitutional involvement occurs much earlier and in a higher degree in sarcoma than in cancer. Anæmia and wasting appear early, sometimes even before the appearance of specific intestinal symptoms. When, however, a tumor has formed, we may be led by the absence of symptoms of stenosis and by the marked and rapid decline of the patient to suspect the presence of a sarcoma.

As regards prognosis and results of treatment, the outlook in sarcoma of the intestine is even more unfavorable than it is in carcinoma.

Benign Neoplasms.

Myxoma, adenomyxoma, lipoma, angioma, are among the forms of benign new growths which may be met with in the intestine. These tumors are designated *intestinal polypi* if they possess a pedicle. All of them are rare affections, which, if they give rise to clinical symptoms, can best be distinguished from the malignant growths by their long and relatively benign course. Peculiar and characteristic symptoms do not belong to them. In many cases they are only accidentally discovered at the autopsy.

The *adenomata* are among the benign tumors most usually met with. They arise from the mucosa (Lieberkühn's or Brunner's glands), have the typical acinous structure, are either attached to the mucous membrane by a broad base, like a button, or else they have a

pedicle. In the latter case they then form polypoid excrescences, which frequently cover long distances of the intestinal canal in extraordinary large numbers. I have in my possession a preparation in which the inner wall of the colon is covered, from the splenic curve to the sigmoid flexure, with such numerous polypi that they hang from the mucous membrane like tassels from a ribbon, and the whole specimen looks somewhat like a gigantic bunch of grapes.

The *fibromata, lipomata, and papillomata* most frequently arise from the connective tissue of the submucous coat, occasionally they also have their origin in the appendices epiploicæ. They are usually met with in the lower segment of the intestine; they vary from the size of a pea to that of an apple and larger.

The *myomata* and *fibromyomata* arise, as their name indicates, from the muscular layer of the intestinal wall, and in particular from the outer layer of longitudinal fibres. These tumors also either assume a nodular form or are supplied with a pedicle, or they consist of broad tumors lying in the wall of the intestine covered by the indurated submucosa and mucosa.

It may be seen from this description that histologically a great variety of tumors may take on the external appearance of polypi, so that fibrous polypi, myxomatous polypi, adenomatous polypi, and lastly myomatous polypi may be distinguished, or polypi may be found which are made up of a mixture of two or more forms in place of a pure type.

Clinically, the polypi are of greatest importance. In the first place they may cause symptoms of transitory or permanent occlusion of the intestine, or produce all kinds of obscure sensations after the fashion of a neurosis, which suddenly cease when the polypus is torn from its pedicle and expelled per rectum. The following case is cited by Treves:

A woman, aged 82 years, was troubled for years by indigestion, attacks of colic, and constipation alternating with diarrhœa. At last a movement from the bowels could be obtained only after an injection. One day after an intestinal examination had been made, a soft polypoid lipoma was passed, and after this all trouble disappeared entirely.

If the polypi, as frequently happens, have their seat in the rectum, they may be discovered by digital examination and moved backwards and forwards; occasionally they may also be extruded in defecation, and caught in the anus after the passage of the stool, or are torn off by the pressure of the fæces. If their seat be in the small intestine they may occasion invagination.

Other symptoms which may be caused by benign tumors are the

same as those caused by malignant tumors. Diarrhœal, mucoid, purulent, or sanguineous discharges, interference with defecation, vague symptoms of obstruction (especially if the seat of the tumor is in the ileocæcum or rectum), and hemorrhages from large vessels are met with here, as well as in malignant cases. Mercer[42] narrates the following case:

A woman, 34 years of age, apparently in perfect health, was attacked with melæna which resulted in death within a few hours. Autopsy revealed a rather large-sized tumor in the median line, 9 cm. in diameter, and weighing 455 gm., which was attached to the ileum by a short pedicle about fifteen inches above the cæcum. On cutting open the ileum, an oval opening was seen in its mucous membrane 3 or 4 mm. in diameter, which corresponded with the pedicle of the tumor at its peritoneal attachment, and directly communicated with an artery about 4 mm. in diameter. The tumor consisted of a highly vascular myoma. A number of myomata were also found in the uterus. It goes without saying that these tumors, if they have their seat in the duodenum and occlude the passage, may produce an enormous dilatation of it, as well as later of the stomach.

Foxwell [43] describes a fibromyxoma of the duodenum, and Reiche an adenoid carcinoma of the duodenum, which had caused an enormous dilatation of the stomach as well as of the duodenum.

HABITUAL CONSTIPATION.

By the term habitual constipation (atony of the bowel, obstipatio alvi, atonia intestinorum) we understand those conditions in which an evacuation of the bowel habitually takes place too infrequently and only with mechanical assistance. There is a very great number of persons who suffer from so-called indolent defecation, *i.e.*, they have a movement of the bowels less frequently than is compatible with a feeling of well-being or than they desire, and when a movement does occur the fæces are voided in hard masses and with efforts at straining. In these persons the normal impulse to stool which the passage of the fæcal masses from the upper portion of the rectum into the cloaca or ampulla of the rectum causes in the healthy once in twenty-four hours as a rule, does not occur at all, or if so, does not produce the proper result, *i.e.*, a spontaneous defecation is not obtained. In many cases the movement is difficult, but may take place without the employment of therapeutic agents; in the greater number of cases, however, it will be necessary to employ auxiliary measures for its consummation.

The number and periodicity of the stools are, however, in themselves no measure for determining the presence of constipation.

Retention of fæcal masses is possible, even when the movements seem to be normal. It is not very rare to find, on palpating the abdomen, that the intestines are filled with hard fæcal masses, and nevertheless to receive the assurance of the patient that a daily evacuation has taken place. A strong purgative will under these circumstances frequently produce the expulsion of enormous quantities of old inspissated fragments together with liquefied fæces. Others may have one stool daily, or even more, but do not obtain entire relief. Sometimes the latter feeling is only an imaginary nervous one, in others there is really a retention of fæcal matter present, due to indolent peristalsis.

Etiology.

The causes of obstinate constipation are manifold, a disproportion of the expelling powers to the work required of them is, however, always at the base of the trouble. It may be that this is found in a primary debility of the muscular fibres of the intestine, an intestinal atony, or it may be that intestinal muscular fibres have become relaxed only in the course of time as a result of the fruitless struggle against a chronic obstruction.

There is no doubt of the existence of habitual constipation, which is to be looked upon as an independent affection, in which either a decreased excitability of the intestinal nerves or a defective development, generally congenital, of the intestinal muscular fibre exists. Nothnagel found in a careful measurement of the muscular structure of the intestine, that is of the thickness of the intestinal wall, that there are cases in which, notwithstanding a robust muscular development of the body, the muscular structure of the large intestine was only 0.12–0.25 mm. in thickness instead of the normal average measure of 0.5–1 mm. It cannot, however, be denied that in some persons an hereditary cause may also have to be considered. These patients enter life handicapped by their inheritance, and usually suffer from early youth from difficult defecation.

In others constipation is in a certain sense inculcated, the parents and teachers not having educated the children to attend to a regular movement of the bowels, and this is especially the case in girls, who are likely to neglect this function. These occasional irregularities are liable to lead to fæcal accumulations, and thus to burden and dilate the intestinal canal, eventually bringing on a motor debility of the muscular structure, and chronic constipation.

In very many patients constipation is the result of an irrational diet. A rich diet difficult to digest, containing little water and leaving behind a large residue, or a diet which is devoid of variety, as, for

example, a pure meat diet, or finally one which is very slightly stimulating to the intestines, consisting of easily absorbable articles which leave but little residue, may lead to constipation. The intestines here have no adequate stimulation, and their motor apparatus acquires a certain torpor. Constipation may frequently be the result of profuse perspiration, for example, in soldiers on a forced march, or of anything which causes great loss of water from the skin, lungs, or kidneys, when at the same time an insufficient quantity of fluid is taken into the system. The same may be said of the abuse of intestinal peristalsis by means of purgatives, which may be followed by a condition of temporary constipation, through a reaction of the fatigued muscular fibres.

Constipation may also be caused by suppressing defecation, or attending to it in an irregular manner and at irregular periods, or it may be the result of departure from the habitual mode of life, especially when this involves little or no bodily exercise, as, for example, on long railway journeys or sea voyages. This constipation, which is at first only temporary, not infrequently becomes chronic if neglected.

The influence of a sedentary occupation and absence of bodily exercise, however, is greatly overestimated, particularly by the laity. Although it cannot be doubted that those persons whose vocation or inclination is accompanied by a great deal of daily bodily exercise, are on the whole more rarely subject to habitual constipation than those who follow a sedentary occupation, still the exceptions are very numerous. Thus we not so very seldom observe obstinate chronic constipation among officers, professional equestrians, cyclists, farmers, and persons who as a hygienic measure regularly take long daily walks and active bodily exercise, while temporary constipation may occur after any violent exercise, such as long marches, athletic contests, etc., which is accompanied by copious perspiration. It is true, however, that a sedentary occupation is particularly liable to produce a venous stasis in the abdominal organs, and thus indirectly occasion a disturbance of intestinal peristalsis, and that this stasis is overcome or prevented from occurring by suitable muscular exercise of an active or passive nature.

A cause of this venous hyperæmia is also found in all those conditions which either directly or indirectly produce circulatory disturbances. To these belong the diseases of the heart and lungs, of the large glands of the abdomen, particularly the liver, and of the other abdominal organs, especially those located in the true pelvis, such as, in women, the uterus and its adnexa, which may by pressure, flexions, and chronic inflammations give rise to engorgement and circulatory obstructions of a local kind. In this connection mechani-

cal pressure should be mentioned, although it is referred to at greater length in another section. Thus changes in the position of the uterus, or of the ovaries, new growths, pelvic exudates, hypertrophy of the prostate gland, etc., may lead to a chronic obstruction of the bowels. A very important and frequent cause during its course, as well as more particularly in its consequences, is pregnancy. Chronic constipation is one of the most frequent complaints of the pregnant woman. In this case a purely mechanical cause, namely, pressure on the intestines, is in the first instance the cause of the constipation. When, however, as is the case with many women, pregnancy is the beginning of an inertness of the bowel which remains through the whole future life, and is the cause of a continued combat with constipation, there are usually two reasons to be found for it: (1) The intestines have passed, during pregnancy, into a condition of relaxation and dilatation, which has become irreparable; and (2) the abdominal parietes having lost their natural tone, become relaxed, and thus also contribute to the affection, not in the sense that the abdominal pressure, leaving aside the act of defecation proper, assists the movement of the intestines, but because peristalsis finds a certain support in the wall of the abdomen, along which the intestine moves, as the screw in its nut. On the other hand, the fæcal masses will remain in the rectum, or at least will be evacuated with much more difficulty if the abdominal muscles exert no pressure at all or only an insufficient amount.

The position of the intestines, particularly the frequently irregular and abnormal one of the large intestine, seems to exert only a very small influence on the regularity of the stools. The colon, as is well known, is sometimes found lying in an oblique position, from right to left and from below upwards, extending even into the concavity of the diaphragm and rising up behind the stomach; sometimes the transverse colon is found in a U- or M-shaped coil coming down as far as the symphysis; or the small intestines are found lying altogether in the true pelvis. But these anomalies may be found post mortem in persons in whom they occasioned no special intestinal torpidity during life. It should also be considered that such displacements may be the result and not the cause of fæcal accumulations, which may by their weight draw the intestine downwards. The varying length of the mesentery also stands in no relation whatever to the movements of the intestines. Whether complete prolapse of the intestines, the enteroptosis of Glénard, can directly cause constipation, or whether this affection arises in consequence of other conditions associated with the enteroptosis, is another question which yet remains undecided. All that we can say at present

is that enteroptosis and constipation are frequently met with together.

Adhesions of the intestines among themselves, or to other organs of the abdominal cavity, or to the wall of the pelvis as a result of old inflammatory processes, as is easily seen, may occasionally be the source of chronic constipation, because peristalsis is interfered with by these adhesions in a way which we are hardly ever able to remedy. Nevertheless I must agree perfectly with Nothnagel that these peritoneal adhesions are exceedingly rare in comparison with the great frequency with which constipation is met with, and that they have been overestimated as a causal element of the latter. A previous general peritonitis may also give rise to constipation by the participation of the muscular layer of the intestine in the inflammatory process of the serosa, thus causing permanent injury in the shape of an interstitial induration process. As regards the rôle played by nervous affections, such as hysteria, neurasthenia, nervous debility, and the like, in the production of habitual constipation, we shall refer to it in detail in the section on the nervous diseases of the intestine.

Symptoms.

There is no other diseased condition which has become to such an extent the playground of the layman's imagination and of the arts of the "natural healer" and the quack than habitual constipation. The "Proktophantast" of Goethe, who is only happy "when leeches suck delight from out his buttocks," the "Staatshaemorrhoidarius" of the funny papers (*i.e.*, the German official who is suffering from constipation and piles), and Molière's "malade imaginaire" are well-known types of this kind. The patients complain of local and general symptoms. The first are naturally referred to the existing constipation, whose presence the patient observes with the greatest anxiety, and the presence or absence of which frequently takes up his whole thoughts. Great is his sorrow when the expected evacuation does not occur, and even greater his joy when it makes its appearance on time and in adequate quantity. Many patients do not complain on the whole of any local symptoms beyond a certain feeling of heaviness and discomfort in the abdomen; in others the abdomen is distended, particularly in the region of the descending colon and of the sigmoid flexure, and when the abdominal walls are thin the individual intestinal coils may be felt or seen as puffy masses. Pressure on the abdomen is not particularly painful, although sometimes it may cause very severe pain. There may be cramps and colic, and sometimes also such general symptoms as

sensations of oppression, a feeling of apprehension, and vertigo, to which we shall refer again. These sensations generally pass away after an evacuation or the expulsion of flatus. Occasionally the symptoms may become alarming and severe collapse and vomiting may excite the suspicion of an intestinal obstruction. This may occur especially after a long coprostasis in the insane, in persons who avoid a passage as long as possible on account of painful fissures of the anus or hemorrhoids, or in the old and debilitated. A copious evacuation either by internal medication or by irrigation or manual procedure will remove these conditions. In old people, however, relief is not always so readily effected, and I have seen two persons die, in whom, without the occurrence of real symptoms of ileus, a retention of fæces existed for six to eight days, and death resulted from collapse. Both cases were in men about seventy years of age.

In constipation of long duration we meet with the so-called fæcal tumors, whose seat may be in various portions of the intestines, but which are usually found in the large intestine; they may, however, also appear in the cæcum. Absolute constipation, however, is not always necessary for their formation, for they may also occur when the evacuations are not copious enough, so that more or less large quantities are retained in the intestine, the so-called accumulative constipation (Field). These fæcal tumors, as already mentioned, may lead to secondary deposits and to changes in the size of the intestines, especially of the large intestine, inasmuch as they frequently attain an enormous size, and may weigh fifteen pounds or more.

These tumors may bring about changes in the intestines in various ways. We will not consider the total obstruction of the intestinal lumen and its sequelæ, as this will be minutely described among the causes of ileus. The production of inflammatory processes of the mucous membrane and of pressure ulcers (stercoral ulcers) with their consequences have already been mentioned. To this may, however, be added that with the accumulation of such great masses of fæcal matter further stretching of the intestinal wall must of necessity be combined, which can only be obtained at the cost of a weakened muscular tissue, *i.e.*, of its atony. The latter again favors the accumulation of the fæces, and in this way an endless chain is formed, which can only be broken by vigorous interference. Another sequel is that a diastasis of the muscle fascicles takes place, in consequence of which the mucous membrane is protruded externally, like a diverticulum or hernia. In these pocket-like protrusions the fæcal matter is caught, dries up and becomes hard, or decomposes. In any case inflammatory reaction occurs in the wall of the intestine, as already described, and

this may extend to the peritoneum, giving rise to a circumscribed peritonitis.

These cases, a few of which are mentioned in the literature, are, however, without doubt very rare. I have met with such a case only once, although I have occasionally seen small protrusions of the mucous membrane between the separated muscle fascicles. The colon alone, and especially the descending colon in its lower portion, is the seat of these diverticuli.

It is obvious that the diagnosis of these fæcal tumors is not always easy, and can frequently be based only on the successful or unsuccessful action of a proper purgative, and the general history of the disease. The reader is therefore referred to the diagnostic measures which have been mentioned in the introductory section.

In spite of all the care used in an examination, mistakes have frequently occurred, which, according to the nature of the wrongly reached supposition, *i.e.*, accordingly as a true neoplasm or a fæcal tumor has been falsely diagnosed, have led the physician to a pleasant or an unpleasant disappointment.

Finally a local sequence of habitual constipation is frequently the appearance of hemorrhoidal tumors; these will be discussed in a separate section.

The *general symptoms* are extraordinarily diverse in their extent and gravity, and may vary all the way from a general feeling of slight discomfort to the deepest mental disturbance and most profound melancholy. There are some persons who pay no attention whatever to existing constipation. They employ purgatives or clysmata year in year out, and when the desired result is no longer obtained by the accustomed dose, another remedy or a larger dose is employed until satisfaction is given. In these cases usually some fulness and heaviness of the head, a certain indolence and dulness, the necessity for a stool, and the feeling that everything will again be well as soon as this has been accomplished are experienced. In others, however, the symptoms increase to loss of appetite, headache, vertigo, perhaps to insomnia, flashes of heat are complained of which are mistakenly called fever, though no rise of temperature is present, and which are of a purely nervous character. Again, other patients may suffer in their whole organism, especially in their mental capacity. They are not able to have a clear thought, cannot work enduringly, are liable to hypochondriacal illusions and whims, and refer their whole mental and bodily well-being back to the process of defecation. The determined effort to have a movement of the bowels and the study of the condition and copiousness of the stools become the great events of the day, and quite frequently the comfort

not only of the individual himself, but that of all about him is dependent upon the nature of his defecation. There are persons who have reduced this whole matter to an absolute system, and thereby bring themselves and others to the verge of despair. This may go to such a length that I have repeatedly met with patients who assured me that at times, when nothing would succeed in overcoming the constipation and bring on a movement, they thought seriously of suicide, but fortunately the saving evacuation would always at last be obtained. Such statements may possibly be somewhat overdrawn, but they prove how deeply constipation may affect the general condition of the patient, and every experienced physician may recall examples, especially in persons with time and leisure at their disposal, whose whole object in life is the aim after a normal passage. Among the symptoms of irritation we may mention a sensation of more or less marked discomfort in the abdomen, burning sensations in the stomach, tenderness from pressure of the clothing, a feeling of fulness, and other uncomfortable sensations; furthermore, palpitation of the heart with a sensation of anxiety, pain on pressure in the region of the heart, and occasionally rapid and irregular pulsations. Gussenbauer reports the occurrence of trigeminal neuralgia, and other authors speak of neuralgic pains in the region of the sciatic nerve, of the lumbar, or of the ovarian plexus. In this connection it is possible to go too far in deciding between constipation and other affections rightly or wrongly looked upon as nervous, and when Federn attempts to connect Graves' disease, or cardiac asthma with constipation, he undoubtedly goes too far. It seems on the whole uncertain as to how far we have to deal with constipation in the so-called sequelæ when they encroach upon the nervous system, whether indeed there is a relation of cause and effect, whether there is simply a coincidence, or whether the constipation may not depend upon the nervous affection. Romberg positively declares that abdominal disturbances cannot occasion hypochondriasis, and Dunin, on the other hand, regards hypochondriasis and constipation as the results of a third affection, namely, neurasthenia. As regards chlorosis, there is no doubt that the poverty of the blood may be the cause of the usually existing sluggishness of the bowels as well as of the numerous nervous conditions.

It has been proposed to connect these nervous symptoms with the absorption of products of decomposition in the intestine by the blood. Von Pfungen has shown that in simple chronic constipation an increased decomposition of the contents of the intestine takes place, and the theory of a chronic intoxication has been based on the conjectured formation of ptomains and sulphuretted hydrogen in the intestine,

which act on the nervous centres. The proportion of sulphuretted hydrogen in the gases of the intestine is, however, exceedingly small. It amounts to less than 0.1 per cent. (Nowak and Brautigam). I have frequently made the attempt myself to prove the presence of sulphuretted hydrogen in the flatus by inflating the intestines with air, and passing the subsequently expelled air, which must have been saturated with the intestinal gases, through lead water. These experiments usually resulted negatively and only quite exceptionally, a very slight reaction was produced.

Rosenheim calls attention to the fact that the formation of ptomains, according to the experiments of Bouchard, is confined to the retained fluid fæces, while the hardened fæcal masses, with which we are concerned in constipation, do not offer a favorable soil to abnormal processes of decomposition. Nevertheless the possibility of a so-called autotoxæmia is not to be dismissed lightly, although at the present time we are not in possession of any facts affording a positive proof of its occurrence.

Diagnosis.

The diagnosis is as easy in most cases, when the patient himself makes it for us, as it may be difficult under certain circumstances. In all cases we have to determine how far habitual constipation may be the result of some other affection; whether existing tumors in the intestines are fæcal tumors or not; whether a discharge of mucus or blood is caused by the constipation, or is, together with the constipation, a sign of some other affection; all these can under certain circumstances be determined only by the most minute examination and by taking into account all the circumstances which have been discussed above.

Prognosis.

The prognosis of sluggish bowels and constipation is favorable *quoad vitam*, but *quoad valitudinem completam* dubious. Grave accidents are the exception to the rule, at the same time we all know that hardly any other affection of a similar not dangerous nature gives the patients so much annoyance, and so completely dominates their whole physical and mental existence as this. The result is often a disturbed mental condition, well known as hypochondriasis. It is true that the idea has been suggested (Virchow, Nothnagel, and others) that hypochondriasis was not the result, but the cause of constipation, or that both have sprung from the broad soil of neurasthenia. This is certainly not true in general, and can be said of only a small number of cases. Nothing is easier than to demonstrate how

the symptoms of a hypochondriacal mental condition will increase more and more as chronic constipation continues, and that too in individuals who have not shown the slightest sign of neurasthenia or hypochondria previously. It is just as easy to comprehend that not all persons suffering from constipation become hypochondriacs, as it is that not all drinkers of whiskey acquire delirium tremens or a gin drinker's liver.

As a great many persons, however, not only look upon the continued sluggishness of the bowel as a source of grave apprehension, but also fear that some great harm may accrue to them through the continued use of drugs, we are justified in pointing out to these individuals, that the intestine may possess a certain natural weakness which needs for its adjustment a continuous assistance, exactly in the same manner as a short-sighted eye needs the complement of glasses. It will, however, only be necessary exactly to compensate the defect and not to exceed its necessity.

Treatment.

At the beginning of every work on the treatment of constipation should be written "as few purgatives as possible." This does not mean that we should without discrimination endeavor to administer no purgatives at all. This of course is the ideal point of view; but there are a number, and a very great number, of patients who are utterly unable to do without laxatives, for the reason that the intestinal canal in them is congenitally weak. These persons demand a corrective in the same way that a myopic needs glasses; and the physician's art lies in the selection of such purgatives as are adapted to the individual necessities of the patient, and compensate for the physiological defect. The one selected should be as unirritating as possible, so that it may be used in the same dose for a long time, sometimes for years without losing its effect.

As a rule every possible therapeutic measure will have to be employed, dietetic, physiological, mechanical, and medicinal. Very rarely will a single one of these measures be sufficient, but recourse will have to be had to a number. We have here to consider all those measures which have already been discussed in the introductory section.

Diet.—Those food products and drinks are to be preferred which have already been mentioned in the section on general therapeutics (page 98) as acting on peristalsis. A diet of meat alone or overloading the intestine with vegetables, especially with those that contain much cellulose or a small percentage of water, is to be avoided,

and the amount of food is to be reduced to a suitable quantity. The administration of abundant fluid, particularly of water, on the empty stomach is advantageous; but it must be remembered that some beverages, like tea, red wine, and under certain conditions also milk, have a constipating effect. If the obstruction is due to the use of food which is too bland and monotonous, a change in the diet with the ingestion of somewhat coarser food is to be counselled.

Coarse bread, much butter, honey, etc., perhaps also different varieties of cabbage (a tablespoonful of raw pickled cabbage is a favorite and frequently effective domestic remedy in Germany), beverages containing carbonic acid gas, particularly sweet effervescing wines, promote in some persons a prompt stool. In this respect, however, we may meet with the greatest individual variation, and what is effective in the case of one person may hurt or at least prove ineffectual in that of another. There are some people who during the summer, when they can eat plenty of raw fruit, have no difficulty with their bowels, but who suffer from constipation during the winter. I am acquainted with people who have a regular, spontaneous movement of the bowels only during the strawberry season, while others are able to eat strawberries and raw fruit in large quantities without any effect at all. In short, each organism in this respect is a law unto itself and must be studied in reference to its special qualities and peculiarities.

Hygienic treatment is especially of assistance and to be relied on as of main importance in those cases in which the cause of the constipation is found to be a general neurasthenia, chlorosis, sedentary occupation, and the like. Under hygienic treatment we must include hydrotherapy, cold bathing, douches to the abdomen, the wet-pack, cool sitz baths, etc. In my experience the so-called Scotch douches, in which the temperature of the water is changed in rapid succession from warm to cold, have given exceptionally good results. The contraction of the abdominal muscles and of the intestinal muscular coat caused thereby is usually a very active one.

The *physico-chemical methods of treatment* consist in massage, electricity, clysmata, and irrigations. There is not the slightest doubt that scientific massage will give excellent results in many cases, especially in lean persons in whom the intestines may be manipulated through the abdominal walls. At the same time there is also no doubt that in many persons, particularly in the obese, massage will give no good result whatever, or at the utmost only a temporary one. The same is true of electricity, whether both electrodes be applied to the abdominal wall or one pole be passed into the intestines. In the first method we may use either a board plate electrode, the

common button electrode, or the electric roller. As regards the construction of the electrode for the rectum the reader is referred to page 105.

It is impossible to decide in the beginning which of the two forms of electricity, the galvanic or faradic current, will act most favorably. *A priori* one would expect the best results from the energetic contractions produced by the faradic current.

Under the title of "Postural Treatment of Constipation," E. T. Williams" drew the attention of the profession a few years ago to the fact that the customary attitude in defecation is not adapted to permit the two main factors instrumental in the expulsion of the rectal contents, namely, the contraction of the abdominal muscles and that of the diaphragm, to exert their full power. Williams therefore suggests that this act should be accomplished in a squatting position. I am able to indorse this suggestion fully. I am acquainted with a number of patients, especially women, in whom, since they have by my advice adopted this posture, the former sluggishness of the bowels has been replaced by a normal and easy action.

Two very important factors in all cases of constipation are, first, regularity, and secondly, perseverence. To visit the water closet daily at the same hour, to remain there without however straining forcibly, although a slight compression of the abdominal muscles is often necessary, and to persevere until success is attained, seemingly appears a small matter, but it is a very important one. Although success may not be attained at once, it is frequently the case that this simple method will eventually work a change. This is of course true only of the light cases of organic sluggishness. In severe constipation it may happen that the hardened scybala must be removed from the anus by the fingers before any result can be obtained from remedies, and I have met with persons who were always compelled to clear out the rectum manually before each enema.

On the manner of giving enemata and irrigations, we have already spoken on page 102. Although this treatment is seemingly the mildest and the most likely to agree with the organism, yet it has the drawback that the time soon arrives in which small quantities of water —hot, cold, or tepid, as the case may be—will not act, and larger and still larger quantities up to one, two, or more quarts will have to be used to produce a stool. Through this the large intestine will become more and more dilated and relaxed, and a condition is gradually established in which even these irrigations are useless. Whenever it is possible the irrigations should not be used daily, but at the longest possible intervals, say about every second day or less frequently, and the temperature of the water should gradually be lowered

from tepid (16°–20° R. or 68°–77° F.) to cold (10°–12° R. or 54.5°–59° F.). We have to consider here not only the direct effect of the cold in stimulating intestinal peristalsis, and in reducing congestion in the large intestine, but also, according to the experiments of Röhrig,* increasing the biliary secretion and stimulating the circulation in the portal system, which again indirectly affects the intestinal circulation.

All of these measures should be assisted by sufficient bodily exercise, if possible directly acting on the abdominal muscles, such as walking, athletic exercises, rowing, lawn tennis, horseback riding, etc. Excellent results have been obtained in habitual constipation from rowing in rowboats with sliding seats in which an efficient and long-continued massage of the abdomen takes place. In many persons, however, these physical or mechanical measures are either from the beginning without any effect or they lose their effect in a short time. One would think for example that an army officer had enough bodily exercise in every way, and still in this class we often find severe constipation and hypochondriacal dispositions which are generally very intractable.

Medication.—All purgatives should be taken as infrequently and in as small doses as possible. The patient's endeavor should constantly be directed to a restrict their employment.

Individual peculiarities of the patient as regards the dose and nature of every active purgative must be taken into consideration, and each case demands especial study. That only is a good purgative which will produce without discomfort (colic, tenesmus, nausea) a soft, not a watery, abundant evacuation. In every case we shall have to ring the changes on the various aperients until the one adapted to the individual case is found.

All purgatives are contraindicated in those cases in which bad results may be expected from their effect on peristalsis, as for example in acute inflammatory conditions of the peritoneum as well as in cases of threatened perforation (ulcer, appendicitis), or when tonic spasm of the intestine is the cause of the constipation, as in lead-poisoning, meningitis, and certain spinal diseases. Ages ago evacuants were divided into mild evacuants (laxatives or aperients) and active evacuants (purgatives), and in this manner our ignorance has been concealed as to the real manner of their action. It is more rational to classify them according as they act more strongly on the small intestine or on the large intestine, and furthermore to make a distinction between those which excite peristalsis and those which promote liquefaction of the intestinal contents.

A minute analysis of the action of individual laxatives belongs to

the text-books on pharmacology, and here we shall consider only a few of these drugs as regards their practical application in the treatment of chronic constipation.

Rhubarb in substance (powder, or pills, or pieces of the root itself) is one of the very best aperients we have providing a good preparation is used, for not all the samples of rhubarb in the market are of equal quality. It possesses, however, more than any other drug, the disadvantage that its primary effect is followed as a rule by a period of more or less severe constipation, thus necessitating the patient's constant return to the remedy, so that, as the saying is, he must sell his soul to rhubarb. This does not seem to be a very serious matter, in case the dose need not be increased. I know some persons who have been taking regularly every day for twenty to thirty years one pill containing 0.1 gm. (gr. iss.) of rhubarb, and are very comfortable with it; as a rule, however, the dose will have to be increased after a short time in order to maintain the effect of the drug, and soon it becomes altogether inert. The same may be said of the compounds containing rhubarb or senna, such for example as the compound licorice powder (senna leaves and licorice root, of each 2 gm.—gr. xxx.; fennel seed and washed sulphur, of each 1 gm.—gr. xv.; and sugar, 6 gm.—ʒ iss.), and the powder of magnesia with rhubarb (magnesium carbonate, 60 gm.—℥ ij.; elæosaccharum of fennel, 40 gm.—ʒ x.; powdered rhubarb, 15 gm.—℥ ss.), and others. Of preparations of this kind, the compound licorice powder, on account of the presence of fennel seed, is to be recommended in cases of great flatulence. When there is much flatulence we may also add from one to three drops of oil of fennel or of caraway, directly to the powdered rhubarb.

In order to obviate this secondary constipating effect, it is well to add to the rhubarb at once some neutral salt. For this purpose the following combination, which was used even in the last century under the name of solamen miseris and which has been recently warmly recommended again by Leube, is one of the best. This is a mixture of powdered rhubarb 30.0 (℥ i.), sodium sulphate 15.0 (℥ ss.), and sodium bicarbonate 7.5 (ʒ ij.). The patient may take of this, according to his necessity, from as much as will lie on the point of a knife to a teaspoonful or more, mixed with a large glass of warm water, at night before going to bed. On the following morning an abundant painless evacuation will be had. Infusion of rhubarb is not to be recommended for long-continued use, but may be used in occasional constipation, particularly in combination with syrup of senna, syrup of manna, syrup of rhamnus cathartica, or the like. The same may be said of the tincture or the extract of rhubarb. The compound

extract (extract of rhubarb, 30; extract of aloes, 10; resin of jalap, 5; and soap 20) belongs to the more complicated and drastic cathartics, on account of its contents of aloes and jalap.

Of equal value at the very least with rhubarb, and even to be preferred on account of its prompt and efficient action, particularly in pediatric practice, stands calomel. It may be given without fear of any unpleasant after-effect in doses of 0.01 to 0.1 and 0.2 (gr. $\frac{1}{6}$–$1\frac{1}{2}$–3), according to the age of the child. In the adult mercury has, even in the form of the well-known blue pill, the great drawback that it may produce an unexpected mercurial intoxication, and that individuals present such extraordinary idiosyncrasies in regard to it that we can never know at the outset what the result will be. I have known a quite severe stomatitis appear after the administration of only two doses of 0.4 gm. (gr. vi.) each of calomel, and I have never been able to understand why so little is heard from England and America of the bad after-effects of these preparations, in spite of their extensive use in disturbances of digestion and constipation.

Closely related to rhubarb are the numerous vegetable cathartics, such as the different varieties of rhamnus, to which belong cascara sagrada (rhamnus purshiana), so much used in recent times; also senna, manna, tamarinds, podophyllum, jalap, etc. Of these senna is particularly liable to produce violent peristalsis attended with pain, caused partly by a bitter principle contained in the leaves, which may be extracted by alcohol. For this reason it is recommended that the infusion be made with senna leaves extracted in alcohol. This should be taken cold.

Castor oil also should be given a place by itself, in so far as it is better adapted for occasional use once or twice than for continued administration. It is best given in the form of emulsion (30 to 180 with three drops of oil of peppermint added) in doses of one tablespoonful every two to three hours. This is well borne for some time without disturbing the stomach, as might be readily supposed. In all cases of constipation due to spasm of the muscular layer of the intestine, for example in lead colic and in certain forms of hysterical constipation, castor oil, combined with small doses of opium, is frequently of surprising benefit. The continued use of castor oil, however, whether it be administered in capsules, in emulsion, or in any other of the various forms, is usually prevented by the appearance of dyspeptic symptoms, and particularly of insipid or oily eructations.

To the more strongly acting drastics belong jalap, scammony, colocynth, euonymin, and finally croton oil. The greater number of these remedies, and especially aloes, act chiefly on the large intes-

tine, and as defective peristalsis of the large intestine is usually present in most cases of constipation we may be sure to obtain from them first of all a prompt and copious action. Their disadvantages, however, are that they exert strong irritation, and in this way not only lead to rapid relaxation and exhaustion of the irritated muscular fibre, but also cause a congestion of this part of the intestine, which may lead to a chronic catarrhal condition. Aloes is the chief ingredient in most of the innumerable purgative pills and mixtures which are advertised so extensively. It is hardly necessary to mention that the strong drastic cathartics, like colocynth, scammony, gamboge, and croton oil should be used only in the most obstinate cases of constipation, and should be avoided in all cases in which increased peristalsis may do great harm, as in internal strangulation, intussusception, or acute intestinal inflammation. I have, however, in a few cases of intestinal obstruction of a high degree by fæcal tumors, directly threatening life, made bold use of croton oil (gtt. ii.–v. in an emulsion of castor oil 50:200, to be taken in three doses), and have seen life saved thereby. The diagnosis in such cases should of course be absolutely positive.

The neutral salts, sodium sulphate, magnesium sulphate, potassium and ammonium tartrate, potassium sulphate, Carlsbad salts, tartarus boraxatus, etc., act mainly it is said through their high endosmotic equivalent, by provoking an increased transudation of water to the intestine, and in this manner effect the liquefaction of the intestinal contents; another factor, however, apparently comes into play here, for we find that Carlsbad salt in a hot solution will have a different effect from the same salt in cold solution. All these salts, as well as the various bitter waters (Friedrichshall, Püllna, Ofener, Marienbad), act, so to speak, more in a chemical or physical manner, and do not affect the muscular apparatus and the nervous system as much as the vegetable cathartics. Their action, if taken in the form of mineral water in greatly diluted solution, *i.e.*, in large quantities of water, is alterative to the general constitution, and this circumstance, taken in conjunction with the changed way of living, with the sole object of strengthening intestinal digestion and the whole general condition, may possibly determine their success when a course is taken at a spa. It cannot at least be explained in any other way why some persons are relieved of chronic constipation for years, or even for their whole life, who had previously used the most varied vegetable and saline cathartics for their affection without any result.

We must not forget to mention sulphur, which has oftentimes an excellent effect as pure sulphur, but particularly so in form of the sulphur waters of Neundorf, Weilbach, Baden, Zurich, Aldenau, Aix

les Bains, and others. I have often made use of sulphur in combination with other cathartics in the following formula with good result:

℞ Washed sulphur and sodium tartrate, . of each, 10.0 (gr. cl.)
Senna leaves, ... 5.0 (gr. lxxv.)
Cardamom seed, ... 2.5 (gr. xxxviij.)
Syrup of rhamnus cathartica, ... q. s.
Make an electuary. S. A teaspoonful morning and evening.

The water clysmata have already been discussed above. In many cases, however, enemata of oils are of particular value, a measure long favorably known in Russia, and which has rightly been particularly recommended by Kussmaul and Fleiner in Germany. Large quantities, 300–500 gm. of pure oil (linseed or sesame) may be used, with the double result of stimulating peristalsis, and at the same time lubricating the lower passage. The frequently asserted softening effect of the oil on the scybala is as good as absent; hard scybala may be immersed for hours in warm oil without any softening effect being evident. In some cases much smaller quanitities (60–80 gm.) will be sufficient. A rapid stimulation of the peristaltic action of the lower portion of the intestine is produced by glycerin through its hygroscopic quality, and this substance is frequently therefore, as recommended by Oidtmann, used for injection. Generally a very small quantity (three or four teaspoonfuls injected by means of a small glass syringe) is sufficient, though we need not fear to increase the amount to an ounce or two if thought necessary. In place of the injection we may also make use of glycerin suppositories. Of course other suitable remedies, as emulsion of castor oil, solutions of the neutral salts, especially Carlsbad salt, may be administered per rectum. Injections of this kind, however, will as a rule have to be limited to a single application, and cannot be employed repeatedly for any length of time.

Finally, there are certain cases of constipation which will not yield to cathartics, but must be treated with quieting and antispasmodic remedies. In these cases constipation is due to a temporary or permanent spasm of a circumscribed portion of the intestine, which may always make its appearance when the intestinal contents have reached the sensitive point. Leaving aside lead colic, we find in these cases, particularly in affections of the spine and of the brain, certain neuroses which are connected with a morbidly irritable condition of the cœliac plexus, and certain forms of hysteria. In these cases opiates, belladonna, chloral, especially croton chloral and extract of calabar bean, will often give wonderful results, which are, however, only temporary.

A prominent place in the treatment of constipation must be as-

signed to the medicinal springs, of which I will discuss from personal experience only the springs of Germany, Austria, and Switzerland. These may be divided into: 1. Waters containing pure chloride of sodium (the Wiesbaden Kochbrunnen—6.83 of chloride of sodium, 0.47 of calcium chloride, 0.42 of carbonate of lime per litre. 2. Waters containing chloride of sodium with a large quantity of carbonic acid gas (the Kissingen Rakoczy—chloride of sodium 5.82, chloride of lime 0.28, carbonate of lime 1.06, carbonic acid gas 1,392 c.c. to the litre). 3. Alkaline saline springs (the Carlsbad Sprudel—sulphate of sodium 2.5, carbonate of sodium 1.3, chloride of sodium 1.0; the Marienbad Ferdinandsbrunnen—sulphate of sodium 5.0, carbonate of sodium 1.2, carbonate of magnesium 0.9, chloride of sodium 2.0, and carbonic acid gas; Tarasp—sulphate of sodium 2.5, carbonate of sodium 3.5, carbonate of calcium 1.6, chloride of sodium 3.8, carbonic acid 4.5). 4. Alkaline muriatic springs (Ems Kesselbrunnen—carbonate of sodium 1.99, chloride of sodium 1.0, carbonate of calcium 0.22, and carbonic acid gas 553.2).

The action of the sodium-chloride springs lies mainly in hastening elimination and absorption, and in this manner increasing metabolism. Their influence on the intestine is probably both direct and indirect, producing a solution of the mucus and liquefying directly the contents of the intestine, and also stimulating the stomach and secondarily the intestine to increased activity. This action is greatly increased by the presence of carbonic acid gas, as is the case in the Kissingen springs. Large quantities of these waters are necessary in order to produce a cathartic effect.

The waters of Marienbad, Carlsbad, Tarasp, Rohitsh, Franzenbad, Elster, and others, act mainly by the sulphate of sodium which they contain, but they vary in their cathartic effect in different individuals. The most positive results in this direction are obtained by the Marienbad waters, on account of their high percentage of sulphate of sodium and their low temperature (7° C. = 44.6° F.), while the waters of Carlsbad, as already stated, not infrequently exert a constipating effect, their action being rather in the direction of relieving the catarrhal conditions which lie at the bottom of the trouble than directly cathartic. The same may be said of the alkaline muriatic springs. We must emphatically warn against carelessly sending delicate and anæmic or nervous individuals to springs containing Glauber's salt, which, on account of their comparatively difficult digestion and their strong percentage of salt, are frequently very badly borne by such patients. The selection of a suitable spa should be left to one who has had much experience and possesses a minute knowledge of the various springs. I am repeatedly consulted by foreign patients, and espe-

cially by Americans, as to the therapeutic value of the spa they are to visit, and frequently find that they have received erroneous and improper directions from home.

As regards the bitter waters, such as Friedrichshall, Seidlitz, Ofner, Püllna, etc., their action is due to the high percentage of sulphates, which reaches for example in the Püllna Bitterwasser to 16.7 gm. of sulphate of sodium, and 12.1 gm. of sulphate of magnesium. Those springs in which the sulphate of magnesium is in excess of the sodium salt are more pleasant in their action than when the contrary is true. Nevertheless, the action of these waters, as of those before mentioned, varies greatly in different individuals.

ILEUS—INTESTINAL OBSTRUCTION.

Ileus, miserere, or passio iliaca is nothing but a symptom which may develop in the different forms of acute and chronic intestinal obstruction and which ought not to be regarded as an independent disease. The name ileus was first applied to intestinal obstruction when only symptoms and not the fundamental causes were understood and differentiated. The reason why we find the name ileus used by physicians and in the text-books even to-day, is because every complete and lasting intestinal obstruction results in ileus. The most diverse causes lead to the same issue and thereby the latter is of importance. And so it is advisable to describe first the symptoms of ileus and then to consider the different parts of the intestine which may be the seat of intestinal obstruction.

By the name ileus we understand a variety of conditions which are brought on by the complete interruption of the passage of the intestinal contents. This may be caused either by a mechanical obstruction or by the lack of motor power, or by a combination of the two. Ileus is always an acute disease as it appears suddenly, but its causes need by no means be acute too. Indeed, the latter may have existed a long time. Thus the loss of motion of the intestines may develop very gradually and suddenly cause complete obstruction, or a complete obstruction may exist a comparatively long time before the symptoms of ileus develop. They may appear only after the intestine is completely filled. In some cases one is able to obtain fæcal matter by means of the stomach tube at a time when no characteristic symptoms of ileus can be observed. These symptoms may develop acutely or slowly, but usually acutely.

Symptoms.

The symptoms of ileus vary, depending on its causes, especially in the beginning, which may be very sudden and violent or come on gradually. Usually it starts with colicky pains, which are caused by internal constrictions or other conditions which lead to sudden intestinal obstruction. In cases of obstruction of the intestine caused by tumors we notice a slower development, although later it becomes acute, as there comes a moment at which the passage is suddenly obstructed by a swelling of the tumor, by scybala, or by foreign bodies, such as gall-stones, etc. Together with the pains a swelling of the abdomen is noticeable, which varies, the intestinal convolutions being sometines outlined on the abdominal wall, while sometimes the abdomen is evenly distended (meteorism). From the beginning of the disease it is self-evident that there is no passage of fæces or gas. But it is possible by means of an enema to obtain a certain amount of fæces, and so the inexperienced may in this way be deceived. This is only the contents of the intestine below the obstruction. The passage can be regarded as restored only if the patient is able to pass flatus or larger masses of fæces.

The patient becomes quickly depressed. The knowledge that the intestine is obstructed weighs upon him more or less, and he suffers from a feeling of anxiety and restlessness, which expresses itself in his contorted face and rapid emaciation.

The more the abdomen is swollen and is in the condition which we call meteorism the more the diaphragm is raised and interferes with the functions of the heart and lungs. The result is a feeling of oppression, dyspnœa, with superficial and mostly costal respiration; the pulse is accelerated, weak, and not very tense. There is a damming up of the intestinal contents above the obstruction. This condition favors decomposition, gas formation, and the production of those peculiar albuminoid substances which cause the characteristic odor of fæces. The appetite is entirely lost, a feeling of heaviness, fulness, and pressure in the abdomen appears and soon becomes insupportable, and the patient endeavors to find alleviation in vomiting or eructation of gas.

Soon eructation of gas comes on, and is followed by vomiting of food, bile, and fæcal matter, which may be so violent that the foul stercoraceous material is passed not only through the mouth but through the nose as well. Then appear cold perspiration, dryness of the tongue and mucous membrane of the mouth, and an unquenchable thirst, which is only partially relieved by swallowing water or small

pieces of ice or by the injection of water in the rectum. Oliguria and even anuria may supervene. The voice becomes weak, whispering, and hoarse. The extremities are icy cold and livid, the eyes are sunken and surrounded by dark rings, the nose is pointed, the cheeks are hollow, and we find a sure picture of the facies hippocratica. In short, there is a condition of intense suffering which can be relieved only by prompt interference on the part of the physician and even then often terminates in death.

Of the various symptoms the following will be described more in detail:

Pain is a frequent but not a constant symptom. It may vary between relatively slight colicky pains and the most severe cramps. They are caused by the tension of the intestine and its peritoneal coat, brought about by the increased peristalsis directed towards overcoming the obstruction. According to the nature and energy of the peristaltic motion, the pain is constant or intermittent. In the case of cancerous stenosis in the aged pain may be entirely absent, and the same is true occasionally in the case of other debilitated individuals.

We may consider as cause of this peristalsis, as Nothnagel has shown, the irritation which the formation and accumulation of gas exerts on the intestinal coat and its nerves. As the peristalsis of the small intestine is normally more active than that of the large intestine, so, when obstruction occurs in the former, pain appears earlier and is more marked than when it occurs in the latter. It is not always referred to the site of obstruction; thus when obstruction exists in the small intestine the pain is felt, as a rule, in the region of the umbilicus, although often the obstruction is found far from the seat of pain. I have more than once seen an incision for laparotomy made on the right side because the patient had referred his pain to this side, and it was accordingly thought that the disease was on this side, when in reality there was in one case a tumor of the splenic flexure of the colon, and in another case a constriction of the small intestine situated in the left hypochondrium. Hence one must be cautious not to lay too much stress on this symptom, especially in the later stages of obstruction, but also in the early stages as well.

Vomiting of fæces begins by eructation of gas and singultus. The gas has already a feculent odor, and by means of the stomach tube one may obtain a large amount of a brownish fluid which also has a feculent odor. There soon appears vomiting of this brownish fluid or semi-fluid mixed with shreds of mucus and undigested food of the characteristic odor. As much as a quart may be vomited. Then the patient is relieved for a while, and in spite of the disgust caused by vomiting feels comparatively well, at least for a few hours, half a day,

or a day, when the vomiting begins anew. It is surprising to note how quickly and in what quantities the contents of the intestines flow up into the stomach, a fact which can be demonstrated by washing out the stomach at intervals of several hours. Formed masses are never vomited. The few cases which have been reported (Rosenstein, Denos, Legendre, Jaccoud) were either based on diagnostic errors or on conditions which had nothing to do with intestinal obstruction, as, for example, the case of Rosenstein, which proved to be one of hysteria. Fleischer states in a recently published text-book that the patient vomits "first food from the stomach, soon after abundant green bilious matter, and then dirty greenish, changing to brownish granular matter with at first only a slightly feculent odor," but my own experience has shown that this is true only of cases in which peritonitis also exists. Usually the vomited matter is fæcal in character from the very beginning. In the majority of cases it is the contents of the small intestine driven back into the stomach, which under normal conditions have no fæcal odor. But in cases of obstruction the putrefaction which normally occurs in the large intestine now takes place in the small intestine. It is easy to see that when the contents of the small intestine are stagnant the bacteria of putrefaction already contained therein find a suitable soil in the liquid albuminous matters, and hence the rapid decomposition and putrefaction. How the vomiting of fæces is really brought about, whether by normal peristalsis or by an antiperistaltic action, and to what extent it is due to a peritoneal reflex is not yet fully known. Nothnagel's experiments show that if a ligature is placed around the intestine, peristalsis ceases. But a movement can occur which he calls "retrogressive contraction," and which has the following characteristics: When the portion of the intestine immediately above the occlusion becomes tense from the accumulation of matter from above, then a contraction occurs here and forces the intestinal contents a short distance backwards. The consequence is that the intestine is completely emptied just above the occlusion. After this process has been repeated several times it refills slowly. Now the whole process repeats itself farther up and so the intestine gradually becomes distended. In this condition it only needs a slight irritation to cause vomiting of the fæcal matter, which has no other outlet. Huguenot, of Montpellier, offered the same explanation as far back as 1713, but it remains unexplained, as Nothnagel remarks, how the undecomposed contents of the upper and the decomposed or fæcal contents of the lower intestine become evenly mixed in the stomach or the intestine.

The Outward Appearance of the Abdomen.—The abdominal wall is as a rule tense and shining, but its appearance varies according to the

length of time the occlusion has existed, and it is either evenly swollen or there are local and circumscribed swellings, or a number of intestinal convolutions are outlined on the abdomen. A strong peristalsis is sometimes visible, at other times a single intestinal convolution appears fixed and distended. A peristaltic movement may sometimes be observed in a fixed convolution. The importance of these various conditions in the diagnosis of the different forms of obstruction, such as strangulation, volvulus, or twisting, will be treated more fully later on.

Palpation.—In general the abdomen feels doughy and elastic. We are able but rarely, unless we have an opportunity to examine the patient in the early stages, to find a resistance deep in the abdomen or an actual tumor at the site of the obstruction. Usually there is no point of tenderness, but all parts of the abdomen are equally sensitive to pressure. If the obstruction is caused by a tumor which has existed a long time we may be able to make out the new growth or there may be a circumscribed area of tenderness. It may also be possible to detect a localized tenderness on palpation if the physician is able to examine the patient immediately after an intussusception or a twisting of the bowel has occurred.

Percussion.—We can obtain information of value by this procedure only when the accumulation of gas in the convolutions and the general meteorism are not far advanced. But percussion may give us some idea as to the mobility of the intestines. If peristalsis still exists the percussion note will vary from time to time in some part of the abdomen or over the same convolution, according as the tension of the contained gas varies as a result of the contraction of the intestinal walls or of the change of relation of the individual convolutions. Hence the note may range from the highest to the lowest, from tympanitic to flat, and this may be even more distinctly demonstrated by means of auscultatory percussion. But if peristalsis has stopped, the percussion note varies little or not at all.

On *auscultation* we may hear loud gurgling or splashing sounds, which cease as soon as the administration of opium stops the peristalsis. The striking fact has not yet been explained why there should be no meteorism in cases of absolute occlusion caused by persistent and chronic constipation, whereas in acute obstruction a large amount of gas forms in a very short time.

This has been explained by Zuntz and Taiker, who have shown by experiments on animals that the gas is absorbed by the blood-vessels of the intestines and is then exhaled from the lungs, only a small portion being discharged as flatus. The absorption of gas from the intestine depends on the integrity of the circulation in the intestinal

wall; this is much impaired in cases of acute obstruction, but only gradually and slowly affected in chronic constipation. From this it results that the gas, which forms quickly and abundantly in the stagnant and liquid contents of the intestine, is retained in the intestines, and the more the circulation is impaired the less gas is absorbed by the blood-vessels. If an acute inflammation and hyperæmia of the mucous membrane be complicated by a paralysis of the muscles of the intestines—as in circumscribed or general peritonitis—the accumulation of gas is extreme.

Peritonitis.—This is a frequent complication of ileus. It is caused usually by rupture or gangrene of the intestine, in some cases by ulceration which extends to the peritoneum or by the passage of bacteria from the intestine to the peritoneum, as has been shown by the experiments of Nepveu, Bönnerken, and Reichel, according to whom bacteria can pierce the usually impermeable intestinal walls if these latter have become paralyzed. There is an exudation or transudation of blood and blood serum, not as a result of bacterial irritation but because of the impaired circulation of blood characteristic of this particular form of ileus. A general peritonitis is more usual in cases in which the intestinal walls have become severely and rapidly injured as in strangulation; it is less frequent when the obstruction is caused by constipation. In conditions of collapse peritonitis is frequent. Collapse is due in part to autointoxication brought about by the absorption of bacterial secretions and of the products of decomposition of the intestinal contents.

I have seen several cases of strangulation which ended by death before the full complex of symptoms of ileus had developed, that is, before vomiting of fæces had set in. Such conditions can result only from acute autointoxication, and may be compared to the symptoms of septicæmia, such as chill, singultus, and cardiac paralysis. The course of the *temperature* varies and depends in great measure on the presence of inflammatory processes, especially peritonitis. When the latter occurs the temperature may rise to 39°–40° C. (102°–104° F.). It may show remissions in the morning, or if collapse occurs, the temperature may suddenly fall below normal. If no complications arise the temperature is as a rule normal or subnormal.

The *urine* in cases of intestinal obstruction is diminished, of high specific gravity, and rich in urates. Of special importance is the presence of indican in the urine. It appears quite early in cases of obstruction of the small intestine, whereas in obstruction of the large intestine it is absent altogether or appears only late in the course of the disease. If the obstruction occurs in the upper portion of the small intestine, then the amount of indican secreted by the kidneys is

very small. This is easily understood when one considers, as Jaffé has proved, and as many others have asserted, that the formation of indican takes place only in the middle and lower part of the small intestine.

Complications.

Various complications may arise in the course of ileus. Perforation of the wall of the intestine is relatively frequent. Fistulæ may be established between the intestine and the external air through the abdominal wall, or between adjacent convolutions, or between the intestine and bladder, and sometimes the fistulæ may open into the uterus or vagina. But these complications are rare and have no practical importance. They are recognized by their specific and characteristic symptoms. Thus intestinal perforation is shown by an absence of liver dulness and development of serous exudation. If the patients are able to tell about their condition, they say that the colic-like pain suddenly disappeared. This is due to the escape of gas into the abdominal cavity and as a consequence the tension of the intestinal wall is relieved.

Diagnosis.

We have to consider two problems, first, whether there exists a true intestinal obstruction, and secondly, the nature and seat of the obstruction.

Diagnosis of Intestinal Obstruction.

Intestinal obstruction may be confounded with general peritonitis, acute typhlitis and perityphlitis, and persistent constipation, less frequently with Asiatic cholera, strangulation of a floating kidney, renal calculus, lead colic, displacement of the uterus, or ovarian tumors. Although there have been some features in common with intestinal obstruction, they may usually be readily differentiated if one is cautious. Only in the early stages is the diagnosis between peritonitis and intestinal obstruction at all difficult. In chronic cases the history is altogether different. Firstly, the course of the temperature is different; in peritonitis the temperature shows exacerbations towards evening, but in intestinal obstruction it is normal or subnormal. Secondly, the presence of conditions which prove that peritonitis exists, especially tuberculosis and carcinoma of the intestine and abdominal wall, will facilitate the diagnosis. In acute peritonitis the history of the case is of the greatest importance. The existence of gastric ulcer, of perforative appendicitis, of an intestinal ulcer, of diseases of the adnexæ, of diseases of the biliary ducts, liver,

and portal vein, would enable us to decide in doubtful cases that the symptoms of obstruction were due to disease of the peritoneum.

We may add that in peritonitis little or no peristalsis is noted, as the intestines are very soon paralyzed, and tympanites is, according to my experience, not so marked as in intestinal obstruction, and is, moreover, not localized. When single convolutions are distended or show active movement, we are probably dealing with intestinal obstruction and not with primary peritonitis. The temperature is from the beginning higher in peritonitis than it is in cases of intestinal obstruction. But, as I have often observed, and as Naunyn and every physician of experience may have noted, the temperature in cases of intestinal obstruction resulting from strangulation or volvulus rises rapidly and may reach 102° or more. Madelung and Lennander have stated that in peritonitis the difference between axillary and rectal temperature is greater than in intestinal obstruction or in the normal state. We must wait to see if their experience is substantiated. Neither Naunyn nor myself has been able to verify this assertion. It may, however, be dependent upon local conditions. Some writers attribute much importance to the fact that in peritonitis the vomiting usually appears sooner and is more violent than in intestinal obstruction. This is not always the case, however. I have seen cases of the latter begin with violent vomiting. Indicanuria is the same in both diseases, especially when the obstruction occurs in the small intestines.

The character of the pain is of importance. In intestinal obstruction it is localized; in peritonitis the whole abdomen is tender.

The diagnosis between ileus and perityphlitis or perforative appendicitis is usually not difficult. The history of the case, the site of the swelling, the course of the temperature, the relatively slight impairment of the general condition of the patient are sufficient data to enable us to make a diagnosis. But if the history of the case is indistinct or cannot be obtained; if the characteristic symptoms of appendicitis are absent; if there is complete occlusion of the intestine without the passage of gas; if the abdomen is swollen with distinctly visible intestinal convolutions; if there is frequent vomiting of a fæcal character, and if this continues a long time—say eight or ten days—then the question whether we are dealing with intestinal obstruction or appendicitis with circumscribed peritonitis is a very difficult one to decide. I have observed two such cases. In both I have concluded from the relatively good pulse and general condition that intestinal obstruction could not exist, and the subsequent observation of these cases confirmed my diagnosis.

Sometimes it is difficult to differentiate between ileus caused by a simple stoppage of the intestinal contents and a mechanical obstruc-

tion. In hysterical patients we may sometimes observe constipation lasting weeks, with extreme distention of the abdomen and complete absence of flatus, which resists all medical agents. However, in such cases there is never much indican in the urine and the washings from the stomach do not contain fæcal matter. Strange to relate, in these cases of hysteria there is no vomiting at all; also the general condition of the patient is but little impaired. The peristalsis, which appears in the distended convolutions and which is characteristic of strangulation, is absent. In spite of the long duration of the constipation the circulation in the intestinal wall is not interrupted, and hence there is not much decomposition or accumulation of decomposed matter. This explains why the patient is not profoundly affected. However, there may be some special reasons why some individuals, and especially nervous and hysterical persons, can continue to be constipated for days and weeks without manifesting those dangerous symptoms which in others arise at once.

Diagnosis of the Nature and Seat of the Obstruction.

After the diagnosis of ileus has been made, we must determine whether the constriction in the intestinal canal is chronic or acute, and then we differentiate the various causes of ileus. Every case of ileus is in the beginning acute but the cause may be acute or chronic. By chronic causes we mean tumors in or adjacent to the intestinal wall, cicatrized ulcers, or the rare cases of partial twisting of the sigmoid flexure. Acute causes are internal kinking, incarceration, strangulation, volvulus, intussusception, obstruction by gall-stones or intestinal calculi, etc.

Chronic constriction of the intestine is determined by the history which points towards a long-existing intestinal disease and disturbances of defecation. Later on mild attacks of ileus may appear or there may be a sudden attack of severe ileus. In one of my cases I have observed three attacks of ileus within half a year. The first two attacks disappeared rapidly, but in the last I was compelled to perform colotomy. It was a case of carcinoma of the splenic flexure of the colon. I therefore attribute more importance to this symptom than does Naunyn. There may be repeated attacks of ileus in cases of incarceration and volvulus of the sigmoid flexure, but they do not occur at such short intervals and are more apt to be caused by the age or habits of the patient. Hilton Fagge and Fenwick as well as Naunyn have drawn attention to the fact that in cases of chronic obstruction of the small or large intestine there is frequently a strong, visible, and palpable peristalsis in certain fixed or slightly movable convolutions. But Naunyn and Schlange maintain that this is not

peculiar to chronic obstruction. In consulting the notes of my own cases, I find this symptom in five cases out of nine of chronic ileus. In one of these cases this symptom led me to infer that the seat of obstruction was in the ileocæcal region, and I accordingly performed laparotomy in this portion of the abdomen, but found that the obstruction was caused by a carcinoma of the splenic flexure. Hence this symptom is not always to be relied on.

Further, ileus in chronic intestinal obstruction is less violent than in acute cases. It is as though the organism had accustomed itself to the difficult passage of intestinal contents through the existing stenosis, and could thus endure more easily the consequences of complete obstruction.

Seat of Intestinal Obstruction.—Nothing is more difficult than to determine the exact position of the obstruction. Except in cases in which the obstruction is situated in the upper part of the duodenum or quite low down in the large intestine it is extremely difficult and often impossible to make out the portion of bowel affected, and in some cases this is very difficult even after a laparotomy. There are, however, a number of features the presence of which facilitates the diagnosis, but they may be entirely absent. The diagnosis is easiest when the seat of obstruction is at the extreme upper or lower end of the intestinal canal. When it occurs at the lower end of the *large intestine:* 1. The presence of a tumor may in some cases be determined by digital or manual palpation per rectum. 2. It may be found that only a very small quantity of water or air can be injected into the intestine; but it must be remembered that many nervous or neurasthenic or even very stupid people cannot retain even the smallest quantities of air or water in the rectum; if these people are trained, however, the desired result may be obtained. 3. The absence of indican in the urine can be determined when obstruction occurs at the lower end of the bowel. 4. The late appearance of symptoms of ileus, marked meteorism, and relatively slight collapse, point towards an obstruction of the lower portion of the intestine.

On the other hand, the symptoms which indicate that the obstruction is in the jejunum or *small intestine* are: (1) The early onset of biliary vomiting, often in great quantities and intestinal but not fæcal in character as a rule; (2) no swelling of the abdomen; (3) sudden collapse, and, as this is an acute form of intestinal obstruction, (4) the appearance of acute pains in the upper part of the abdomen. The absence of indican in the urine is noted when the obstruction is seated in this portion of the intestine as well as when its seat is in the rectum.

Cases in which the seat of obstruction is somewhere midway between these extremes are exceedingly difficult to diagnose, as regards

both the character and the situation of the obstruction. According to my own experience, we must not place too much reliance on what has been said about the location of the pain, the existence of single distended convolutions, and the more or less distinct tumor, which may be palpable or give circumscribed dulness.

In favorable cases the course of the disease and the appearance of the various symptoms may be such as to enable one to make a precise diagnosis, but no one symptom can be relied upon. Here we must mention the existence of a distended convolution. My experience coincides perfectly with that of Naunyn, who says that no one symptom has deceived him so often, in making a diagnosis of the seat of obstruction, as the presence of a distended convolution.

The following features are important, as they point, either singly or when taken as a whole, towards the seat of the obstruction: (1) The position of the fixed and distended convolution which may show peristalsis; (2) the existence of a puffy swelling as determined by percussion and palpation; (3) the character of the swelling—whether it is a tumor, a fæcal collection, a distended knuckle of intestine, or of other nature; (4) the results of a digital examination of the rectum, vagina, and all orifices and places where hernia may occur; (5) the result of the injection of water or air into the bowels; (6) the character and location of the pain. I have mentioned above the value of these various symptoms. In my experience I have found that it is easier to judge approximately of the location of the acute obstruction in children. In adults the seat of a chronic obstruction is more easily determined than that of acute obstruction, especially as the seat of a tumor is so often in the splenic flexure or in the descending colon. For the precise determination of the seat of obstruction in acute cases a weighing and comparison of all the important symptoms are necessary.

Varieties of Intestinal Obstruction.

Acute intestinal obstruction may be caused by internal incarceration, by volvulus or flexion, by intussusception, by obstruction by gall-stones, intestinal calculi, fæcal masses, or foreign bodies, and lastly by external compression.

Internal Incarceration.

This is most frequent in persons under forty and usually has its seat in the small intestine. The greater the irritation of the peritoneal coat the more intense are the initial symptoms: pain, vomiting, and collapse.

Internal incarceration may result from (*a*) peritoneal bands and adhesions between the various organs, (*b*) Meckel's diverticulum, (*c*) mesenteric fissures and perforations, (*d*) internal hernia. All these various results have, in spite of their anatomical differences, practically the same effect on the intestine, viz., constriction and strangulation. Acute and chronic inflammations of the peritoneum may result in the formation of bands which may cause constriction of the intestine. These bands may vary in thickness from a mere thread to a wide, flat, ribbon-like formation of varying length. The omentum also may be converted by chronic peritonitis into a ropy adherent mass. These adhesions may form between the uterus, ovaries, and mesentery, causing constriction of the intestine, a band may be attached at both ends to the omentum and embrace a convolution, etc. Finally the vermiform appendix may be attached by an inflammatory process to an adjacent coil of intestine or to the omentum or to the wall of the pelvis or to a fixed organ, such as the kidney or the uterus, and so constrict an incarcerated knuckle of intestine. In the same way the Fallopian tube may become attached to the intestine and cause obstruction.

A surprising variety of combinations of incarceration results from the relation of these bands to the intestinal convolutions, but they may be divided into two groups: The incarceration may be under the band, *i.e.*, the intestine is simply compressed between the band and some firm body, or the band forms a sling or knot in which the intestine becomes engaged and is thus constricted. Thus the most complicated twistings occur which are difficult to disentangle even by dissection.

Leichtenstern has mentioned in his text-book all the possible complications based on a large number of cases observed by him. In Fig. 16 are represented some of the principal types. They are more of anatomical than of practical interest. It is of practical importance to know only whether the incarceration or entanglement is one that might loosen by itself or whether it can be relieved only by an operation. This is determined by the course of the disease. In general the incarceration under a band is more common than the formation of knots or slings; the proportion being 6:1 (Treves) or 2.3:1 (Leichtenstern). Rare forms are the hanging of a segment of intestine over a band and an acute bending caused by traction. In the first instance the intestine hangs over the band, just as a wet rag would hang over a string, whereby the lumen is entirely closed. In the second case the angular bend is caused by the band being attached at one end to the intestine and at the other end to some organ which suddenly becomes dislocated or changes its form, as, for example, a

floating kidney, a punctured ovarian cyst, or the uterus after expulsion of the fœtus.

Meckel's diverticulum is formed by the persistence of the omphalomesenteric duct (it must not be confounded with the occasional hernia-like protrusions of the intestinal mucous membrane through an orifice in the intestinal wall, which never cause intestinal constriction). The diverticulum will have the same effect as the peritoneal bands if the end becomes attached to anything. But it may also, if it hangs free in the abdominal cavity, encircle an intestinal convolution and constrict it. When this occurs the diverticulum must have on its free end a button-like swelling which prevents the end from slipping away after the sling has once been formed (see Fig. 17).

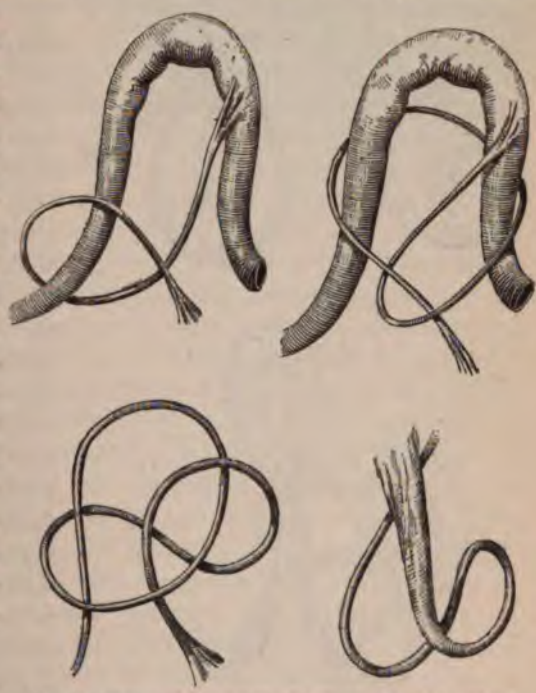

Fig. 16.—Types of Constricting Peritonitic Bands. (After Leichtenstern and Treves.)

Incarcerations in tears or perforations of the mesentery and omentum, either congenital or resulting from trauma, are rare. The convolution slips into one of these apertures when empty, then, after it becomes gradually filled, it can no longer slip back.

Of the so-called internal herniæ, hernia into the foramen of Winslow, diaphragmatic hernia, and Littré's hernia are of practical interest. The hernia which occure in the pockets of the peritoneum, in the recessus duodenojejunalis, præjejunalis, intersigmoideus are only of anatomical interest. This department of medical literature is rich in curiosities of this sort which are of more importance to the pathological anatomist than to the physician.

Among the herniæ of the diaphragm we distinguish that which has a sac formed of pleura or peritoneum and that which passes directly into the pleural cavity. The former is more rare than the latter, the ratio being about 1:10 of all herniæ of the diaphragm.

Usually the opening is in the left side of the diaphragm (six times oftener than in the right), for on the right side the liver forms a barrier against the protrusion of the abdominal organs into the pleural cavity. We distinguish congenital and acquired openings in the diaphragm. The former are round with smooth edges, the latter are caused by tearing of the diaphragm by external traumatism, and are rents with irregular, ragged edges. Congenital apertures in the right side are rare, because of the fact that in early fœtal life the liver is closely connected with the diaphragm. We meet much oftener with acquired herniæ of the diaphragm. Thoma[10] in 1882 reported two hundred and ninety cases. Since then many more have been reported which in most cases ended with symptoms of incarceration and intestinal obstruction. Of practical importance is the rare case of Abel,[11] because it proves that diaphragmatic herniæ may be recognized during the life of the patient with comparative ease. The case was that of a woman thirty-three years of age. The symptoms suggested incarceration of the intestine; they were sudden onset of vomiting after a restricted diet, profound collapse, constipation, retracted abdomen, marked swelling of the left side of the thorax. The stomach could not be irrigated, the tube being obstructed before the cardiac orifice was reached, and the fluid which was poured in being at once regurgitated. The diagnosis was based on the following symptoms: (1) The boat-like retraction of the abdomen; (2) the distention of the left thorax, over which the percussion note was loud and tympanitic with absence of respiratory sounds; (3) considerable displacement of the heart towards the right; (4) no passage of fæces or gas; and (5) profound and sudden collapse.

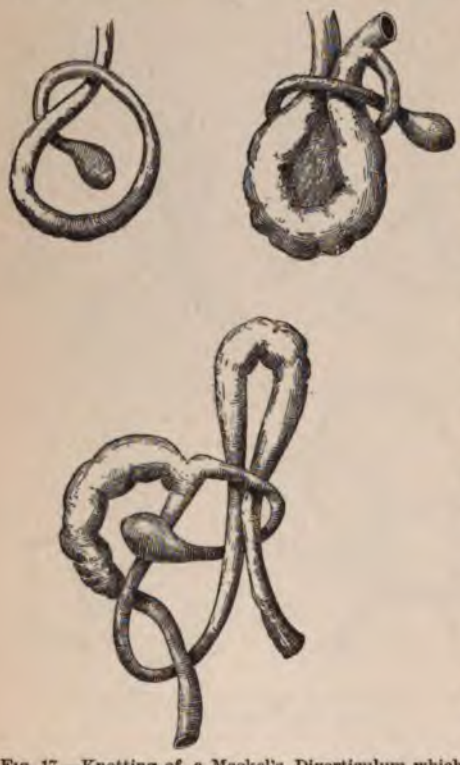

Fig. 17.—Knotting of a Meckel's Diverticulum which has a Button-like Swelling of its Extremity. (Treves.)

Leichtenstern,* in one of his cases, was able to demonstrate the presence of intestine and stomach in the pleural cavity, and could note the changes in size of the latter by the introduction of water through the stomach tube. This was not possible in the case of Abel, in which it was seen at the autopsy that the œsophagus was twisted on its axis so that no fluid could reach the stomach. Of special importance, as P. Guttmann has remarked, is the displacement of the heart, and this is more pronounced when the stomach and intestines are distended. If the distention subsides then the heart, or rather the apex beat, retreats again to the left. By this symptom, apart from the mode of origin (usually after an error in diet), the retraction of the abdomen, and the stoppage of fæces, we are able to differentiate between this condition and pneumothorax. The incarceration is produced in the same way as in other herniæ—namely, the intestinal convolutions and stomach gradually become distended with their accumulating contents, and then the disturbance of circulation in these organs causes a transudation of serum, an exudation of blood, rapid formation of gas, and swelling of the walls of the incarcerated organs, all of which prevent their return to the abdominal cavity. It is characteristic in cases of incarceration of the stomach that the greater curvature is turned upwards while the lesser curvature is directed downwards. If the fundus only is dislocated while the pyloric end remains in place, then a twisting on its own axis may result. The stomach and transverse colon are dislocated with equal frequency, while only rarely do we observe diaphragmatic hernia of the other abdominal organs.

Internal incarcerations occur almost without exception in the lower portion of the ileum, almost never in the colon or jejunum, never in the rectum or duodenum. This is due to anatomical conditions, as the ileum has a broad mesentery which permits of great motility, and has also a close relation to the pelvic organs. The length of the incarcerated intestine varies from an inch or two to a yard, but this has no influence on the severity or course of the clinical symptoms.

A diaphragmatic hernia may be diagnosticated, but all other incarcerations of this nature can be made out only on autopsy.

Pathological Anatomy.—The changes in the wall of the intestinal coil which is the seat of an internal incarceration are the same as those seen in cases of strangulated hernia. They depend upon the nature of the incarceration, the length of the convolution, and the duration of the disease. First, there is a venous stasis in the convolution above the incarceration, and the convolution is filled usually with liquid intestinal contents and gas. The wall of the intestine is swollen and of a dark red hue. The mucous membrane and the underlying tissue may be the seat of a diphtheritic or gangrenous inflammation

which may lead to perforation and its usual consequences. These changes may be due, in part, to so-called pressure necrosis, but only, of course, when hard masses are contained in the intestine; or, what is more frequent, there is a thrombosis in the vessels of the mesentery and this causes gangrene (Kader). The perforations, as Kader asserts, may be too small to be seen with the naked eye, yet the contents of the intestine may escape through these small perforations and then the intestine collapses, and in this way the incarceration may be relieved.

Volvulus.

In the majority of cases of volvulus the intestine turns round its mesentery or more rarely round its own axis, whereby a half or a whole turn is effected. Most frequent is the mesenteric volvulus of the sigmoid flexure. This is favored by a very large sigmoid flexure and a relatively long and narrow mesentery, so that the main portion of the mesocolon is changed into a kind of pedicle around which the segments of the convolution may turn. Such a condition is usually brought about by chronic constipation, whereby the bowel, loaded with fæces, exerts a constant pressure and tension. It is less frequently caused by chronic peritonitis and is rarely congenital. A violent irregular movement of the intestine, a jump, a trauma, or (as in a case of Israel) a high injection may cause the torsion which may be double or even triple. A twisting of one convolution around another may be from above downwards or from behind forwards, or there may be a combination of the two. Only rarely do the convolutions return to their normal position without surgical interference. Usually the volvulus becomes more and more pronounced, caused partly by the increase of the contents of the intestine through the accumulation of exudation, blood, and gas, and partly by the weight and pressure of the overlying convolutions. Moreover, there is present an atonic condition of the intestine brought about by the long antecedent constipation. This is the reason why volvulus occurs mostly in advanced years—above forty.

A curious case is reported by Leichtenstern of a boy of eleven years, who died of pseudoleukæmia and who had never suffered from constipation and consequently not from symptoms of intestinal obstruction, in whom there was found a volvulus of the sigmoid flexure. When air was injected into the colon the sigmoid flexure returned to its normal position, but again became twisted as soon as the air was evacuated from the bowel. Whether this was a chronic condition arising *intra vitam* or was a premortal occurrence it is difficult to say, for we know that premortal invaginations may occur.

The sigmoid flexure soon becomes so distended in cases of volvulus that it fills the abdomen and may even drive the diaphragm upwards so that disturbances in the thoracic organs may result. Treves reports the case of a patient who died on the seventh day, in which the diaphragm was pushed up as far as the third rib, and the lower lobes of the lungs were hepatized and contained no air.

In this form of intestinal obstruction perforation is rare. Treves found only two cases in twenty. Peritonitis, however, is frequent and of early occurrence. It begins at the sigmoid flexure and rapidly spreads over the whole peritoneum. The tenderness is accordingly first local and soon becomes diffused over the whole abdomen. The pain is moderate and sometimes intermittent, and comes on usually very suddenly. If there is any vomiting, it is rarely stercoraceous. The duration of this and of the following form of intestinal obstruction, which end by death unless operation is resorted to, is short—usually five or six days. It seldom drags on for a longer period, but Treves has reported a case which lasted twenty days. Death results in all such cases from peritonitis and septic infection or from disturbance or cessation of the function of the thoracic organs, especially paralysis of the heart, caused by the displacement upwards of the diaphragm. Several cases have been reported in which syncope and death occurred, while the general condition of the patient was relatively good, and on autopsy no sufficient cause could be found. This can be explained by assuming that there was a sudden paralysis of the heart.

Volvulus in other parts of the colon or small intestine is less frequent. Here smaller or larger portions of the gut become twisted. Even the whole ileum or jejunum may be the seat of volvulus, but the course of development is the same as in volvulus of the sigmoid flexure.

A turning of the intestine around its own axis or a lateral inflection, which according to Leichtenstern is rare, occurs exclusively in the cæcum and ascending colon. And in these cases there must be abnormalities in situation of the convolutions or in the length of the mesentery which allows an extraordinary mobility of these organs.

The entanglement of intestinal convolutions among themselves, which is most frequent between the sigmoid flexure and the ileum, more rarely between the other convolutions and most rarely between the cæcum and the small intestine, is very diverse in nature and sometimes so complicated that the most intricate mariner's knots are simple in comparison and one cannot conceive how they could be caused. Especially in old medical literature we find curious instances of this condition. At the present day the interest of the practical

physician in these pathological curiosities has diminished and all such cases are simply "obstruction by strangulation." It requires an extensive mobility and a very long mesentery to effect these changes. Leichtenstern and Treves, in their repeatedly quoted monographs, give numberless instances of such twistings and knots; but as we can only refer to them here, the accompanying illustration from Leichtenstern's work (Fig. 18) may serve as a type. In regard to the clinical symptoms and course of this form, I can only refer to what I have stated concerning volvulus of the sigmoid flexure. In the condition under consideration, as well as when the sigmoid flexure is the seat of the trouble, the onset of symptoms is more or less sudden, the vomiting is violent, the pain is severe, and there is absolute constipation.

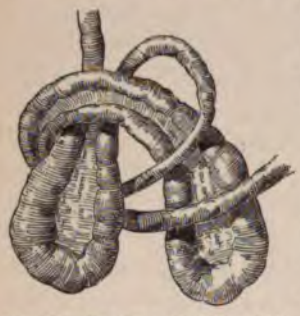

FIG. 18.—Knotting of Coils of the Ileum with each other. (Leichtenstern.)

In the diagnosis of these forms of strangulation, one symptom, to which Hilton Fagge, Wahl, and his pupils give much attention, is of special importance. It consists in the fixation and distention of the convolution immediately above the point of strangulation—the so-called fixed distended convolution which is the seat of marked peristalsis, which may even be visible, as in two of the twenty cases of Treves.

That this is by no means always the case, however, is shown by the observations of Schlange and Naunyn. The disturbed convolution may be obscured by an overlying coil of intestine, as may be observed in volvulus of the sigmoid flexure.

In the beginning, the left inferior part of the abdomen, or the region of the umbilicus, is distended and there is a springy, elastic tumor to be felt which has no peristaltic motion. Here the percussion note is tympanitic or dull or flat, according to the degree of fulness of the distended convolution of the sigmoid flexure. Gradually as the accumulation of gas increases the convolution rises and fills the upper right side of the abdomen. Finally the entire abdomen becomes uniformly distended, and then we are unable to say whether other symptoms, for instance peritonitis, may not be concerned in the causation of this swelling of the abdomen.

Such a sudden peritonitis with a free serous or hemorrhagic exudation into the peritoneal cavity is a frequent complication of strangulation. Naunyn draws our attention to the passage of blood with the stools—in one case a quart of blood was passed on the third day. In the coils above the strangulations blood may be exuded from the

congested mucosa into the lumen of the intestine, and if the patency of the intestinal canal is restored this blood will be passed with the stool. Such cases have been repeatedly observed.

The following symptoms are important in the diagnosis of this condition: Sudden collapse with violent, often intermittent pain, and slight but feculent vomiting. In these cases collapse is regarded as a consequence of septic infection. It therefore appears sooner in obstruction of the small intestine than when the sigmoid flexure is the seat of obstruction. Other symptoms of volvulus of the sigmoid flexure, besides the above-mentioned characteristic position of the distended fixed convolution, are pain in the left side of the abdomen, the impossibility of introducing water or air through the rectum, absence of indican in the urine, and early tenesmus.

When a part of the intestine is twisted so that a half-turn only is effected, then the symptoms are those of a partial stenosis of the intestine; but if a mass of fæces converts this condition into one of complete obstruction, then the symptoms of strangulation appear, but they may disappear suddenly if the obstructing mass of fæces is passed on.

The course of the symptoms in strangulation is rapid. The climax is reached in a few hours, and unless an operation is performed, except in cases in which only a half-turn is effected, the patient quickly succumbs.

Intussusception.

This is the invagination of one portion of intestine into the lumen of the adjoining portion. It may occur in either of two ways—the upper part passing into the lower or the lower into the upper. We distinguish between the intussusception with an inner and an outer tube, and the intussusception or sheath which encloses the invaginated intestinal segment. This may be demonstrated with the finger of a glove. The sheath may itself become invaginated so that there may be a double or even a triple intussusception (there being then seven cylinders of intestine).

The intussusception may be physiological or pathological. The physiological or agonal invagination occurs just before death in the small intestine, usually in children. These invaginations are small, often multiple, and occur in either or both directions—upwards and downwards. As they occur shortly before death there are no inflammatory adhesions between the layers, and so the invagination may be reduced by a gentle pull. They arise, according to the recent acceptation, by reason of the fact that during the agony different portions

of the intestine die at different times, and the peristalsis of the still living parts causes the invagination. Nothnagel found this condition in a healthy living rabbit. Cruveilhier, Rafinesque, Treves, and Bell have also observed the condition during life. The latter found an easily reducible invagination during a laparotomy, so it seems this may also occur physiologically in man at other times than during the agony.

In contradistinction to the above is the pathological invagination which occurs during life and is inflammatory. The invagination may be descending or ascending—the former being more frequent. Leichtenstern found only eight cases of ascending invagination in five hundred and ninety-three cases. Besnier (invagination of the sigmoid flexure into the descending colon), D'Arcy-Power (transverse colon), and Johns (descending colon) report cases of ascending invagination. Hectoen reports a very rare case of fourfold invagination of the ileum, all ascending. Multiple invaginations, invaginations at more than one portion of the intestine, are exceedingly rare.

According to the seat of the invagination we differentiate enteric invagination (small intestine into small intestine), colic invagination (large intestine into large intestine), and ileocæcal invagination (small intestine into large intestine). This last variety is the most frequent —fifty-two per cent., and in children even seventy per cent. Next in frequency is enteric invagination—thirty per cent.; then colic invagination—eighteen per cent. If the ileum is the seat of invagination it occurs in the lower part; if it occurs in the colon it affects the sigmoid flexure. Very exceptionally we find invagination of the duodenum.

There are two opposing theories as to the mode of production of an intussusception. Some believe that a segment of intestine becomes paralyzed and then falls into the portion of intestine immediately below. Others think that the annular muscles of a limited portion of the intestine contract in such a way as to form a cone or funnel, which is driven by peristalsis of the upper portion into the lower adjoining intestine—spasmodic invagination. Nothnagel believes that the motion is not in the upper but in the lower portion, which later forms the sheath. He succeeded in producing an intestinal invagination in animals by electrical stimulation of a limited portion of intestine, and could then see the lower segment draw itself like an umbrella over the contracted cone, and by the wormlike movements of the longitudinal muscles it embraced the upper segment more and more. We must therefore look for the active force in the part below what is finally the internal cylinder, that is, the part which forms the middle and outer cylinder, or sheath. This explanation holds good only for

descending invagination. In ascending intussusception the whole process is reversed.

In cases in which the intestinal wall is subjected to a direct pull, as when there is an intestinal polyp or other kind of tumor arising from the intestinal wall, the explanation is much simpler. It is easily understood that such a cause leads primarily to a partial invagination, that is, of a segment of the intestinal circumference. But through the pressure exerted by fæces the invagination soon becomes complete. We are forced to admit, however, with Nothnagel that such tumors and polyps are often so small that their weight could exert no appreciable traction, hence they may be disregarded as a causative factor. In such cases we must assume the presence of spasm as the cause of the invagination.

Carcinoma seldom causes invagination, but benign tumors, such as adenoma, fibroma, myoma, or lipoma are more apt to cause this accident.

Conditions which cause abnormal active peristalsis, partial paresis, and paralysis favor invagination. Leichtenstern has collected 326 cases in which the cause is given. Only 111 cases occurred in previously healthy individuals. In all the other cases there was a previous history of diarrhœa, contusion of the abdomen, pregnancy, disturbances of function of various organs, and other symptoms of a weakened general condition of the patient.

Invagination is most frequent in children. According to Leichtenstern half of the cases occur between the first and tenth years. In 593 cases, 131 were in children under one year, and 80 were in children between four and six months old. The male sex is more predisposed than the female sex. Hillier gives the proportion of 7:5; Pilz, 3:2.

Pathological Anatomy.—The mesentery of the invaginated intestine is pulled into the space between the inner and outer cylinders. This causes an impediment to the flow of venous blood and hence a marked congestion develops in the intussusception. As traction on the mesentery is exerted the invaginated portion becomes bent so that the concavity looks towards the root of the mesentery and in this way the lumen of the bowel may be reduced to a mere slit.

The necrosis and gangrene of the invaginated intestine, which are caused by the disturbances of circulation, are of great importance. If the necrosis is confined to the beginning or neck of the invagination, a circumscribed inflammation of the adjoining parts ensues which causes an agglutination of the neck with the outer cylinder, and then the invaginated portion may separate. If there are no adhesions between the two inner layers of the invagination, either the inner or

outer of these two layers may be separated and eliminated, and hence the outside of this separated portion may be either serous or mucous. Fig. 19 illustrates this. The length of the eliminated intestine may vary from an inch to several yards. This course is most frequent in acute cases. In 125 cases Leichtenstern found that it developed in 94 before the fourth week. In chronic cases the necrosis is slow and

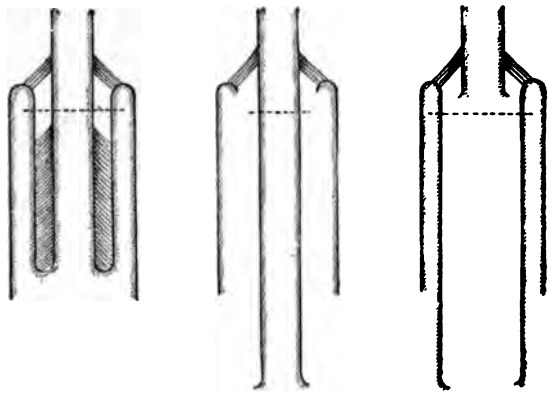

FIG. 19.—Intussusception, showing the Three Ways in which the Invaginated Portion may Slough Away. *A*, The inner and middle cylinders are joined by adhesions and, when separated at the point indicated, are cast off as a single tube with a mucous layer outside and inside; *B*, there are no adhesions between the two cylinders; the middle tube is separated first and is then unfolded, the cast-off tube, when final separation occurs, having an outer serous and an inner mucous coat; *C*, there are adhesions, as in *B*, only at the neck of the intussusception: the inner tube first separates and unfolds, and when the middle cylinder finally sloughs the cast-off portion is a tube with outer mucous and inner serous coat. (After Treves.)

gradual and the elimination of the invaginated portion takes place piecemeal.

The sheath or outer cylinder seldom shows marked changes, but it may be infiltrated or thickened. Ulcerations of the mucous membrane, which may cause gangrene or perforations of the intestinal wall, are rare. Such perforations, which may also occur in the intestine above the intussusception, are more frequent in chronic cases. Refinesque found twelve in twenty-five cases; Leichtenstern twenty-eight in one hundred and seventy-five. Occasionally the intussusception itself may protrude through one of these perforations of the outer cylinder. Treves gives an instructive illustration of this. In cases of chronic invagination peritonitis develops almost regularly. It is frequent also in acute cases (according to Treves in more than fifty per cent.). It begins usually on the second or third day and varies in extent and intensity. Sometimes only loose adhesions are formed, sometimes the adhesions are firm. In some cases a limited portion

only (usually the neck) of the intussusception is involved, but in others the whole mass is the seat of peritonitis, and then a complete union of the inner and middle cylinders takes place and the peritonitis spreads to the adjacent portions of intestine. If the separation and elimination of the necrotic intussusceptum occurs before this adhesive peritonitis causes a union at the neck of the invagination, then a fatal general peritonitis results. Finally we must mention, as a complication, intestinal stricture and stenosis, which may develop as a consequence of the ulceration, especially at the neck of the invagination.

Symptoms and Course.—Intussusception may be acute or chronic. The former may be divided into peracute cases, with death in the first twenty-four hours, acute cases lasting a week, and subacute cases lasting three to four weeks. The peracute cases are very rare and occur only in children, as a rule. In Leichtenstern's statistics of 270 cases there are only 5 peracute cases, and of these 4 were in children. Next in frequency come the acute cases—48 per cent.; then the subacute cases—34 per cent.; and then the chronic invaginations—18 per cent. (Treves).

Intense colicky pain occurring suddenly, often in the midst of seemingly perfect health, at other times preceded by symptoms of disturbed intestinal digestion, constipation, diarrhœa, flatulence and pains in the abdomen, may usher in the disease. The seat of the pain is not characteristic. It is usually located in, or referred to the region of the umbilicus, even if the invagination is situated in some other part. Vomiting and, in children, convulsions appear. These symptoms may soon cease only to reappear in a short time. Thus periods of comparative quiet and comfort alternate with paroxysms of pain, which are most intense in ileocolic intussusception. The pain in invagination is caused by the inflammatory swelling of the invaginated intestine and increased peristalsis. If, therefore, the pain ceases, it means that the invagination has become loosened. On the other hand the conclusion that the continuation or repetition of the pain means that the invagination is progressing does not necessarily follow.

In the vast majority of these cases of intestinal obstruction the symptoms of obstruction appear early. As we shall see presently, the invagination soon causes a more or less complete occlusion. But other factors may operate to bring about complete occlusion besides the invagination itself. Firstly the previously mentioned narrowing caused by the traction of the mesentery which produces a flexion in the intestinal lumen; this, together with swelling and infiltration of the intestinal wall, may cause complete occlusion. Again, hard, undigested masses, fæcal or biliary calculi, more frequently polypi or other tumors

which at first caused the intussusception, may be instrumental in completing the obstruction, and gangrenous portions of the invaginated intestine may lodge in the constricted lumen.

Two symptoms are especially characteristic—namely, the passage of portions of the intestine from the rectum, and the intestinal tumor. The discharge from the rectum is often rich in mucus and blood, but sometimes only blood is present in the stools. This results from the intense catarrhal inflammation and venous congestion of the affected intestine. The discharge varies according to the stage of the intestinal obstruction and according to the degree of obstruction. If the invagination is high in the intestine, even when there is nearly complete occlusion, the fæces of the lower portions of intestine are passed in the beginning. Thus the discharge is at first fæcal, but when complete obstruction is established the discharge quickly becomes bloody and contains mucus, as in dysentery. The resemblance becomes greater when, especially in deep-seated invagination, a tormenting tenesmus is present. But as there is sometimes fæcal matter in the discharge in dysentery, and as this never occurs in complete intestinal obstruction caused by invagination, the two are generally not to be confounded. But if the occlusion is incomplete there may be occasional passages of fæcal matter in the form of diarrhœa mixed with mucus and blood, and these passages may occur as frequently as ten to twenty times a day. Here also a certain periodicity in the symptoms may be noted. The discharge of gangrenous fragments of intestine is marked by an offensive, putrid odor, but this is not the case if a long piece of intestine is passed *in toto*.

In many cases a tumor may be felt at the site of the invagination, especially in children. It is cylindrical, sausage-shaped, often distinctly curved, smooth, and of firm elastic consistence. But this resistance—and in this I can confirm the statement of Nothnagel—is variable, depending on the varying conditions of contraction of the intestinal wall. The tumor may be altogether absent if there be no spasm. The location of this tumor is as unreliable an index of the actual seat of the intussusception as is the location of the pain. In cases of ileocæcal invagination, for example, the tumor is usually felt on the left side of the abdomen just above the umbilicus. Only when the colon is the seat of invagination the site of the tumor corresponds to the position of the lesion. Here the tumor is situated in the left lower abdomen. I once, however, saw a case of colic invagination in which the tumor lay across the umbilicus. If the intestine is not fixed a certain locomotion of the tumor may occur. In some cases of invagination of long standing the mass may prolapse and may then be felt through the anus. Cases have been reported in which an ileocæcal

invagination passed down through the colon and appeared together with the appendix in the lower part of the rectum. If the tumor disappears it may be because the invagination has been spontaneously reduced, because the intussusception has separated, or because the entire mass has descended into the pelvis and been covered with coils of small intestine. The subsequent course of the disease shows which of these events has taken place.

In acute cases the symptoms of obstruction are the most prominent, or the patients die of general peritonitis, of gangrenous destruction of the intestinal wall, or of septico-pyæmia, and the consequences of the same. The mortality is 70 per cent.; in children 80 per cent. The mortality of the different varieties is, according to Leichtenstern: ileocæcal, 71 per cent; enteric, 58 per cent.; colic, 70.9 per cent.

A spontaneous cure of invagination may occur (1) by spontaneous reduction, (2) by elimination of the invaginated intestine, or (3) by formation of a fæcal fistula.

Occasionally an invagination can reduce itself. This occurs in the above-mentioned physiological cases. In pathological invaginations this is as a rule impossible, partly on account of the quickly formed adhesions and bands, and partly on account of the excessive swelling of the invaginated portion of the bowel, especially at its extremity; partly also on account of the twisting of the invaginated portion round its axis, or, in ileocolic invagination, on account of the ileocæcal valve which incarcerates the intestine. Treves says that the earliest appearance of adhesions is on the third day. I have seen them well developed on the second day. They occur in eighty per cent. of chronic cases; in acute cases—less often in, say, about half of the entire number. A spontaneous reduction occurs but rarely only in quite recent cases, and usually only in enteric invagination. Cases in which a spontaneous reduction has taken place have a tendency to recur. Senator reports a case in which nine relapses occurred in seventeen days.

Nothnagel reports a case of his own of an intussusception in which a spontaneous reduction had taken place, which recurred after a year and a half and then required operation. He found an ileocæcal invagination at the extremity of which was an intestinal polypus. Strange to relate, there was not the slightest indication of peritonitis. Such cases are difficult to diagnosticate, as it is necessary to differentiate between invagination and fæcal tumor.

The formation of a fæcal fistula is very rare. I know of only one instance; it was the case of a man sixty-seven years of age who passed fæces with the urine during three or four months before death. At the autopsy we found a short intussusception of the colon into the

sigmoid flexure with a fistula which opened into the upper part of the bladder.

Spontaneous elimination of the invaginated intestine occurs in about 42 per cent. of all cases. More frequently with women (54 per cent.) than with men (31 per cent.). It occurs in ileocæcal invagination in 20 per cent., in colic invagination in 28 per cent., and in enteric invagination in 61 per cent. It is very rare in children, as they usually die before this can occur. We find only 2 per cent. of all the cases during the first year of life.

Spontaneous elimination of the intestine does not always mean the cure of the patient. More than forty per cent. die from the direct consequences of the intestinal lesion. We have mentioned the different varieties above. There may be a perforative peritonitis caused by the incomplete union of the intestinal cylinders at the time of sloughing, an intestinal rupture by ulceration, or an occlusion of the intestinal lumen by the portion of intestine which is eliminated, or finally death may result from pyæmia or hemorrhage or chronic diarrhœa, which may be brought on by ulcerative processes of long standing. Also cicatricial formations may obstruct the lumen of the intestine after a spontaneous reduction.

Chronic intussusception is characterized by constipation alternating with diarrhœa and paroxysms of pain alternating with periods of comparative comfort. They may last months or even years. Pohl reports a case which lasted eleven years.

The *prognosis* is always grave. The more acute the onset and the younger the patient the more unfavorable is the prognosis.

The *diagnosis* is based on the following symptoms: Violent and usually sudden pain, vomiting, muco-serous discharge, tenesmus, symptoms of complete intestinal obstruction, appearance of a characteristic tumor. Of all diseases which cause intestinal obstruction acute invagination is the one which is relatively the easiest to diagnosticate, especially in children.

Much more difficult is the diagnosis of chronic invagination, especially if the history fails to reveal the presence of the characteristic symptoms. In these cases many diagnostic errors may be made, but they can sometimes be avoided if there is a perceptible and pronounced tumor.

Obturation of the Intestine.

Gall stones of ordinary size easily pass through the intestine after having passed the biliary ducts, but when of considerable size they may cause intestinal obstruction. This size is such that the lumen of the bowel is completely filled so that the intestinal wall fits closely to

he calculus, and does not allow of the passage of intestinal contents at his point. However, there have been cases (Israel reports one such) n which intestinal obstruction occurred, although the stone did not ompletely fill the lumen of the intestine, but was easily movable in he same. In such cases we may assume that the stone causes a re- lex contraction of the wall of the intestine and is thus firmly grasped. Such a contraction may after a time relax and then the symptoms of ntestinal obstruction will disappear spontaneously.

In order to occlude the intestine the calculus must have a diameter of at least 3 cm. (1⅛ inches). As a rule the calculi do not increase in size by a deposit of salts after they have entered the intestine, so we must believe that they gain entrance into the intestine through a fis- ulous opening between the gall-bladder and the duodenum, excep- ionally between the gall-bladder and the colon. This is the only ex- planation that is plausible, as stones of this diameter could not pass through the common bile duct. A calculus has been observed pro- truding half-way into the gall-bladder and half-way into the lumen of the intestine—like a button. This piercing of the bladder and intes- tine is caused by pressure necrosis and previous inflammation of the part involved. In some cases, after the stone has found its way into the intestine, it may pass through without causing any marked symp- toms, but in other cases symptoms of colic occur.

Occasionally the history of the case may reveal the existence of previous hepatic disorders. I once saw a patient, a woman of fifty- two years, in whom no cause for the symptoms of intestinal obstruc- tion could be found, but the history showed that she had been in Carlsbad several years previously for the cure of a then doubtful biliary colic. At the laparotomy I soon found the seat of obstruction and was able to extract a calculus of the size of a walnut.

These calculi may be arrested at various places but usually in the lowest part of the ileum, that is, at the ileocæcal valve. Then symp- toms of obstruction in the upper portion of the small intestine appear; abundant biliary vomiting, which later becomes stercoraceous, active peristalsis, and pain. In spite of the vomiting of fæces there may be passages of gas or fæces, or symptoms of mild ileus may alternate with diarrhœa. This can occur only when the stone does not entirely fill the intestinal lumen. It is also possible when the occlusion is only temporary and is caused by contraction of the intestinal wall or when portions of food or fæces get wedged between the stone and intestinal wall or above the stone. Occasionally the stone or the distended con- volution above it can be felt. Only seldom does the pain denote the seat of obstruction. The pains are severe, perhaps intermittent, localized or diffuse. They depend not only upon the cramp-like peri-

stalsis above the occlusion, as in other forms of intestinal obstruction, but also upon ulcerations of the mucous membrane caused by pressure of the stone on the intestinal wall. These ulcerations may extend through the mucosa to the muscularis and the serosa and may cause perforation or adhesive peritonitis. They may cause a secretion of mucus and an extravasation of blood which is found in the stool. If the stone is not dislodged either spontaneously or by operation the patient dies in collapse.

The chances of a spontaneous cure, however, are fairly good. It occurs in about 50 per cent. of all cases. According to Courvoisier it occurred 56 times in 125 cases. According to Schüler and Naunyn 44 times in 82 cases. If the calculus becomes dislodged and passes, the general condition of the patient improves at once. The average duration of the symptoms in such cases is from two to ten days. I can recall cases in which the symptoms continued fourteen days and even three weeks, and yet recovery took place. If the stone becomes incarcerated first in the upper portion of intestine and later in a lower segment, then there occur repeated attacks of obstruction; but after the stone has been finally dislodged and passed into the large intestine it may remain there for days or weeks without causing any symptom of obstruction. The stone need not be discharged immediately after the obstruction disappears. Even large stones may be passed without symptoms of obstruction if they escape from the ductus choledochus or bladder directly into the colon.

The *diagnosis* is based on the history of the case which points to preëxisting symptoms of gall-stone colic, or the stone itself may in some cases be felt. The condition is most frequently met with in old people—oftener in women than in men. The symptoms are those of obstruction situated in the upper intestine, abundant biliary vomiting, ileus of varying intensity, moderate tympanites, blood in the stools, and the presence of a stone, which can be detected on palpation. This last-named sign is, however, usually not to be obtained on account of the extreme distention of the abdomen, and then a positive diagnosis cannot be made, for it can be based only on supposition.

Obstruction by enteroliths is very rare. I have in my long and extensive experience never seen a genuine enterolith, which is to be distinguished from fæcal masses. It consists of a mixture of inspissated organic substances with minerals such as calcium and magnesium phosphate, calcium sulphate, and salts of ammonia. The stones have usually an oval or cylindrical or irregular form with a slightly rough surface. A concentric stratification around a nucleus is noticeable. The nucleus is as a rule a small foreign body, such as a splinter of bone, a fruit seed, a needle, etc. Calculi have been found of enormous

size, 25 cm. (10 ins.) in circumference, and weighing up to 2 kgm. (4½ lbs.). They may be formed by the long-continued medicinal use of mineral substances such as calcium carbonate, calcined magnesia, or magnesium carbonate. The obstruction caused by them is usually chronic, and but rarely acute.

Compression of the Intestine.

Generally speaking, every intestinal incarceration may be regarded as an intestinal compression. Usually, however, we denote by this term a pressure which another organ, such as a displaced uterus, a dislocated spleen, or a floating kidney, or any tumor of the abdominal or pelvic organs other than the intestine, exerts upon the bowel.

Occasionally the cause of intestinal obstruction may be very difficult to recognize. In most cases, however, knowledge of the existence of a floating or dislocated organ will lead to the correct diagnosis.

Circumscribed Intestinal Paralysis.

There are many cases of intestinal obstruction in which, not only during life, but also at the autopsy, no cause for the occlusion can be found. It is supposed that in these cases the intestinal obstruction is due to paralysis of the intestinal muscles, and they are therefore called "paralytic ileus." Henrotin and Rosenbach have made extensive researches in this direction.

Such paralyses may be caused by traumatic and inflammatory processes or may be of nervous origin. To the first class belong cases in which the intestine has been subjected to an external traumatic influence. In the other cases the obstructive symptoms remain after the original cause of the obstruction, for example a strangulated hernia, has been removed by operation.

Other cases have been known in which an acute peritonitis or, more rarely, embolism of the mesenteric artery, has caused intestinal paralysis.

By nervous paralyses, we mean those which occur in hysterical patients or which are the result of operative procedures or of inflammatory processes.

Symptoms of acute intestinal obstruction have been observed in cases of inflamed hydrocele, contusions of the testicle, operations on hemorrhoids, inflammations and abscesses in the inguinal region, and, according to Nothnagel, after tapping the abdomen in ascites.

In some cases the cause of the paralysis is not easily discovered. In a large number of cases it is caused by reflex irritation of the in-

the stomach become feculent, and how rapidly the organ refills after lavage. One can repeat the lavage several times a day and each time find an astonishing amount of fluid. By lavage of the stomach the violence of the symptoms and the sufferings of the patient are much diminished, and thus the patient can better endure the operation.

Puncture of a distended convolution has been frequently recommended. I have performed it repeatedly without much success. A very markedly distended convolution is, under aseptic precautions, punctured with a hypodermic needle which is connected with a rubber tube filled with a solution of salicylic acid. In favorable cases abundant gas escapes, but in other cases the result is very unsatisfactory as the point of the needle becomes clogged. We may endeavor to restore the passage through the needle by pressure on the tube, and if we do not succeed another puncture must be made. Even when I succeeded in obtaining large amounts of gas I could see no beneficial effect on the symptoms. Other physicians, however, among them Curschmann, of Leipsic, report more favorable results. Another strong objection to this procedure is that in cases of intestinal paralysis the puncture made by the needle may allow the contents of the intestine to pass into the abdominal cavity and thus cause a local or general peritonitis.

In a few cases of paralytic ileus caused by gall-stones and foreign bodies the use of massage and electricity has yielded very gratifying results. As a rule, however, such attempts are from the very outset useless.

We may mention here the administration of metallic mercury, formerly much in vogue. It has been given up altogether after it was found that it did much harm by its weight in cases of strangulation, twisting, invagination, etc. In some cases it even caused a rupture of the intestine and was found free in the abdominal cavity. It is, however, recommended by some authors as a last resort in cases of persistent constipation. It is said to become finely divided in the fæces and to soften the same.

It is very rarely that we have to give any dietary prescription, as the patients are usually too exhausted to desire food. If it should be required, as in chronic cases, mild and unirritating articles of food should be given, such as milk, beef tea with peptone, or Mellin's food. There is a possibility that the food will not be absorbed but vomited instead. Thirst is a very tormenting symptom. For some time or at least in the beginning it is possible to quench the thirst by frequent rinsing of the mouth with water, or by swallowing pellets of ice or small amounts of water. It is advisable to inject water into the intestine. The subcutaneous injection of a physiological salt solution

(0.6 per cent.) in cases in which the blood is very deficient in water has been advocated, but according to my experience the effect is slight and transitory. I allow my patients to drink as much water as they please and I frequently add lemon juice, claret, or cognac. Of course I try to prevent large accumulations by frequently washing out the stomach. This treatment diminishes somewhat the unsupportable and tormenting thirst.

The question of operative interference in the treatment of ileus is one that is difficult for the physician to decide. In every case of ileus he must decide whether it is necessary or not to call in a surgeon. If the circumstances were such that we could in every case determine the site and nature of an intestinal obstruction the problem would be much simplified. But experience has shown that even after the operation has been successfully performed the patients sometimes die. We have stated above how difficult or impossible it is to differentiate the various forms of ileus, and even on the operating table it is often impossible to find the seat of obstruction without long and injurious manipulation.

There are few diseases which so diminish the power of resistance of the patient against abdominal operations as ileus. Even the exposure of a considerable length of intestine may bring on rapid and fatal collapse (Schede).

In general nearly every physician admits that in cases of strangulation in which the site can be approximately determined it is better to entrust the patient to the surgeon as soon as possible. In other varieties of ileus it is recommended to observe the patient for a while and to be guided by the general symptoms, pulse, temperature, etc. The prognosis of the operation is now more favorable than formerly, as at present the surgeon usually makes a primary enterostomy or colotomy, and a secondary operation later when the condition of the patient is better, owing to the discharge of intestinal contents through the primary incision and the relaxation of the intestines.

Naunyn has done much in the way of establishing the indications for operation, and his statistics, based upon his own experience, are very valuable and instructive.[9] The importance of the subject induces me to give his conclusions, as they coincide with my own. An early operation on the first, or at least on the second day, is indicated in cases of incarcerated hernia and they ought to be referred to the surgeon at once. The same is true in cases of ileus in which there is a history of abdominal disease or in which there has been a hernia relieved by operation or by taxis. According to Naunyn the results obtained in early operation (seventy-two per cent. cures) are much more favorable than when the operation is postponed until the third

or fourth day. Likewise an early operation is indicated in cases of ileus in which the physician suspects an internal obstruction by angular flexion, or twists, or constricting bands, or internal hernia.

The following symptoms indicate immediate operative interference: Sudden appearance of ileus in the midst of perfect health, violent pain depending upon distention of the peritoneum, early vomiting, also stercoraceous vomiting (obstruction in the small intestine), and very profuse vomiting. The more violent these symptoms are, the sooner they appear, the less they are influenced by opium, and the more pronounced is the initial shock the greater is the danger of delay. The operation ought to be performed at once and then the outlook is favorable. There is little hope that in cases of incarceration with complete obstruction a reduction can occur without operation, and there is great danger of intestinal paralysis and peritonitis. Even if profound collapse (incarceration shock) is present at the beginning, it is no contraindication to the operation. Sometimes the patients do not recover from this initial shock (Schede) and so waiting would only diminish the chances of success. Without operation the patients rapidly decline, whereas after the incarceration has been liberated the symptoms of collapse soon disappear. Sometimes even during the operation the site and nature of obstruction are hard to determine, and in cases in which the obstruction cannot be removed an artificial anus should be made; then after the patient has regained sufficient strength a secondary operation should be performed if the obstruction still persists. If the diagnosis of intussusception is made, which as a rule is not difficult (dysenteric, fœtid, hemorrhagic, or serous discharges), the case ought to be transferred to the surgeon, and it should be left to him to decide whether or when it is advisable to operate. In the case of small children early operation is advisable if medicinal treatment fails, as the course of the disease is very rapid, and according to the experience of H. Braun laparotomy gives better results when the intussusception can be replaced than when resection is necessary.

In the case of adults the operation need not be performed so early, as the disease is more benign in the beginning, and at times the intestine again becomes permeable and the intussusception may after some time, eighteen days (Schramm) even six months (Czerny), be reducible. However, in view of the dangers which threaten the patient in chronic stenosis of the intestine, in most of these cases surgical interference seems desirable.

In volvulus of the sigmoid flexure each case must be considered by itself and no general rule regarding operation holds. However, if there suddenly appear evidences of complete twisting (360°), as shown by

severe symptoms of strangulation, such as intense pain near the umbilicus, considerable and rapidly increasing tympanites, and tenderness on pressure in the left iliac fossa, then immediate operation is indicated. If the disease takes a milder course (half volvulus) and the intestine again becomes permeable, the further observation of the case will determine whether the cure is permanent or whether a recurrence takes place. If the symptoms show that the volvulus persists then an operation is indicated.

Until recently the results of operation in cases of *obstruction by gall-stone* have been unsatisfactory and so discretion must be observed. Each case must be decided on its own merits. If it can be shown that the stone moves gradually down the small intestine, and if it causes no injury to the intestinal wall (ulcers, hemorrhages, peritoneal irritation), the operation can be postponed as the prognosis for spontaneous cure is not very unfavorable. But if the stone remains for a long time in the same place (impaction), if the symptoms of ileus are very severe, if there is violent pain at the point where the stone is arrested, and if blood appears in the discharge, an operation may eventually become necessary.

As a rule, *foreign bodies* become arrested in the intestine only when the lumen is narrowed at some place. It must be determined whether the foreign body which has been swallowed has a sharp, pointed edge, and if this be the case an operation should be performed in order to prevent wounding of the intestinal wall. If one does not know the size and shape of the foreign body, the indications for surgical interference are the same as in cases of chronic stenosis, the treatment of which has already been discussed.

HEMORRHOIDS.

There is hardly any other affection of the intestines which has in so great a measure attracted the attention of the layman as well as of the physician for centuries as hemorrhoids (phlebectasia hæmorrhoidalis) and the hemorrhage resulting therefrom. The humoral pathology, according to which most diseases were derived from a deterioration or contamination of the blood, recognized logically in hemorrhoids a localization of the hemorrhoidal virus, and looked upon hemorrhage from these tumors as a favorable occurrence, that is, as a discharge of this virus.

Bleeding of the piles was considered to be an effort of nature to rid itself of noxious material, and for this reason physicians dared not check the hemorrhage or remove the hemorrhoidal tumors, for fear of driving the noxious material to the internal organs; the dis-

turbance resulting from this was called dispersed or displaced hemorrhoids. In recent times this view has been abandoned and the development of hemorrhoidal tumors is referred to purely mechanical causes. It has been recognized that the appearance of hemorrhoids is the expression of circulatory disturbances, which can never be of any advantage, but quite frequently injurious to the patient. As, however, preconceived opinions among the great mass of people are tenaciously adhered to, hemorrhoids play even to-day a much greater rôle than they are often justly entitled to.

Under the term hemorrhoids we understand varicose dilatations of the hemorrhoidal veins either in the external part of the anus or in the submucous tissue of the lower portion of the intestine. The view held by some writers, for example by Damaschino, that a purely functional disturbance and a secretion from the rectal vessels exist, is probably based on a confusion of proctitis and ulcerative processes of the intestinal mucous membrane with hemorrhoids.

A distinction is made between *external hemorrhoids*, situated outside of the sphincter externus, and arising from the subcutaneous connective tissue of the anus, *internal hemorrhoids*, which are situated above the sphincter internus, that is to say, which arise from the submucous tissue of the mucous membrane, and which may occasionally be found as high up as the sigmoid flexure (F. L. Petit), and *mixed hemorrhoids*, which are situated in the cutaneous area, as well as in the area of the mucous membrane. The size of hemorrhoidal tumors varies from that of a pea to that of a pigeon's egg, and may change very much in the same patient, according to their condition of fulness.

External piles are seen as bluish or dark bluish-red tumors, which usually become markedly reduced in size after a movement from the bowels. At first they are covered by normal skin, which is movable over them; later, however, generally as a result of inflammation, the cutis becomes adherent to the varix nodule. This causes profuse exudation and irritation of the surrounding tissue, by which extensive inflammatory processes, infiltrations, and swellings take place in the tissue surrounding the veins, which finally may lead to the formation of large, firm, cavernous tumors.

The cutis may also gradually become so thinned by continuous pressure that the tumors may be ruptured by straining at stool, with somewhat stronger pressure, and thus the danger of a profuse hemorrhage may arise. Larger cavernous tumors attaining the size of a small apple may be formed by the blending of smaller ones and may be enclosed in a firm capsule as a result of connective-tissue hyperplasia. Should the hemorrhoids, by reason of a tardy circulation and an inflammatory process in the walls of the veins, retrograde,

and this be accompanied by solidification of their contents, the latter may calcify and thus produce a venous concretion. Otherwise only a tag of skin resembling a condyloma somewhat, but rather more relaxed, bloodless, and of a brownish color, will remain in the outer vicinity of the anus.

Internal hemorrhoids are sometimes sessile, sometimes pedicled, and are as a general rule more vascular than external piles. Occasionally they are in communication with capillary or even arterial vessels, and Allingham has for this reason divided internal hemorrhoids into three groups: (*a*) *Capillary*, which consist of small, spongy tumors, with a soft, dark-red, and easily bleeding surface; this form is frequently the cause of periodical loss of blood during defecation; (*b*) *arterial*, which are larger and harder tumors, frequently pulsating distinctly, having a smooth, shining surface; as a consequence of their injury, larger hemorrhages in which bright red blood is lost, may occur; (*c*) *venous*, which occasionally are very large, rather firm, and have a pale bluish-red color. These are apt to protrude, and when they bleed, the blood is of the dark-colored venous variety.

On account of the pressure exerted by these tumors on the mucous membrane covering them, the latter gradually becomes ulcerated, so that any slight dragging, for example from the passage of the fæces or from increased tenesmus during defecation, will occasion a more or less profuse hemorrhage.

Of especial significance is a *prolapse of the hemorrhoidal tumors*, which is particularly liable to happen in the pedunculated variety. In this manner large tumors may protrude from the anus, which become of a dark, bluish-red color in consequence of strangulation, and are very vascular. If these tumors do not retreat of their own accord, or if they are not replaced, they may become inflamed or even gangrenous.

We may, however, meet in the rectum itself with inflammation of the walls of the veins, that is, phlebitic and periphlebitic processes, which will lead either to coagulation of the blood and a wasting of the tumor, as already mentioned above, or to hemorrhoidal ulcerations, or even, if the process becomes extensive, to periproctitic abscesses, and complete or incomplete fistulæ and fissures.

The mucous membrane of the rectum in internal hemorrhoids is nearly always in a condition of chronic catarrh. The tissues are spongy, very vascular, and the surface is covered either with a thick coating of mucus or with pus and blood.

Etiology.

Hemorrhoids are caused in the first instance by mechanical influences. Even under normal circumstances the conditions for a frequent recurrence of congestion in the hemorrhoidal veins are very favorable. In the first place the latter are in a dependent situation in any position of the body. Secondly, they are compressed by the contraction of the muscles present in the vicinity of the lower portion of the rectum, and by the fæcal masses entering into the rectum, in such a manner that the circulation is temporarily rendered difficult or altogether obstructed. As the hemorrhoidal veins are void of valves, the blood which has just passed through them is forced back again. These circumstances produce, even in the normal condition, a certain tendency of this venous district to ectasia, the more so as the veins in the surrounding cellular tissue are very loosely embedded, and therefore yield easily. The following factors bearing on the formation of hemorrhoids, and which are denominated by the French as mechanical and constitutional causes, are therefore to be taken into consideration. First among the causes are a sedentary occupation, and such a mode of life and dietary as must lead to a continued hyperæmia of the lower portion of the rectum. In this way hemorrhoids are brought on in clerks, students, some artisans, as for example shoemakers and tailors, in persons who do much standing on their feet or horseback riding, and lastly in those who by overeating or eating much highly seasoned or fatty food produce periodically a great fulness of the portal circulation.

Another cause is chronic constipation or inflammation of the large intestine or of the rectum; in which case the detrimental factors alluded to above assert themselves.

The same may be said of cicatricial and carcinomatous strictures of the lower portion of the intestine. Temporary causes of congestion in the hemorrhoidal veins may also be found in the pressure exerted by the pregnant uterus, and chronic hemorrhoids may easily be developed from this cause.

As distant causes may be mentioned diseases of the uterus and its adnexa, of the ovaries, the bladder, the prostate gland, and other pelvic diseases.

Those chronic diseases, within the area of the radicles of the portal vein and the liver, which may lead to a continued stasis of the blood in the portal vein are especially important. In this connection cirrhosis of the liver holds the first place. Indirectly, all diseases of the respiratory and circulatory apparatus may become the causes

of hemorrhoids, by reason of which they occasion stasis in the vena cava.

Whether excesses *in venere et baccho*, as is oftentimes claimed, may in reality produce hemorrhoids is more than doubtful. This might with more truth be said of the abuse of drastic cathartics, which may perhaps give rise to hemorrhoids by causing a congested condition of the mucous membrane.

All of these essential factors may be divided according to their nature into temporary and permanent causes, and their results, that is the hemorrhoids, will be more or less amenable to treatment accordingly.

Hemorrhoids are usually developed between the ages of thirty and fifty. If they are met with in children or even in the new-born (such examples have been recorded by Klein and Lannelongue), they are here certainly only abnormal phenomena for which no reason whatever can be assigned. It is said that hemorrhoids are more frequently met with in men than in women, although in the latter the extraordinarily frequent occurrence of constipation as well as pregnancy and the numerous uterine affections seem to be sufficient cause for the production of hemorrhoids. According to my own personal observation, the latter do not occur less frequently in women than in men.

In some peoples and races, for example in Russian and Polish Jews and in those of the Oriental races, hemorrhoids are very frequently met with. This may be referred to a stronger influence of those noxious factors which have already been mentioned above, especially to an irregular way of living. Whether climate exerts a direct influence here, as has been claimed, is doubtful.

Hereditary influence is recognized by some authorities, but denied by others. The same may be said of the importance of certain diatheses, as, for example, gout, rheumatism, and the herpetic diathesis of the French. A positive decision as to cause and effect in these cases can, according to my judgment, only be reached with difficulty, owing to the great frequency with which hemorrhoids occur.

Symptoms.

Hemorrhoids may present no symptoms at all or only slight ones, or they may be the cause of severe local and general symptoms, although the latter occur only in a small proportion of the cases. As a rule the complaints are varied, and change according to the number, size, and distention of the varicose dilatations. The clinical picture may also differ according to the etiology and the complicating processes. The hemorrhoidal tumors may be moderately distended at

times and are then soft, not very prominent, and wrinkled on their surface, or they may be congested, full, hard, and painful. These two conditions may be present alternately.

Among the first symptoms are evidences of irritation, such as itching, burning, stabbing sensations, or irregular throbbings about the anus, and the sensation as of a foreign body in the latter, or a continued feeling of heat. Stronger paroxysms of pain of a spasmodic or tearing character may also occur, particularly after some hygienic or dietetic error; these attacks are called by Romberg "hemorrhoidal colics," and are looked upon by him as a neuralgia of the sympathetic. In some cases, as a result of these continued sensations of irritation, nervous phenomena of a general character may appear. A great many victims to hemorrhoidal disease complain of anorexia, sluggishness of the bowels, dulness of the intellect, ringing in the ears, vertigo, indistinctness of vision, disinclination to exertion on the one hand, and easy excitability on the other, and a hypochondriac disposition. In some cases palpitation, irregular heart action, precordial pains, and subjective dyspnœa are added. Defecation is always more or less painful, particularly in the case of internal hemorrhoids, and when the fæces are firm, the pain may be so severe as to cause fainting and convulsions. Many patients, through fear of the pain, defer defecation as long as possible, forgetting that the affection is thereby only made worse. External hemorrhoidal tumors are not infrequently complicated with moist excoriations which cause violent and distressing itching. When these tumors become much inflamed or go on to suppuration, which is usually due to a phlebitis occurring in the deeper tissues, they become greatly swollen, of a dark-red to a bluish-red color, hot, and exceedingly painful, so that the patient is very careful to avoid any contact with them and remains lying on his side or back.

The most violent symptoms, however, are caused by the prolapse outside of the anus of an internal hemorrhoidal tumor, which becomes strangulated by a spasm of the sphincter muscles.

The finding of pedunculated internal hemorrhoids or the internal tumors of mixed hemorrhoids outside of the anus after straining, or, when the sphincter muscles are in a weakened condition, after coughing, sneezing, or even standing, is not a rare occurrence. In this case the tumors usually return spontaneously or are easily replaced. When, however, they remain outside for some time, violent and sometimes most excruciating pain is experienced. This is due to an increased irritability of the sphincter muscle resulting from a periproctitis or some other inflammatory process. The patient is unable either to stand or to sit, but lies on his side with the knees drawn up, anxiously

avoiding the slightest movement, even the drawing of a deep inspiration, sneezing, or coughing. Increased frequency of the pulse with febrile movement, even chills, singultus, nausea and vomiting, and dyspnœa may occur. The prolapsed piles are of a bluish-red color, very tense and tender on pressure, and if reposition is not possible, or surgical aid is not quickly procured, gangrene with its sequelæ, in which sepsis, periproctitis, peritonitis, etc., assume a prominent rôle, will rapidly occur.

Hemorrhage is of frequent occurrence, but is not invariable, neither does it occur with any regularity, and many patients go for months or even years without a hemorrhage. The amount of blood lost may vary greatly, the hemorrhage being sometimes very slight, sometimes dangerously profuse. Many patients experience decided relief after a hemorrhage, as regards the local symptoms, the congestion of the brain, and any nervous symptoms which may be present. Generally the hemorrhage is at first small, and becomes more copious only after a few days, when it may again cease. In some cases the hemorrhage makes its appearance only in conjunction with defecation. Usually the blood is of a dark venous color; should the hemorrhage, however, be from an internal arterial tumor, it may be of a bright red color. It is always found on the surface of the fæces. Sometimes a greater or lesser quantity of pure blood, or a thin fluid tinged with blood, passes off only after the expulsion of the fæcal matter, in which case we usually have to deal with a capillary hemorrhage. Under certain circumstances the hemorrhage may occur so frequently and be so profuse as to give rise to a high degree of anæmia and resultant debility. In the greater number of cases such anæmias are readily referred to their true source. When, however, we have to deal with internal hemorrhoids, and the hemorrhages are not very abundant, but only dangerous to the patient by their frequent recurrences, then they may not be discovered either by the patients themselves or by the physician. I have myself been consulted twice by patients who had been treated with all kinds of roborant and iron preparations, and who were promptly cured after I had discovered the real cause of the anæmia and instigated surgical treatment, namely, cauterization of the bleeding varices.

Diagnosis.

The presence of external hemorrhoids, which are discoverable to the naked eye, is easily diagnosed, and a mistake between them and broad condylomata or hypertrophied skin tags is hardly possible with the exercise of ordinary care, particularly when other signs of a possible syphilis are present.

When, however, we have to deal with polypi of the mucous membrane of the rectum, which may eventually also become prolapsed, the diagnosis will be somewhat more difficult. The appearance of these polypoid tumors, however, will usually enable us to guard against a mistake; if necessary they may be punctured by a hypodermic needle, by means of which some blood would be withdrawn in the case of hemorrhoids, but not so if a polypoid tumor was punctured. To this may be added the fact that polypi are more frequently met with in children and young persons, in whom hemorrhoids appear with extraordinary rarity. As a rule, internal hemorrhoidal tumors may be brought into view when the patient bears down strongly and at the same time the nates are pressed apart with the hands. If this does not succeed, the mucous membrane should be inspected as high up as possible by means of a speculum.

Deeply seated carcinomata of the intestine, and also tuberculous dysenteric ulcers may be mistaken for hemorrhoidal tumors or ulcers. The former, however, are distinguished by their ragged, hard surface and other corresponding symptoms, such as suppuration and cachexia, and the ulcerous processes may easily be recognized in the same manner by their shallow loss of substance; we must be careful, however, not to be deceived by the occasional puffy bulging tags of mucous membrane. On the whole, hemorrhoidal tumors need hardly ever be confounded with these affections, and a mistake may always be avoided with some care.

Nevertheless, I have myself seen a case in which a surgeon repeatedly referred a hemorrhage from the intestine to hemorrhoids, and after dividing the external sphincter cauterized the intestinal mucous membrane. The case was really, however, one of dysentery, running a somewhat abnormal course, and the result was that the patient had an insufficiency of the sphincter added to his former affection.

Course and Prognosis.

Hemorrhoids, as a rule, exist for a long time, not infrequently throughout life, the cause of their presence being usually of a permanent nature. Life is seldom threatened by them unless the grave complications mentioned above supervene. In those cases in which the cause can be removed the hemorrhoids may again disappear. Generally their course is marked by intervals of remission and exacerbation.

Treatment.

The first thing when dealing with a case of hemorrhoids is to insist upon a rational way of living. Exercise in the open air, a

regulation of the diet, the avoidance of all kinds of excesses, attention to a daily stool, and the performing of light gymnastic exercises, which are adapted to antagonize hyperæmia and congestion of the abdominal vessels, must become our first endeavor.

The diet should be so regulated as to leave behind the least possible solid residue, and it must not contain irritating substances such as strong spices, very acid articles (pickles), strong alcoholic drinks, or very strong coffee and tea. All vegetables containing much cellulose and the leguminous vegetables are therefore as a whole to be avoided, and meat, lettuce and cabbage, fruit and preserves are to be preferred. The regulation of the bowels is of the greatest importance, and the reader is referred for the measures adapted to this end to the section on chronic constipation. Persons who incline to hemorrhoidal development or whose calling predisposes to the affection should carefully see to it that their passages are always of a soft and slightly mushy consistence. These persons should be advised not to sit on upholstered chairs or to lie in feather beds, but wooden-seated chairs and hair mattresses are to be preferred. After each stool the neighborhood of the anus should be sponged off with a weak solution of carbolic acid or lysol, and dried with a piece of lint. (This is a measure that ought to be practised even where no hemorrhoids exist.) When the tumors are protruding, or if there is a complicating rectal prolapse, reposition should be made slowly, after the prolapsed portion has been coated with borovaseline, cocoa butter, or oil, with the cleansed and oiled fingers; this is best and most easily done when the patient is stooping. The tumors may also be touched with a solution of iodine and potassium iodide (potassium iodide, 2.0; iodine, 0.2; glycerin, 40.0) by which they are made tough and more resisting, and smaller nodules may even be made to contract. At the same time, cool sitz baths and douches to the anus for the purpose of hardening these piles and preventing inflammatory processes are to be recommended.

An olive-pointed, double-current catheter may also be passed into the bowel. This procedure, however, is one which cannot be practised for any long period, and at the same time is very troublesome. The insertion from time to time by the patient himself of small pieces of ice into the rectum will be found much more feasible and successful.

For *symptoms of irritation*, especially for excoriations, the application of an ointment, composed of vaseline, lanolin, or cocoa butter, containing either morphine, extract of belladonna, or cocaine, will be found useful. The following is an example: Lanolin, 20.0; extract of opium 0.3, or extract of belladonna 0.3, or else suppositories with

extract of belladonna or extract of opium, 0.04; or the piles can be touched with a solution of cocaine two per cent. or one of nitrate of silver one per cent. by means of pledgets of absorbent cotton saturated with them, or the solution may be injected through a vaginal syringe. In case of inflammation, the application of ice water or an ice bag, or else moist heat in the form of poultices, hot baths, etc., is to be recommended. Symptoms of very severe inflammation may even necessitate the application of leeches. When the piles show extraordinarily severe symptoms of inflammation an incision or other surgical measures may become necessary. We may try to ward off the stage of congestion by administering mustard foot baths.

Hemorrhoidal ulcerations are best cauterized every second or third day with nitrate of silver until cicatrization has been obtained, and in the mean time they may be treated with zinc ointment or other salves.

Hemorrhoids which have prolapsed and are strangulated by spasm of the sphincter should be subjected to an effort at reposition. Under certain circumstances, some perseverance is necessary in exerting a steady but not too forcible pressure for a quarter of an hour or longer. If this method is not successful in returning the piles, ice applications, or perhaps hot compresses or steam, may be used; this should, however, not be continued too long, but if unsuccessful should be soon followed by reduction under chloroform narcosis. Any existing gangrene of the piles should be treated according to the rules of surgery.

As a *prophylactic* measure against the extension of hemorrhoids, and also against the spasm of the sphincter, we may occasionally obtain good results by the passage of rectal bougies; it is not possible, however, to cause a total disappearance of the piles by this means.

Hemorrhages are generally of no great importance and cease of their own accord. The application of cold in the form of cold sitz baths, or pieces of ice or ice water passed into the rectum, is usually sufficient. Should the hemorrhages return very frequently, injections of astringent solutions of tannic acid (1 to 2 per cent.), alum (1 to 3 per cent.), acetate of lead (0.2 to 0.5 per cent.), nitrate of silver (0.5 to 1 per cent. and more) are to be employed. External piles may be treated by chrysarobin ointment after Unna's formula (chrysarobin, 0.8; iodoform, 0.3; extract of belladonna, 0.6; vaseline, 15.0) several times a day; internal piles by a suppository of a similar composition. This treatment is said not only to stop the hemorrhages, if continued for a long time, but also to relieve the tenderness and reduce the size of the piles.

In cases of very violent hemorrhage, tamponing the rectum with pieces of cotton previously dipped in solution of the perchloride of iron may be resorted to. It is, however, better still to touch the pile with the galvanocautery. Internal remedies like ergotin, hamamelis virginica, or hydrastis canadensis, in the form of fluid extracts, are of little or no value.

Operative removal of the hemorrhoidal tumors is to be practised when the latter by their painfulness, by strangulation, by interfering with defecation, or by prolapse give the patient so much trouble that he wishes to be rid of them under any conditions. Another indication for operative interference is when the patient, on account of the intensity and frequency of the hemorrhages, becomes anæmic and debilitated. We must, however, remember that operative interference is not always free from danger in the first place, and in the second place that it does not always protect against recurrence. For a description of the methods employed—crushing, destruction by the galvano- or thermocautery, cutting, forced dilatation, carbolic acid injections, or cauterization with fuming nitric acid, the reader is referred to the surgical text-books. Whenever possible the operations should be undertaken only by expert surgeons. We may, however, say a few words concerning the two last-mentioned methods.

Injections of carbolic acid were first practised in America (Pooley, Agnew, and others) and greatly praised. The opinions of experienced surgeons in regard to this treatment are divided, but are on the whole favorable. The following points are to be borne in mind: The tumors must not be inflamed, and should first be covered with an iodoform ointment in order to guard against any cauterization of the mucous membrane. If necessary, the injection of a one-per-cent. solution of cocaine may precede the carbolic-acid injection. According to the size of the pile, various quantities of the carbolic acid solution (one part of carbolic acid to three parts of glycerin) are injected, but care must be taken that the outside of the needle is wiped off so as not to produce a cauterization at the point of entrance, and that the injection is made into the centre of the pile.

The pile must not be perforated so that the carbolic acid is injected into the cellular tissue, as this is apt to cause an abscess. Not more than five drops should be injected in each pile, and a few days should intervene between the single injections, but a number of piles may be treated in one sitting. Following the injections, the patients are to keep quiet for a few days and live on a light diet; should pain come on, cold compresses are indicated. This operation, if carefully and properly done, is painless and safe, and will give good results. I have no personal experience with it, as all the cases coming under my

observation which required surgical measures have been treated with the thermocautery after ablation of the piles with von Langenbeck winged forceps, and there have been no unpleasant results.

Cauterization by fuming nitric acid was first recommended by Houston, of Dublin. After the piles have been cleansed and well dried they are touched with a gold or glass rod, previously dipped in nitric acid. The skin of the anus should first be protected from injury by thickly coating it with oil or grease. After the surface of the pile has assumed a yellowish-green tint, it is to be dried, painted with oil, and returned into the rectum. In a few days the cauterized portion will be thrown off.

NERVOUS DISEASES OF THE INTESTINE.

(Intestinal Neuroses.)

In the intestinal wall are situated two large deposits of ganglionic masses that spread on the one hand in the submucous tunic and on the other hand between the layers of longitudinal and circular muscular fibres. They are known as Meissner's and Auerbach's plexus respectively. As is well known, the nerve supply of the intestine is derived from the splanchnics and the vagus. While we know little more about the sensory nerves and their course than that they extend into the splanchnic, we are much better informed as to what influences motility, thanks to the investigations of Pflüger, Nothnagel, Braam-Houkgeest, and Ehrmann.[50] Pflüger was acquainted with the inhibitory influence of the splanchnic nerve, irritation of which produces vascular contraction and anæmia, and he proved that paralysis of this nerve is associated with hyperæmia of the intestinal vessels and is followed by increased peristalsis. Ehrmann[50] ascribes to the splanchnic and vagus a double and crossed action upon the longitudinal and circular muscles of the intestine which may be schematically represented as follows:

Longitudinal fibres moved by the splanchnic	×	inhibited by the vagus.
Circular fibres moved by the vagus		inhibited by the splanchnic.

The circulation is of importance for the movements of the intestine in so far as hyperæmia, such as is connected even physiologically with the act of digestion, produces spontaneous intestinal movements, while anæmia of the intestine arrests or at least lessens the movements. That external and internal irritations of a solid, liquid, or gaseous nature may stimulate the intestine to contract is a matter of daily experience and has, moreover, been tested experimentally by

Bokai. Nothnagel's experiments are well known; he exposed the testine and caused it to contract by placing upon it small crystals a sodium or potassium salt. But attention may be called to peculiar fact that a sodium salt produces a contraction which exte for a distance of several centimetres towards the stomach, while application of a potassium salt is followed by very limited local eff After paralysis or death of the intestinal nerves, the longitudinal c traction after application of the sodium salt ceases and the local ef alone remains.

Our information is scant, however, with reference to the secret nerves, and the experiments upon the direct or reflex irritation of afferent intestinal nerves agree only in their very uncertain and c tradictory results. But there is no doubt that the nerves of the int tine, like those of the stomach, are immediately controlled by the br and spinal cord and this not alone with reference to the normal cou of their vegetative functions, but they may also be implicated by normal processes of a psychical or physical nature in the cent nervous system.

Among the neuroses of the intestine are to be included all morbid processes which are not based upon a demonstrable anato cal cause. They may be divided into disturbances (1) of motility, of sensibility, and (3) of secretion.

Motor Neuroses of the Intestine.

These depend upon either an increase or a diminution of the n mal irritability of the motor nerves. In the former case the result i

Nervous Diarrhœa.

This term is not quite correct; for, while it is true that in the m jority of cases of so-called nervous diarrhœa thin stools having a di rhœal character are passed, isolated cases occur in which the sto are simply increased in number, and though the fæces are soft th are formed and by no means fluid. In other cases again a diarrh stool may be passed, but it is limited to one or at most two ev uations and therefore the type of a true diarrhœa is lacking. T cause is usually a morbidly increased irritability of the periphe nervous apparatus of the intestine; in other cases reflex proces springing from the central nervous system or from other organs at fault. The persons affected by these conditions are almost inv ably—unless direct organic changes, especially of the spinal cord, in question—nervous subjects, neurasthenics, in short, those hav

an irritable nervous system. In some cases the irritation is obviously propagated from the stomach. There are persons who immediately after having partaken of any kind of food or after having swallowed a mouthful of fluid, especially cold and carbonated drinks, or a tablespoonful of medicine, have a diarrhœal evacuation. Not rarely it is a certain article of diet, *e.g.*, butter or chocolate or eggs, which produces the reflex. In other instances it is some psychical excitement or a picture of the imagination, fright, fear, or merely the performance of a special task differing from the ordinary daily routine, such as making a public speech, a summons to court, etc., even the idea of defecation, that causes at once a thin evacuation or possibly a diarrhœa lasting several days. That many soldiers are affected with diarrhœa immediately before or at the beginning of a battle is a fact cited by Trousseau and drastically described by Zola in "Le Débacle." There are persons who are completely barred by such conditions from all extensive social intercourse and when they cannot evade the latter are placed in extreme embarrassment. Frequently the evacuations consist first of firm fæces as long as they are still present and later they become semifluid and liquid.

We must regard as pure reflex neuroses also those forms of diarrhœa which are caused by morbid changes of an organic nature in the central nervous system, especially in the spinal cord, or which are produced by diseases of the sexual organs. In rare cases, as for instance in tabes, there occur also, beside the anal crises which consist in a spasm of the sphincters, diarrrhœal crises, *i.e.*, attacks of several days' duration of obstinate watery passages, the patients having eight, ten, twenty, and more thin-fluid stools which are usually free from mucus and almost odorless. Characteristic of these attacks is the fact that they, like the so-called gastric crises, coincide generally with the preataxic stage of tabes.

In the same class belong the diarrhœas connected with the sexual organs of both men and women; but these forms, as a rule, do not reach the intensity of the above-named tabetic crises and are also much more amenable to local treatment. I have met with numerous instances of such "nervous diarrhœas" which occasionally present very peculiar forms, but it would be unnecessary to relate them, as the morbid picture is sufficiently evident and easily recognized.

As nervous diarrhœas in a wider sense should be regarded those forms which appear in certain maladies in which the blood is overloaded with toxic products, for instance in some infectious diseases, in chronic uræmia, and in septicæmia. In these cases the cause evidently lies in the irritation of the intestinal nerves produced by the blood which is altered and perhaps charged with the products of

metabolism, those of the specific bacillus, or with retained noxious substances.

It is by no means certain whether in all these cases we have to deal with an increased peristalsis, with an associated augmented secretion of intestinal juice, or merely with an excessive transudation. Usually all the factors named are probably active at the same time.

The *diagnosis* as a rule will be readily made by excluding organic and other causes, especially when the well-known symptoms of hysteria, neurasthenia, etc., are present. Still, serious mistakes are liable to be made. I have repeatedly seen cases of intestinal cancer in relatively young persons who, before a tumor was palpable, presented the symptoms of nervous diarrhœa. Obviously in these cases the diarrhœa was temporarily caused by the irritation of the existing or developing neoplasm. As in every instance marked hysteria was associated with the tumor the diagnosis of nervous diarrhœa was apparently well founded until the further course, the occurrence of blood in the stools, the demonstration of a tumor, and the increasing cachexia made the nature of the case clear.

Occasionally a chronic intestinal catarrh may be confounded with a nervous diarrhœa. One characteristic difference above all is the fact that in chronic intestinal catarrh there are usually loss of weight and signs of a general disturbance of nutrition. The loose passages occur regularly during the night or in the early morning and are distributed through the whole day. The intestines and especially the colon are sensitive on pressure. Diet and medical measures in general have a marked influence upon the consistence of the evacuations and upon the condition of the patient, although at times a prolonged and consistent treatment is required to produce lasting results.

In intestinal neuroses, on the other hand, there is often an obvious contrast between the long-continued diarrhœa and the good nutrition of the patients, who often present themselves complaining that no one will believe that they are sick because they look so well. The diarrhœa, as a rule, occurs about the hour of normal defecation or in connection with the taking of food. The stools follow each other in rapid succession and then cease for a greater length of time. Not rarely marked aortic pulsation and a painful point on pressure upon the aorta or iliac artery are present. The well-known stigmata of hysteria and the manifold complaints of the neurasthenic make the true character of the trouble clear. Diet produces hardly any effect nor does medication, and at times it may even happen that an apparently most unsuitable mixed diet relieves the disturbance.

The *treatment* of the nervous affections of the intestine will be

considered together at the end of the present section in order to
repetition.

Among the neuroses of the intestine with increased irritabil
the motor elements is to be included also—

Spasm of the Intestine (Enterospasmus).

In this affection we are not dealing with the normal or inc
though still physiological innervation which results in the peri
movements of the intestine; but instead of the alternating im
which, as shown in the above schematic representation, proceed
the vagus and the splanchnic nerve by turns and under normal
tions produce the normal peristalsis, both nerves act simultane
The consequence is naturally a combined contraction of the
tudinal and circular muscles of the intestine, *i.e.*, a more or less
plete occlusion of the lumen of the canal. Such a spasm may i
a variable length of the intestine, that is, it may be diffuse or ci
scribed and may differ in its duration. The nature of a spasm,
ever, involves the eventual fatigue of the muscle with relief
contraction. Still it always causes a more or less prolonged ar
the intestinal contents. We may distinguish an idiopathic
secondary or symptomatic spasm, which latter is a concomit
basilar meningitis and of chronic lead poisoning (lead colic). A
ing to the extent of the intestinal contraction the abdomen is ret
in the shape either of a boat or of a trough and gives a tensely
feel on palpation. But the circumscribed form which prese
external characteristics is more frequent than the diffuse.

A so-called spasmodic constipation results which may be ve
stinate. When this, however, persists for days, the final con
would no longer be a spasm but rather a paresis of the intes
The evacuations are either of the form of a lead pencil or ri
like, or globular, resembling sheep's dung, and dark in
owing to their long retention in the intestinal canal. But, a
been explained above, these stools have no pathognomonic diag
value.

Occasionally, however, the spasmodically contracted inte
segments can be felt as swellings through the relaxed abdominal
In that event a careful and thorough examination is required t
tinguish their nature and to guard against confusing them
malignant tumors, invaginations, etc. Inasmuch as the inte
especially when the spasm involves the lower segments, can onl
difficulty or not at all be distended with air or water throug
rectum, we must rely upon the other previously described cr

the smooth tumid form, their passive mobility, and the absence of other characteristic signs of a neoplasm, in our diagnosis.

Pain, so-called colic, is frequently present in enterospasm, but it is not constant. It is usually referred to the umbilical region and is described as pressing, stretching, boring, or cramping. Movements of the body, passive pressure, massage and the like increase the pain; after the passage of fæces it moderates or disappears entirely for a time. Occasionally the spasm of the intestines is associated with spasm of the sphincters, thus rendering defecation extremely difficult or impossible. The act is always accompanied by great pain.

When enterospasm has existed for some time, it is nearly always complicated with an atonic condition, a paresis of the intestine above the contracted portion, and it is said that there may be a distention and filling of this part of the bowel with fæces and gas as in an organic stricture. I have never seen anything of the kind and I must confess that among a large number of nervous diseases of the intestine I have very rarely met with such cases of idiopathic enterospasm.

When the dejections are covered or mixed with much mucus, we are justified in inferring the coexistence of colitis. In many cases this condition or an increased irritability of the intestinal wall may furnish the first cause for the development of enterospasm. This is certainly the case when we apply the name enterospasm to the occasional colicky pains which occur in the course of such catarrhal conditions. While this would be literally correct, nevertheless we usually understand by the term enterospasm an independent affection.

A typical example of the disease is furnished by the following case:

P. B., aged 45 years, has complained for more than ten years that in the early hours of the morning, beginning between five and six o'clock, he suffers pain in the abdomen, which often radiates to the anus and at times becomes almost unbearable. At the same time there is continual tenesmus to which he is often forced to yield, although in about fifteen efforts something is passed only three or four times. The dejections are sometimes hard, sometimes soft, and often mixed with mucus.

These disturbances, which confine the patient to the house the whole morning and when he attempts to go out frequently force him to return in order to yield to the tenesmus, moderate towards noon to recur the next morning at the same hour. The patient is a gardener by profession and has always been irregular in the summer, when he loses a few pounds in weight, which he regains in winter. He has been under treatment for a number of years and has received many different narcotics and enemata. Galvanization, iodide of potassium, and of course a carefully selected diet have been tried without avail.

An examination showed descensus of the stomach without special symptoms of stasis, but with absence of free hydrochloric acid.

There was a slight difference between the pupils, and the knee jerks were absent. There were no disturbances of sensibility, no ataxic phenomena, no Romberg symptom. No tumor could be demonstrated in the abdomen.

There is no doubt that this was a case of enterospasm, and it is probable that the symptoms were the preataxic phenomena of irritation of a gradually developing tabes. Possibly the occurrence of these early morning spasms is due to the fact that during the night the rest or a definite position causes an accumulation of irritative factors which are lacking during the day. It may perhaps appear surprising that tabes—if we understand by this term the classical symptomatic picture of pronounced sclerosis of the posterior columns —should cast its shadows before for so long a time.

In this respect I may state that in the course of many years I have observed a number of cases in which the clinical picture of tabes developed with extreme slowness and was inaugurated by irritative phenomena in the digestive tract, such as gastric crises, enterospasm, and anal crises, which persisted for many years, while only very insignificant symptoms and certainly no ataxic disturbances could be demonstrated. Yet the eventual development of the orthodox symptoms of tabes did not admit of a doubt that the first-observed symptoms of irritation must likewise be ascribed to the development of this insidious disease.

Women are affected more frequently than men, and this fact depends upon the larger percentage of women suffering from all diseases of this class.

The affection is often very protracted in spite of every therapeutic measure, so that the patients suffer greatly in their nutrition and general condition and become in fact unfit for any kind of work.

The *diagnosis* must be based upon the careful examination of the abdominal organs, upon inspection of the fæces, and above all upon the exclusion of all other causes which may produce spasm of the intestine, whether by direct influences or by reflexes from other organs.

A variety of enterospasm is—

Proctospasm or Spasm of the Rectum.

This consists in attacks of painful contraction of the sphincters and is, in the majority of cases, a secondary affection that may occur with inflammatory and ulcerative processes in the rectum and colon, with fissure of the anus, with periproctitis, and with inflammatory diseases of neighboring organs, the bladder, the prostate, the uterus and its adnexa.

Independent of such local causes, spasm of the sphincters occurs

in very nervous, hysterical persons, in tabetics, and after sexual excitement and excesses.

The affection may depend either upon an irritation of the sphincter muscle or upon a reduced excitability of the inhibitory nerves.

The spasmodic attacks may vary in duration and intensity. They may pass with lightning rapidity, or remain for a day and night, sometimes for several days, or recur at regular intervals, as for instance in tabetics. In the latter case they are very intractable. One of my patients with periodically recurring anal spasm due to a slowly developing tabes has now been three years under my observation. As a rule the spasms are not very violent, but they become aggravated by a digital examination so that a thorough exploration of the rectum can be made only under anæsthesia.

Peristaltic Restlessness of the Intestine.

There is a peristaltic restlessness of the intestines, that is, a morbidly increased mobility which is not dependent upon coarse mechanical causes, such as a stenosis of the intestine and the like or inflammatory irritative conditions, which ordinarily provoke increased peristalsis. These so-called *tormina nervosa*, for which no other cause can be found than the heightened irritability of the nervous system, occur especially in nervous persons, hypochondriacs, neurasthenics, and hysterics. It was Kussmaul in particular who in Germany called attention to this condition in connection with so-called peristaltic restlessness of the stomach.

The patients not only complain of the sensation of agitation and contractions in the abdomen which follows especially after meals, though at times apart from any such cause, but the vermicular movements of the several distended intestinal loops can also be recognized through the abdominal walls. In this way is produced a peculiar and extremely characteristic picture which of course is to be observed only when the abdominal walls are relaxed and the cushion of fat is not thick. In some cases this peristalsis is not visible. In place of it loud rumbling, gurgling, or squeaking noises become audible even at a distance, and these may be extremely embarrassing when they occur in ladies and particularly at unseasonable hours, *i.e.*, in society. It is chiefly the small intestine, rarely the colon, which is the seat of this increased activity; but it is not always easy to determine which segment is involved or whether it is the intestine at all that is affected. For when, as is very often the case, the stomach is at the same time dilated or descended, the movements or noises may be perceptible below or around the umbilicus and as they cease when the stomach

is distended, transillumined, or filled with water, there is no reliable diagnostic sign to show which portion of the digestive tract is involved. Not rarely, too, both stomach and intestine may be affected.

These conditions are not, as might be believed, connected with increased or normally frequent dejections, but on the contrary with constipation, whence it would be reasonable to suppose that the colon is not implicated and that the movements terminate at the valve of Bauhin. It is also possible that a downward peristalsis is prevented by an antiperistalsis.

Usually no demonstrable cause can be found for these conditions. Not rarely they occur in connection with psychical excitement. In women occasionally, but by no means always, tight lacing seems to produce them; at least the gurgling sounds cease in some patients when the corset is discarded and the skirts are not fastened at the waist but are suspended from the shoulders.

Atony of the Intestine.

It may remain an open question to what extent so-called atony of the intestine, *i.e.*, the disproportion between the expulsive force present in the bowels and the labor to be performed by them, is to be considered as a purely nervous affection, as a functional neurosis, or is to be ascribed to a paresis of the intestinal muscles due to a mechanical cause producing a more or less complete insufficiency for the work assigned to them. Unquestionably both causes have the same effect, *i.e.*, more or less obstinate constipation.

Habitual constipation, its causes and results, has already been discussed at length in another portion of this article, where reference was made to atony of the intestine, *i.e.*, chronic constipation.

In this connection, therefore, it will be sufficient to point out that habitual constipation occurs also as a result of a nervous insufficiency of intestinal mobility and may present the features of an independent disease. According to Nothnagel, it is due to an abnormal nervous inception of the peristalsis of the colon and rectum, that is to say, to an abnormal function of the intestinal nervous system. By this explanation, however, the subject is by no means exhausted, for in many cases—for instance, in hysteria—there is surely not only a defective peristalsis of the colon, but the whole length of the intestine, and frequently the stomach as well, has a weakened peristaltic motion. Persons of this class do not react to any variety of cathartics, whether these act upon the small intestine or the colon. The condition is not one merely of an "abnormal nervous inception," but a real weakness, an atony, or a paresis is present, which manifests itself by the fact

that such drugs as would remove "an abnormal nervous inception" produce no effect. It is hardly necessary to state that the patients suffering from this form of nervous atony are chiefly of the neurasthenic and hysterical class, and those with psychoses of different forms, in whom the most obstinate varieties of chronic constipation occur, which resist every medication.

In these cases it is often a matter of experiment to determine whether the constipation is caused by the above-mentioned spasms or by atony of the intestines. In the former case the colicky pains are rarely absent in the morbid picture and we may succeed (but by no means always) in securing a passage by antispasmodics, especially small doses of opium. The quality of the fæces, however, presents nothing characteristic of these conditions, though Fleiner maintains the opposite view.

As nervous constipation in a more extended sense we must regard that form which occurs in diseases of the central nervous system, such as tabes, myelitis, meningitis, and cerebral tumors. This form, however, is not a morbid entity, but the atony, and in severe cases an intestinal paralysis, occurs merely as a symptom of the principal disease.

The same remark applies to—

Paralysis of the Sphincters.

This is a secondary phenomenon occurring usually with prolonged organic disease of the rectum, such as proctitis, hemorrhoids, ulcerative processes, strictures, and carcinoma; or with affections of the central nervous system, the brain or spinal cord, in which the patients lose the power of contracting the external sphincter at will. This paralysis of the sphincters may be of different degrees, ranging from a slight weakness to complete paralysis. In the former case the patient when attentive and at rest can usually retain gas and semi-solid or hard fæces; but during coughing, sneezing, and micturition a portion of the fæces, especially when they are fluid or semi-liquid, escapes into the linen or the bed. In some persons this is quite a common occurrence, in the absence of other particularly morbid conditions. When the paralysis is marked (incontinentia alvi) the intestinal contents escape involuntarily and without the knowledge of the patient, and especially with associated surgical injuries there is a continual flow of a muco-sanguineous matter which macerates and excoriates the skin around the anus.

Sensory Neuroses.

In the following remarks we shall pass over such conditions of increased sensibility, so-called enteralgia or neuralgia mesenterica, which may occur now and then as concomitant symptoms of the most heterogeneous intestinal diseases. These include also the colicky pains which may follow direct nerve irritation in over-stimulation of the intestine, in inflammatory, catarrhal, and ulcerative processes, in neoplasms, distortions, and displacements of the intestine, in foreign bodies, enteroliths, gall-stones, worms, etc. These have been discussed in connection with the respective conditions.

There is, however, a true sensory neurosis of the intestine, a real neuralgia, due to a morbid increase of the normal irritability of the sensory intestinal nerves. It is associated with or follows upon neurasthenia, hysteria, hypochondriasis, and tabes. It may occur in the form of so-called irradiated pain with disease of various abdominal organs—liver, kidney, bladder, ovaries, and uterus. Finally it is caused by direct alteration of the intestinal nerves or the ganglionic plexuses in lead colic and malaria.

Symptoms.—Enteralgia sometimes sets in with general dyspeptic symptoms, eructation, nausea, anorexia, etc. The pain rarely begins with great violence but increases gradually, assuming most variable types, such as boring, pinching, irregular, cutting, etc. It is usually confined to a certain spot and the patient indicates accurately whether it is felt more superficially or deeply, or whether it radiates towards the sacrum or the sides. It may attain an extreme violence so that the patients bend double or faint. Sometimes it occurs at regular intervals, not rarely soon after meals; at other times the periods are quite irregular, weeks and even months apart. There may be only a single attack or one prolonged for a few days, but the attack may also continue several weeks and longer. In some patients the attacks are brought on by a special cause, such as a cold or carbonated drink; in others, however, no cause is to be discovered. When the enteralgia is limited to the lowest segments of the intestine it is termed hypogastric neuralgia; then the painful sensations are located chiefly in the lower abdominal region and are associated with a violent pressing sensation in the rectum and bladder, which may extend to the perineum and thighs in case the nerve fibres of the hemorrhoidal plexus are implicated.

These true enteralgias should be clearly differentiated from the pains occurring in the abdominal muscles, which are due either to a hyperæsthesia of the abdominal walls or to rheumatism of the abdo-

minal muscles. The former are easily recognized by the fact that the patients evince a special sensibility when a fold of the abdominal wall is lifted and pinched. Rheumatism of the abdominal muscles will hardly ever appear isolated, but will usually be combined with other rheumatic pains. The pain increases during contraction of the abdominal muscles, for instance, in stooping and straining; it moderates when the patient lies quiet and it often changes its seat. *Ex post facto* it may be recognized by its reaction to antirrheumatic drugs.

Secretory Neuroses of the Intestine.

The only manifestations of a morbidly increased activity of the secretory nerves of the intestine are mucous colic and membranous enteritis. In the former the colic is the predominant element; in the latter, the catarrh of the mucosa.

Membranous or Mucous Enteritis.

When, however, the morbid secretion of mucus now under discussion is based upon a neurosis, neither term corresponds exactly with the condition. A discharge of mucus occurs very frequently without any colic and without causing any sensation to the patient, and even when it is associated with pain it is by no means always due to an inflammatory condition of the intestine, as the term membranous enteritis would indicate. As a matter of fact, in mucous discharges from the intestine different processes prevail, which invariably cause a profuse secretion of mucus to appear on the surface of the bowel, in one case without any change in the mucosa, in another associated with slight inflammatory (catarrhal) conditions of the latter. The last-mentioned state in a more pronounced form has been referred to in connection with intestinal catarrh. In this condition, however, the secretion of mucus is of a secondary importance and results from the inflammatory irritation of the mucosa. Only those cases should be interpreted as a secretory neurosis in which the morbid secretion of mucus is the original trouble. That in course of time a secondary alteration of the mucosa may likewise occur need cause no surprise.

As to the composition of the evacuations, thorough investigations have been reported by Kitagawa, Wolff, Ewald, and others; but only two autopsies with careful microscopical examination of the mucosa are recorded by M. Rothmann, and by O. Rothmann and C. Ruge.

The membranous mucous masses discharged from the intestine appear on macroscopic inspection sometimes ribbon-like, sometimes tubular, sometimes membranous or ragged, sometimes they even form thick solid lumps or are arborescent somewhat like coral. They

are quite friable and but slightly tensile, and are at times evacuated in enormous quantities. Their color is usually grayish-white, though brown, almost blackish masses also occur. The dejections are sometimes intermixed with sago-like granules which are clearer and translucent, resembling frog's spawn.

Under the microscope the discharges are seen to consist of a uniformly turbid ground substance which on the addition of acetic acid becomes opaque and striped. It is interspersed with a cellular detritus consisting partly of strongly refractile granules, partly of cellular elements, desquamated epithelial cells, round cells, and peculiar glossy flakes. The epithelia are rarely intact, usually they are markedly granular and without demonstrable nucleus, partly in a state of vacuolation or disintegration. Besides there are found cholesterin crystals, needles of fatty acids, triple phosphates, remnants of undigested food, pigment granules, heaps of bacteria, and occasionally red and white blood corpuscles.

Chemically, a mucin-like body, fibrin in small amounts, a little nucleoalbuminate, and globulin have been discovered. Microchemically the mucus can be differentiated from fibrin by means of triacid.

At times the stools also contain a sand-like concretion, whitish or gray, resembling pounded white pepper, but which may be much finer or coarser. Now and then it may be present in considerable quantities. According to the analyses of Mathieu and Richard,[91] these concretions consist of about 30 to 50 per cent. of organic material, 60 per cent. of phosphate of lime, 3 to 5 per cent. of carbonate of lime, and 2 to 3 per cent. of other mineral substances. They can be derived only from the desquamated epithelia and the mucus, and may arise in the same manner as do similar formations in the obliterated gall-bladder, in the nasal cavities, in the tonsils, and in the bronchi, *i.e.*, they may be deposited from the stagnant secretion. Thus R. and W. Koch[92] have found concretions containing magnesia and lime in the epithelial detritus which had accumulated in the colon below an artificial anus. The almost constant constipation present in membranous enteritis permits the mucous masses to stagnate for a considerable time in the intestine.

As regards the condition of the intestinal mucosa, it was perfectly normal in the case of so-called mucous colic reported by O. Rothmann and C. Ruge, but in the other case described as membranous enteritis the mucosa of the transverse and descending colon and of the rectum was injected and swollen, and covered at various points with membranous or stringy masses of mucus which filled the space between the folds and projected into the lumen. They could be pulled off without loss of substance, but they penetrated so deeply into the

mucous membrane as to displace completely its epithelial investment t the more markedly injected spots. It is obvious that even this ery slight alteration of the mucosa cannot be traced to a genuine nflammatory process but represents a secondary product. In my pinion there is no reason to separate the nervous secretions of mucus nto two distinct forms, on pathologico-anatomical grounds.

Symptoms.—The most important criterion of mucous colic is the assage of the above-described masses. At times they are evacuated ithout any special sensation and the patient notices them by acci- ent. In other cases the dejections are associated with more or less iolent colicky pains which begin sometimes suddenly, sometimes nore gradually, and have their seat either in the left inferior abdom- nal region (descending colon) or are diffused over the abdomen. Disagreeable sensations in the bladder and genitals, and radiating ains in the thighs are also complained of.

Obstinate constipation is almost constant and the mucous masses ass either at the same time with the fæces, or they follow immediately fter, or on attempting to defecate nothing but the mucous masses are vacuated.

Mathieu distinguishes several forms: Those with acute or subacute ttacks which have a certain resemblance in their course to dysen- ery and even to typhoid fever, beginning as they sometimes do with a febrile gastrointestinal catarrh; also a continuous form, whose ourse is sometimes slight and sometimes more serious and is asso- iated with particularly violent pain, burning, abnormal sensations in he abdomen, nausea, etc. Solis Cohen observed two cases in which he symptoms simulated those of a relapsing form of perityphlitis. In other instances confusion is possible with gall-stone colic (the bortive forms of the latter which pass without icterus or demonstrable tone), when the pains occur intermittently and are located in the egion of the right flexure of the colon.

Although it might hardly be believed that these comparatively slight secretory anomalies would give rise to injuries of a general haracter, this is actually often the case. It is surprising how greatly he patients are worried by this condition. Of course it cannot be lenied that the affection occurs nearly always in nervous, neuras- henic, or hysterical persons, especially women, more rarely men, and quite exceptionally children. It may be asked, and the question has been frequently discussed, what relation these affections bear to each other. Some authors believe that mucous colic is to be regarded as a symptom of general nervousness; others, like Brunner, look upon the intestinal affection as the cause of the nervous symptoms. This may be the case in isolated instances, but in general the relation is un-

doubtedly the reverse. I have observed, not only once, but with extreme frequency, that in such patients the mucous colic developed only in the course of a long-standing nervous condition.

The disease is most intractable and very irregular in its occurrence and disappearance. It involves no special danger to life and no particular weakening of the system; the latter, should it develop, is to be ascribed to the fundamental nervous disturbance. Still, it may become very annoying and be the source of prolonged trouble and discouragement to the patients.

Treatment of Nervous Diseases of the Intestine.

The treatment of the nervous disturbances of the intestine may be considered together, since the measures to be taken apply to all neuroses.

In the first place it should be observed that the specific symptoms, such as constipation, diarrhœa, colic, etc., are to be treated with the several measures mentioned above under the head of the various intestinal diseases and in the introduction. Resort will be had, therefore, to the different laxatives, enemata, electricity and massage, astringents and antispasmodics, narcotics and sedatives, according to the necessities of the case.

One principle, however, should be placed at the head of every mode of treatment of the neuroses, and that is, never to employ too active measures which irritate the general condition of the patient as well as the local process and thus fail to do good but frequently do harm and make the trouble worse. This applies especially to those conditions which are based on an increased irritability of the nervous system, *i.e.*, enterospasm, the forms of peristaltic restlessness, the sensory and secretory neuroses.

The greatest care should be bestowed upon an appropriate dietetic and hygienic regimen. The diet, barring cases of nervous atony and paralysis of the intestines, should be bland and unirritating, *i.e.*, it should be composed of food substances which require the least digestive activity of the bowels and contain no material (skins, shells, seeds, cellulose, etc.) that irritates them. The kinds of food here in question have been enumerated in the introduction. The hygiene should not be limited to the physical side but include also the psychical element. The moral treatment in these conditions is a task not to be underestimated by the physician. Hence we must not confine ourselves to the treatment of the local disturbance, but should strive to improve the general health of the patient and thus to relieve the irritability and relaxation of the nerves by such customary reme-

lies as iron, arsenic, and the cinchona preparations, together with corresponding hygienic and dietetic measures, open-air life, gymnastic exercises, sports, and restriction of mental activity to a degree within the limits of the patient's powers.

In the therapeutics of the several neuroses the following will be of value:

1. *Enterospasm.*—Sedatives and antispasmodics, such as opium, belladonna, perhaps morphine, codeine, hyoscyamus, and the like. Codeine is preferable to morphine, as it is less paralyzing and more antispasmodic. Chloral by enema, best in an antispasmodic menstruum, such as infusion of camomile or valerian, or injections of warm oil, have repeatedly done me good service. When opium per os in small or medium doses fails, it is often effective hypodermically. Large doses are to be avoided, since they cause obstinate constipation. The latter is to be combated with mild laxatives only, such as rhubarb, cascara sagrada, castor oil, lukewarm enemata, or injections of oil, avoiding all harsh measures, such as drastics or ice-cold high injections, which are more apt to do harm than good. Otherwise, according to circumstances, we may try also the various sedatives; water, mud, or meal cataplasms; frictions with oil of chloroform or of hyoscyamus; bromide preparations, and the like. Occasionally in proctospasm lukewarm sitz baths and the insertion of suppositories containing narcotics prove useful.

2. *Peristaltic Restlessness.*—The treatment is to be restricted to the measures cited under number 1, but general sedatives such as bromine and arsenical preparations, possibly galvanization, will be given the preference. In some cases the administration of methylene blue has been of service, the movements of the intestine ceasing after even a few doses of 0.1–0.2 gm. (gr. iss.–iij.) of the powder.

3. The *atonic* and *paralytic conditions* of the intestine should be treated on the principles discussed under the head of Habitual Constipation. It is very important to accustom the patients to go to stool regularly at the same hour each day and to devote the necessary time to it. The injection of nitrate of strychnine (0.001–0.0015 at a dose) into the folds of the anus, recommended by Rosenheim, has never proved effective in my hands.

4. The treatment of *enteralgia* requires the same measures cited above under number 1. It would be simply a repetition to mention them again.

5. *Mucous Colic.*—The treatment of this condition has to meet different indications which Germain Sée described as follows: It should (*a*) evacuate the bowels without the resort to powerful and especially saline purgatives; (*b*) relieve the pain without narcotics; (*c*) prevent

the accumulation of gas, pyrosis, and meteorism; (*d*) counteract possible autointoxication. For these purposes are recommende flaxseed (a large spoonful of the seeds, steeped for three or fo minutes in cold water, after each meal); also senna (fol. senn 2–4 gm. = ʒ ss.–i.) in combination with hydrastis canadensis, whi is meant to correct the intense congestion of the intestinal vesse caused by the senna (fluid extract of hydrastis canadensis, 2 gm. = gr. xxx.–xlv.; senna extracted with alcohol, 6 gm. = ʒ i Make 30 pills. One pill three times a day); or from time to tin a dose of castor oil or three to four tablespoonfuls of olive oil i ternally, repeated on three successive days, or in the shape of an enema. As an anodyne use is made of bromide of calcium and chl ride of calcium (calcium bromide, calcium chloride, āā 50 gm. = iss.; water, 500 gm. = O i.) and of extract cannabis indica (0.1 gm. 120 aq. = gr. iss. in fl. ʒ iv., one tablespoonful three times a day) a of an alcoholic solution of menthol (0.1–0.15 menthol in 180 alcoh = gr. iss.–ij. in fl. ʒ vi.). As antifermentatives are recommend phosphate of sodium (3–4 gm. = gr. xl.–lx. as a dose), salicylate sodium (0.4 = gr. vi. as a dose), and borax. As intestinal disinfe tants are enumerated benzonaphthol, salol, resorcin, and salicylate bismuth, but these are considered inferior to phosphate of sodiu The dietetic treatment should avoid all solid and liquid foods whi constipate and encumber the bowels.

In this respect it should be stated that according to my experien there is hardly an affection of the intestine that resists all therapeut efforts more stubbornly than nervous mucous colic. Even the loc astringents, such as tannin and silver solutions per enema, which a not sufficiently emphasized in the above remarks, usually fail con pletely. The best results are obtained by large regular oil enemat once or twice a day, as recommended by Kussmaul and Fleiner. B even these produce often enough merely a temporary effect. T condition improves so long as the enemata are administered dail and recurs with its old intensity whenever they are discontinued f some time.

CONCLUSION.

Finally a few words may be said applicable to diseases of the i testines in general. Owing to the intimate relations between t stomach and the intestine it is obvious that many diseases of the i testinal tract will act reflexly on the stomach. Or, as Oppler h shown, the intestinal disorder may have its origin in the condition the stomach. Therefore it follows that a thorough examination the stomach is called for in many cases of intestinal disease, and

proper treatment of any gastric disease thus discovered will conduce greatly to a cure, or at least to anamelioration, of the enteric disturbance.

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HERNIA.

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HERNIA.

A HERNIA, or rupture, is the protrusion of a viscus from its natural cavity through an opening in one or more layers forming the wall of this cavity, usually the abdomen. The external skin generally remains unbroken, although it may be greatly distended.

Hernia may thus be of either congenital or acquired origin. In the *congenital* variety the sac exists prior to the birth of the child, but the contents of the hernia itself may not have appeared in the sac until after birth. In the *acquired* variety the sac is formed after birth, and the hernia simultaneously appears.

Frequency.—Hernia occurs much more frequently than the inexperienced world suppose. According to the report of the Surgeon-General's office, from among 335,000 recruits, 17,000 were rejected because of hernia. At the London Truss Society more than 8,000 cases apply every year, and at the Hospital for the Ruptured and Crippled in New York more than 5,000 are treated each year.

Sex.—Malgaigne estimated that about one male in every thirteen and one female in every fifty-two were ruptured. This is rather a liberal proportion; but it is true that a much larger number of men than of women are ruptured. This is because men subject themselves to greater strains in their more severe labors, use more violent exercise, and more often receive serious accidents.

Age.—The study of various statistical tables shows: First, that a very large number of herniæ occur during the first year of life; this is probably due to the fact that there is not an early and firm closure of the abdominal rings. Second, that the largest number appear during that period of life when the physical energies are most active. This may be demonstrated from the following table of Macready.

	Birth to fifteen.	Sixteen to fifty.	Fifty-one to end of life.
Males ruptured	25.4 per cent.	37.5 per cent.	36.9 per cent.
Females ruptured	29 6 "	47 1 "	23.1 "

Etiology.

Hernia was recognized by the early Greek surgeons, but they knew very little as to its causation. Galen was practically the first to study it with any degree of care, and he concluded that it was due to a *rupture*, or tear, of the peritoneum and portions of the abdominal muscles. In general this theory persisted until the seventeenth century, when dissections were more frequently permitted. Fernelius demonstrated by their aid that the peritoneum and the muscles were not ruptured, but that the hernia was due to a stretching and pouching of the tissues. Numerous theories were devised to explain this dilatation, but many of them proved unsatisfactory, such as the ingestion of various foods and drink, compression due to high breeches and to swaddling bands, diseases of the heart and lungs, etc.

One of the earlier theories which caused considerable discussion was that of the *lengthened mesentery*. It was supposed that the intestine could not pass beyond its limits in the abdominal cavity unless its mesentery had been stretched, but Callender demonstrated by many experiments that in most cases the small intestine had enough freedom of movement with its normal mesentery to prolapse from the abdomen. Lockwood also found that as life advanced the mesentery tended to lengthen and prolapse, so that it might press against the lower abdominal wall. He therefore concluded that this might cause a bulging of the wall, and so later produce a hernia. Macready has given two excellent reasons against this. First, the weight of the mesentery and intestines is too small to produce such a bulging; second, the statistics do not show such a general increase in the number of herniæ in old age as the acceptance of this theory would demand.

The Pull Theory.—In many cases it was noticed that the growth of lipomata was associated with the development of a hernia. These lipomata were derived from the subperitoneal fat tissue which formed one of the coverings of the hernia. Thus it was supposed that, as the lipoma grew and increased in weight, it pulled down a pouch of peritoneum, which later became the sac for the hernia. Closer study demonstrated that lipomata were of quite infrequent occurrence as compared with herniæ, and also that in the largest number of cases of herniæ there was no lipoma present.

The Pressure Theory.—This theory was first advanced about 1700, when it had been shown that the true cause of hernia was dilatation, and not rupture. It presupposed that the wall was weaker than usual about the area where the hernia later developed, and that when the intra-abdominal pressure was increased much greater than normal

this weakened area began to bulge so as to form the pouch into which the hernia later descended. As a rule, the hernia develops gradually, but at times it may occur suddenly as the result of some direct violent strain. Again, if this pressure is continued long enough under certain unfavorable (predisposing) conditions, even a normal wall might give way. Macready concludes that the principal cause of hernia resides in an imperfection of the structures which form the openings through which the hernia escapes. But a single factor is not as a rule sufficient to produce a hernia. This is seen by the occasional presence of a congenital opening without a protrusion.

The indirect or predisposing causes which gradually weaken the walls may be as follows: 1. Inheritance; very little knowledge can be obtained about this factor, but it probably acts only very remotely. 2. Occupation; this is a very potent factor. The largest number of herniæ occur among those classes of laborers who do the hardest work. Men are more apt than women to have double herniæ. 3. Parturition; this is also an important cause, for it has been shown that a much larger number of herniæ occur among women who are mothers than among other women of the same age who are childless. Also, the largest number occur during the period (twenty to forty-five years) when child-bearing is most frequent. 4. Straining more or less constant or repeated, as in chronic bronchitis or asthma in the aged, habitual constipation, etc. 5. Abdominal tumors, ascites, or anything which lessens the capacity of the abdominal cavity and so increases the intra-abdominal pressure. 6. Old age; as age advances there may be a large accumulation of fat (obesity) in the tissues, so that the muscular fibres become atrophied; they are then so lax that they stretch quite readily under the impulse of a cough or a strain. The bulging is not marked at first, but gradually it develops into a pouch, and later the hernia appears. In some this bulging may have existed for years without any hernia, but when old age comes hernia develops.

The parts of a hernia are a sac and its contents. This sac has a mouth, a neck, and a fundus. The opening of the sac into the abdomen is called its *mouth*, which may be round, oval, or merely a slit. The narrow constriction below is its *neck*, and the lower bulging portion is the *fundus*. The opening through the abdominal wall through which this sac and its contents escape is termed the *ring*.

The formation of this sac is as follows: When the intra-abdominal pressure is increased sufficiently there occurs a slight bulging of the peritoneum at the weakest point of the wall, the site of the ring. This is more evident to the sight than it is to the touch. When this

process has been repeated sufficiently the dilatation persists, and the sac is formed.

At first the sac may be free to slip back and forth between the tissues in the canal, but later it becomes adherent to the adjacent structures and cannot be completely reduced into the abdominal cavity. The wall of the sac is thin at first, but gradually becomes thicker and firmer. In many cases, if the sac is empty, there is a tendency towards contraction at the mouth. This may become complete or it may remain partial, so that the two cavities still communicate by a narrow canal. In some cases this constriction may later close above the lower portion of the sac. If this now become filled with a collection of serum a *hydrocele* of the sac is formed.

Contents of the Sac.—A hernia which consists of intestine alone is designated an *enterocele;* one consisting of omentum alone is an *epiplocele;* one consisting of both intestine and omentum is an *enteroepiplocele.* In general it may be said that those portions of the viscera which are the most freely movable will be the ones most often found constituting a hernia. This is evident from the fact that most herniæ consist of portions of the small intestine and the omentum. The most dependent portions are those first prolapsed, so that on the right side the ileum is frequently found, and on the left the sigmoid flexure of the colon. At times each one of the various organs has descended into the sac, the kidney, the spleen, the ovaries, the uterus, the bladder, the vermiform appendix, etc., as well as dermoid cysts and other abdominal tumors.

As the inner coat of the sac is the same as the peritoneum, it is susceptible to the same causes of irritation and inflammation, and, as a result, adhesions are often formed between the sac wall and the omentum, though less frequently embracing the intestine. These adhesions sometimes become so stretched out as to form bands, which are so situated as to constrict or compress the intestine, and may even produce obstruction and strangulation. In rare cases the sac wall may become the seat of new growths, sarcoma, carcinoma, etc. Tuberculosis has also been found here in twenty-five cases.

Inguinal Hernia.

Anatomy.—A hernia may be classified according to the opening through which it occurs, and thus an inguinal hernia is one which emerges through the inguinal canal. The term inguinal canal (*inguen*, groin) designates the space occupied by the spermatic cord as it passes through the abdominal wall. It extends from the deep, or internal, to the superficial, or external, abdominal ring. At birth

all the other varieties, and in the male more often than in the female, it is apparent that the cause must exist in some peculiarity of the inguinal canal. This becomes evident when the descent of the testis and the development of the canal are studied.

Etiology.

The testicles are first differentiated from the Wolffian bodies, and then normally begin to descend from their original lumbar position, arriving at the internal ring about the sixth month, and entering into the canal during the seventh month. They usually reach their final normal position in the scrotum about the eighth month, so that a month before the birth of the child the outlet through the peritoneum becomes obliterated, the walls of the canal grow together, and there is left merely a narrow slit for the passage of the cord. The left testicle generally precedes the right by about four weeks, owing probably to the downward pressure of the rapidly growing sigmoid flexure. Thus, as the opening on the left side becomes obliterated several weeks in advance of that on the right side, it has time to become much stronger than the right; and so at birth the right side is weaker, which fact probably accounts for the greater proportion of herniæ upon that side.

In a considerable number of cases this normal development may be arrested, and the more complete the arrest is so much the greater is the liability to rupture in early life. The testis may be detained at any point along its course, but detention generally occurs in the canal. The internal ring and canal thus remain distended, and a portion of the intestine may enter the unobliterated processus vaginalis, which projects into the canal, and we have a "bubonocele"—a beginning hernia. It may not advance much farther for years, but it generally acts as a dilating wedge, and the increasing intra-abdominal pressure ultimately pushes it downwards into the scrotum, forming the ordinary *congenital scrotal hernia*. If the testis does not come down in infancy it generally does soon after puberty, and is then almost always accompanied by a hernia. As this arrest is most often on the right side, most ruptures are found upon that side.

At a period considerably prior to the descent of the testis from the abdominal cavity a pouch of the peritoneum (the processus vaginalis) already extends down into the scrotum, and into this the testicle enters from behind as it descends. A short time before birth the upper part, or neck, of this pouch becomes constricted and obliterated from the internal ring down nearly to the testicle. The lower portion remains as a closed serous sac surrounding the testicle, and

is called the tunica vaginalis. In some cases the neck is not obliterated, so that the cavity of the tunica vaginalis is continuous with that of the abdominal cavity. When a hernia protrudes through and into this congenital defect it comes into contact with the testis and is designated a *congenital hernia*. In other cases, the acquired forms, the processus vaginalis remains open above, closing only at its lower end just above the testicle, thus giving rise to the funicular process of peritoneum which passes through the internal ring and canal down into the scrotum. If a hernia protrudes into this pouch it is designated a *funicular hernia*. This funicular hernia cannot be definitely diagnosed by an external examination alone without an operation. It is generally narrow, and the hernia seems to rest almost on top of the testis.

Further, the neck of the processus vaginalis may become closed at intervals only, leaving a series of sacculi along the front of the cord above the testis. If a serous collection occurs in one of these little sacs it is called a *hydrocele of the cord*. This is not infrequently diagnosed as a hernia, especially by those who are not familiar with the diagnosis of hernia in children. This formation must not be confused with a hydrocele of the sac, when the fluid accumulates in the true hernial sac. The former occurs more frequently in young children, while the latter is found most often in adults.

In another variety, the *encysted* or *infantile hernia*, the processus vaginalis closes merely at the upper end, and is not obliterated for any length. If a hernia descends in this case it is covered with a distinct sac, which is again invested by the upper end of the tunica vaginalis. In operating, the true hernial sac is found only after another serous sac, the tunica vaginalis, has first been opened. The term infantile refers rather to the condition of these parts than to the period of life when the hernia occurs, for this variety has been found by the writer in an adult of thirty-four years.

In the female there sometimes exists a predisposing factor in those uncommon cases in which the canal of Nuck remains unobliterated. This is homologous to the inguinal canal in the male, and transmits the round ligament of the uterus to its attachment on the spine of the pubis.

Symptoms.

Pain is generally felt prior to any protrusion, and it is often said to radiate from the umbilicus; it is dragging or tearing and colicky in character, and may sometimes be referred to the course of the ilio-inguinal or genitocrural nerves. This pain is aggravated by straining, as at stool, by coughing, heavy lifting, and, in a word, by anything which increases the intra-abdominal pressure.

Tumor.—A swelling can generally be recognized over the area of the internal ring and along the canal, yet in some cases there is a lateral bulging over the entire inguinal region and extending upwards towards the iliac region. This swelling, which is better seen than felt, is more evident when the patient is standing, and if it is slight, coughing or straining will outline it more distinctly. In the early stages, when the hernia is just beginning to protrude through the internal ring, it may be so small that it is not easily recognized by inspection, but it may be detected by invaginating the skin of the scrotum over the tip of the finger and inserting the finger through the external ring and upwards through the canal. If the patient now coughs, the *impulse* of the descending hernia will be felt upon the tip of the finger. This symptom is next in importance to that of the pain, and should be more often recognized and treated early, for in children the hernia might be quickly cured. If the patient lies down the tumor will often retract into the abdominal cavity. The tip of the finger may now be passed through the canal, and the internal ring may be clearly defined. In children a hernia is often overlooked at the first examination, because it is frequently difficult to obtain the necessary symptoms in order to make an exact diagnosis. If they feel the pain due to hernia they cannot locate it, and unless they cry or strain, the hernia may not protrude.

Diagnosis.

When a swelling is found in the groin, one should first decide whether or not it is reducible; if it is reducible, it is probably a hernia, and inspection and palpation will decide its variety.

An inguinal hernia may be either direct or indirect. An *indirect* or oblique hernia is so designated because it enters at the internal ring and takes an oblique course downwards through the canal and generally emerges later at the external ring. During the transition period, while it remains in the canal above the external ring, it is called a bubonocele or an incomplete hernia. In the male it generally passes the external ring and descends into the scrotum, forming a complete or scrotal hernia. In the female it descends into the labium majus, forming a labial hernia. This variety is also called an external hernia because the neck of the sac comes out from the abdomen external to the epigastric artery.

The largest number of inguinal herniæ are oblique (Macready says ninety-three per cent. in the male), because the largest number develop through a defect at the internal ring, and for this same reason a large number are congenital. This is probably true in a large

number of cases which are examined during infancy. The acquired form develops much more slowly and does not generally occur until later in life. This question can be settled best by dissection, but this will be possible only when more operations have been performed in children. Those who have operated upon a considerable number of children have found the congenital form often present. In typical

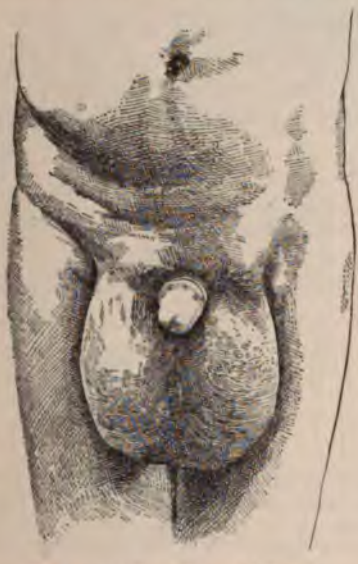

Fig. 21.—Indirect Inguinal Hernia.

cases the testis can be distinctly made out separately, below the hernia.

A *direct* or internal hernia is one which protrudes through the posterior wall between the epigastric artery and the border of the rectus; its neck is therefore internal to the epigastric artery. It thus enters the inner and lower part of the inguinal canal and emerges directly through the external ring; it may in old, long-standing cases descend into the scrotum. It is never congenital but always acquired and rarely is found before adult age. It occurs more often on the right side, but occasionally there seems to be a tendency for it to develop on both sides. The protrusion projects more abruptly than the regular oblique form and in outline it is more spherical and circular than oval (Fig. 23). The canal is short and straight rather than oblique, so that the finger tip can be pushed directly through the canal into the abdominal cavity. The edge of the conjoined tendon and the posterior upper surface of the pubis can be felt at the inner side, and the epigastric artery at the outer side of the opening.

In old, long-standing cases of oblique hernia the rings become so stretched and enlarged that the oblique direction of the canal is changed so that the internal ring is directly behind the external ring; the finger thus goes directly through the canal and into the abdominal

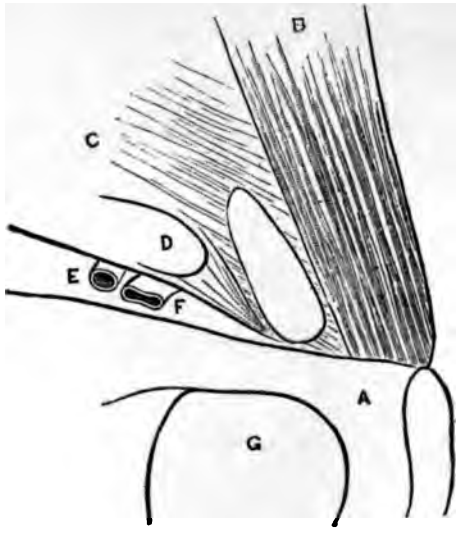

FIG. 22.—The Inner Aspect of the Inguinal Region from which the Peritoneum has been Removed. The aperture of a direct hernia is seen occupying the greater part of the space between the rectus and internal ring. *A*, The pubis; *B*, the rectus; *C*, inner wall of canal; *D*, inner ring; *E* and *F*, femoral artery and vein, *G*, thyroid foramen. (After Hasselbach.)

cavity. A direct hernia is thus simulated and the differentiation can be made only by a careful dissection of the parts.

Differential Diagnosis.

In a considerable number of cases a swelling may be found in the groin which is not at once readily recognized and so may cause an error in diagnosis.

A *femoral hernia* is not infrequently confounded with an inguinal hernia, but this should not occur if the landmarks, the spine of the pubis and Poupart's ligament, are clearly defined. The inguinal hernia always protrudes through the external ring just at the spine of the pubis, and lies above the fold in the groin made by the sharp edge of Poupart's ligament, whereas the femoral protrudes below this fold, and if the finger can be introduced into the opening the sharp, tense Poupart's ligament can be felt, and also the femoral vessels behind. In very fat patients it may be difficult to find the spine of the pubis, so the leg should be abducted to make the adductor longus tense, and this will bring out its point of attachment to the spine;

empty. The testis is small, oval, elongated, and solid, and the testicular sensation and pain are felt on pressure. If the patient is above the age of puberty a hernia also will probably be discovered.

Hydrocele (a collection of serous fluid in the cavity of the tunica vaginalis testis) is of frequent occurrence and is often mistaken for a hernia. The history of the case is that swelling begins below, about the testicle, while with a hernia it begins above and descends. The swelling is pyriform in shape with its neck above, and while the larger portion is below, its upper end is well defined, whereas a hernia is better defined at its lower end. It is tense, translucent, with a smooth surface, and fluctuates, but does not transmit an impulse on coughing. The testicle is inside the tumor, and the cord can be felt running up from the tumor and into the canal. The hydrocele cannot be reduced nor does its size diminish.

In infancy there is sometimes found the uncommon congenital hydrocele. A small opening communicates with the abdomen and so the fluid can be squeezed slowly into the abdomen when the patient is lying down, but when he stands up the fluid will run down again. If a truss be applied or a finger be held at the external ring the fluid will gradually come down, whereas if it had been a hernia the truss would have retained it.

Hydrocele of the Cord.—This occurs infrequently in children, rarely after puberty, and is often diagnosed as hernia by those who are not very familiar with hernia in infants in whom the parts are so small. It is generally between the external ring and the testis, is well defined, fluctuates, feels like a cyst, and is freely movable when the cord is pulled to and fro; the cord is of the same size above and below the tumor.

Hydrocele of the canal of Nuck sometimes simulates a hernia. This hydrocele occurs generally into the cellular tissue about the round ligament, or the cyst may be formed in a true peritoneal prolongation or diverticulum of Nuck. It occupies the canal and gives a peculiar elastic sensation, feeling like an elongated cylindrical cyst. It cannot be reduced and it does not transmit any impulse on coughing. It is of slow growth and does not vary in size. A hypodermic exploratory needle will confirm the diagnosis.

Varicocele (a tumor of the scrotum, composed of enlarged and tortuous spermatic veins) is less common than hydrocele, yet it is quite frequent. It is rare under fifteen or over forty years of age, and is most often found on the left side; the enlarged veins may often be seen just under the skin. It may be reduced on lying down, but the veins fill when the patient stands, and if a truss is applied the fluid runs down underneath the pad. There is no distinct impulse or im-

pact from the bowel, but a sort of thrill is felt which is due to the rushing movement of the fluid under the pressure of the finger tip. The patients generally complain of an aching, dragging pain beginning in the loins and running down the course of the cord to the testicle. Upon palpation the mass feels like a bag of worms; it is soft and compressible and painless, the testis can be felt at the lowest point and it is often softer than the corresponding healthy testicle; in advanced cases it may be quite atrophied.

In the labia majora there may sometimes be found cysts which simulate a hernia, also large varices of the labial veins, but when these are present there are generally varices elsewhere, so the local varix is but a part of a general condition.

Orchitis is often mistaken for a hernia, but upon careful examination it will be seen that the skin is slightly reddened over the tumor; that the tumor is very low down in the scrotum, that its outline is distinct, that there is no neck and that it does not extend upwards to the ring. The history is of but a few days' standing, and it may have been due to an injury or to some venereal infection. There is no impulse, it is not reducible, it is very tender on pressure, and harder to the feel than a hernia; there may be some slight fever.

Hæmatocele.—The history of the case is important, and it will be often found that the swelling comes on quickly after some injury, a blow, etc. The skin is discolored, and ecchymosis may be present; there is no impulse; fluctuation exists, but the tumor is not translucent.

Psoas Abscess.—In some cases this points directly in the region of the inguinal canal, and as it is soft, fluctuating, and may give an impulse on coughing, and even disappear on lying down, it simulates an indirect inguinal hernia. If a hernia is not present the canal will be empty, and if the back and spine of the patient be examined a deformity may be found; symptoms of tuberculosis may also be discovered elsewhere.

New Growths.—Sarcoma and tuberculosis of the testis are sometimes found, but careful examination will be sufficient to differentiate them from hernial protrusions. They simulate rather an orchitis or a hydrocele, but involve the testis only, and do not extend up the cord.

Diagnosis of the Contents of the Sac.

It is of importance to determine what comprises the hernia, for any of the viscera which have sufficient range of motion may be found in the sac. The most common element is the *small intestine*, for it normally has the greatest freedom. If gas is present in the

bowel percussion produces a tympanitic resonance; generally, however, the bowel is collapsed. If any fluid is in the intestine one may hear a gurgling; when the sac walls are very thin one can sometimes feel the smooth slippery walls of the intestine, and in rare cases the peristaltic movements may be recognized. In reducing the bowel the last part goes in suddenly.

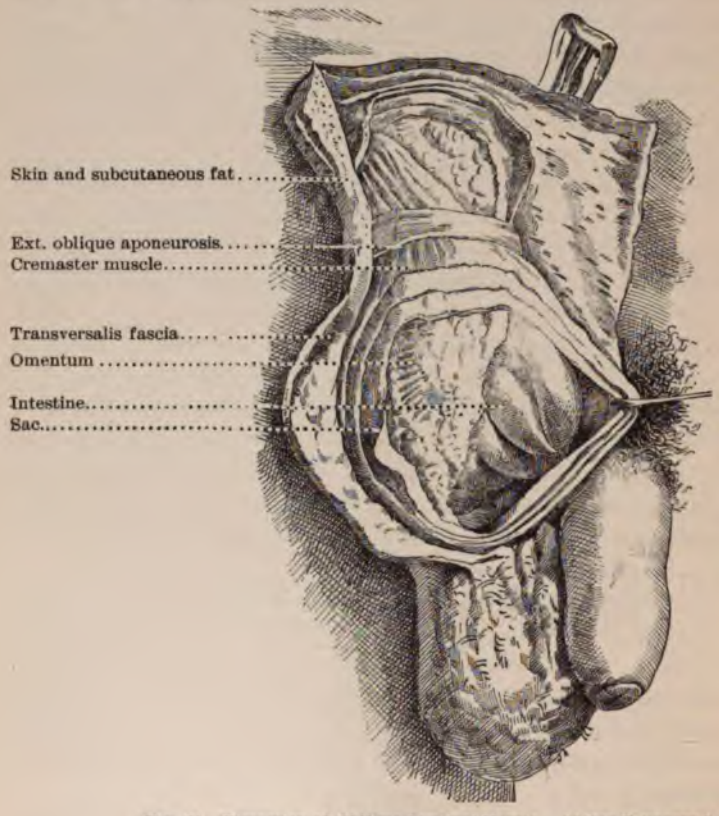

FIG. 24.—Dissection of Oblique Inguinal Hernia. (After Bourgery.)

The *omentum* is next in order of frequency and is generally reducible. It is usually in front of the bowel and is the last to return. In some cases it is adherent to the cord. When it is reduced into the abdomen, the testicle is also drawn up, and if the testicle is pulled down the adherent omentum comes down also. In other cases, after it has been down for a long time and under the pressure of a misapplied truss, it may have become irritated and inflamed and adherent to the sac. It then becomes compressed into a thickened, pedunculated, pyriform mass which can no longer be reduced. The neck of

the sac now becomes plugged, and an effusion may develop about the omentum and so a hydrocele of the sac is formed. The thick, firm neck of the omentum can be felt running up into the canal and is surrounded by the fluid. The testis can be distinguished outside and below this tumor. In rare cases the omentum itself may be the seat of malignant growths or of tuberculous deposits.

The ovary on account of its mobility is occasionally found in cases of inguinal hernia, and more rarely in femoral cases. It is due probably to an anomalous descent somewhat similar to that of the testis and so may act as a predisposing cause of the accompanying hernia. Further, the majority of patients in whom it occurs are under one year of age, and it is stated that in the largest number of these cases the ovary returns before the age of puberty; so that it may be said to be a transitory condition of infancy, although it occasionally is found in the adult. In children it is often reducible and may be retained inside by a truss, but if the condition persists until adult age adhesions will have formed which may prevent its reduction. If it remains unreduced it may become inflamed, or degenerate into a cyst, or become cancerous. It may be difficult to diagnose an ovary in the sac, but one should be suspicious if a small, oval, somewhat hard, tender, and very firmly movable body with a thin cord is found; and especially if, in adults, this temporarily swells or becomes tender during the menstrual epochs, or if, upon vaginal or rectal examination, it is found that by tipping or moving the uterus this suspected body also moves with each variation of the uterus.

Treatment.

This may be either mechanical or surgical. Formerly the "injection" treatment was tried in a large number of cases; but it was so unsatisfactory that it never gained much favor, and it has now been abandoned by those of the largest experience.

Mechanical Treatment.

By this is understood the use of a suitably adjusted truss, and in order to select a truss which shall be suitable for a definite case, the case itself must be carefully considered and every peculiarity noted. The chief aim of a truss is to keep a small, well-fitting pad directly over the internal ring and canal (when the hernia and its sac have been entirely reduced), so as to close the mouth of the canal and thus to destroy the communication with the abdominal cavity. A truss consists of a spring and a pad. The longer the spring and the more perfectly it fits about the body the steadier will the pad be held and the

easier will it retain the hernia. The shape of the pad should be oval, but not too conical, otherwise it may be forced into the ring, and thus in time gradually dilate the opening. Some have claimed that unless the pad has a groove on its under side it will compress the cord and thus cause atrophy of the testis, but the best authorities state that this groove is unnecessary. The pad may be covered with leather or chamois skin, but during the warm weather a smoothly polished hardwood, ivory, or rubber pad is the least disagreeable, especially if the patient perspires freely. In certain cases in which the patient is so very thin that the hard pad presses upon the pubis, much relief will be found by replacing the hard pad with a water pad. This is a rubber sac made of the size and shape of an ordinary pad, but it is filled with water or oil so as to be softer and act rather more as an elastic cushion than as a hard, unyielding pad.

The truss should be so adjusted as to fit perfectly the individual patient, for unless it is comfortable very many patients will not wear it. The spring should lie above the glutei muscles, so that in walking their action will not tend to disturb the position of the truss, and it must be so adjusted that its point of pressure comes over the hernial opening, while its point of counter-pressure comes behind the hip. The larger the spring the more closely it fits the body and the more evenly is the counter-pressure distributed, thus rendering the pad more steady. The latter should press backwards and upwards, and never downwards upon the spine of the pubis. The pad is so adjusted that its lower border is just above the os pubis and its inner border just in contact with the outer edge of the rectus.

The truss should be well made so that it will not give way suddenly when the patient is away from medical relief, and it is well to have an extra truss on hand in order to be prepared for such an emergency. One used for children should be impervious to moisture. It may be made of hard rubber or celluloid and should be carefully cleaned and dried whenever it becomes wet. A child must wear the truss day and night, and it should be removed only for bathing and cleaning, and then only when the child is lying upon his back.

The adult should put on his truss in the morning while lying in bed, and carefully hold it in position with his hand until he has assumed the erect position. It is to be worn during the day and removed after retiring. In some few cases among the aged who cough considerably at night and cause the hernia to be protruded it may be safer to advise the wearing of a light truss during the night. For these cases the elastic truss is very satisfactory, but as a rule it is not the best variety to wear during the day, for it cannot maintain a constant even pressure over the internal ring; sooner or later it loses

its elasticity and the hernia gradually begins to slip down and escape from under the pad.

The best truss is that which holds the pad in position, keeps it

FIG. 25.—The Frame Truss.

there under all movements, counteracts any expulsive action of the hernia, and causes little, if any, discomfort. The lighter it is under

FIG. 26.—The Opposite-Side Truss.

these circumstances the better, and the closer it is adapted to the body the more comfort it affords.

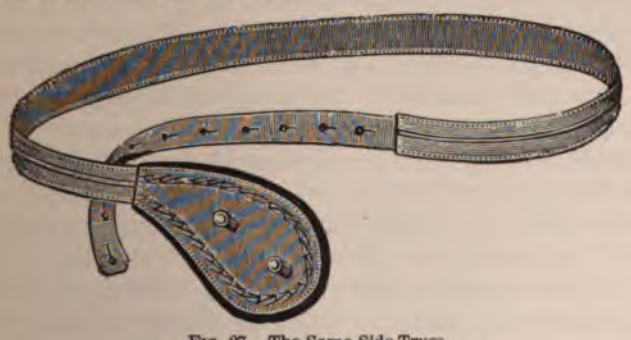

FIG. 27.—The Same-Side Truss.

There are three general forms of trusses which may be used in appropriately selected cases:

1. The "frame" or "hood" pattern (Fig. 25). This is probably

the most satisfactory variety of a truss, from the fact that it fits the pelvis so accurately and also retains the hernia so perfectly without undue pressure; firm pressure being exerted only when the hernia attempts to descend.

2. The "opposite-side" truss (Fig. 26), so designated because it is applied from the sound side and the arm to which the pad is attached, passes across in front of the lower abdomen towards the affected side.

3. The "same-side" truss (Fig. 27), so called because it is applied from the affected side, is well represented by the well-known French truss.

In taking the measurement for a truss one should first measure the distance between the external rings, then measure round the hips midway between the crest of the ilium and the trochanter major.

The cases which present themselves for trusses may be divided into the following general classes:

1. When there is a simple bubonocele—that is, an inguinal hernia in which the hernia remains in the canal, and has not yet passed through the external ring and descended into the scrotum. Any one of the above trusses may be satisfactorily used for this class.

2. The more advanced cases, in which the hernia has passed down into the scrotum, and the canal has become dilated but it retains its obliquity; here more skill is required to select and to adjust the truss, for the opening has become larger, and it is more difficult to keep the pad in the proper place so as to retain the hernia perfectly. The "frame" or "opposite-side" truss may best be used here.

3. In old, long-standing cases in which the internal ring has been so largely dilated that the canal has become obliterated and the hernia comes directly through the external ring; here the opening generally admits at least two finger tips and the hernia is a very large one, and thus a much stronger spring and a larger ovoid pad will be needed. In some cases, when the pad tends to ride upwards and to allow the hernia to slip out underneath, it will be necessary to retain the pad in place by a perineal band passed between the legs and under the gluteal fold.

In a few special cases in which the opening admits three or four fingers and the hernia has increased to the size of a child's head, no truss which can be applied will retain the hernia. A specially devised suspensory bag must be made, and these are generally quite satisfactory.

In the case of a very stout patient with protuberant abdomen it is difficult, and oftentimes impossible, to use any other than the "frame" or "same-side" truss.

In regard to *children's trusses* one must be very careful to see that the springs are not too stiff, for they frequently have this fault, pressing too strongly upon the thin, tender, abdominal walls so as to pain the child. The child becomes fretful, cries, and continually disturbs the truss. He should be kept as quiet as possible and free from all strain. In some cases of very thin, poorly nourished infants, it is difficult to retain the hernia with the usual forms of trusses. Recourse may be had to the hank truss, which is so soft that it may be very closely applied. But the general use of this truss is to be discouraged; it cannot sustain its pressure directly over the internal ring and the hernia continually slips down.

In some cases the treatment is complicated by the presence of an undescended testis. The end to be desired is to close the ring and to retain the testis in the scrotum. For this purpose the pad should be applied above the testis so that it will not only close the opening but also will tend to press the testis down towards the scrotum. From time to time the parent should apply gentle traction to the testis to facilitate its descent into the scrotum. If after prolonged and thorough treatment the testis still remains in the canal and keeps the ring open, surgical measures should be considered which will fix and retain the testis in the scrotum.

It is necessary that children should be very carefully watched, and that their trusses be exchanged for larger ones as they outgrow the old ones, for each truss must fit accurately if a cure is expected.

It may happen in any variety of hernia, as time progresses, that the hernia slips down, adhesions form, and the hernia becomes irreducible. In an acute case no truss should be applied. If the patient comes to the clinic, a compression bandage, generally a spica, is applied, and the patient is directed to go to bed, placing himself under careful observation, so that should the hernia become strangulated it may receive immediate attention. There are, however, quite a number applying for trusses who have had a small irreducible hernia for several months, sometimes for years, without any apparent trouble. In these cases it is sometimes possible to adjust a truss with a concave pad which will just fit over the irreducible mass, and which will so compress the structures about the canal as to prevent any further increase in the hernia. In a certain number of cases the hernia later becomes reducible from being subjected to a constant even pressure, but one that is not too strong. Among two hundred and fifty-eight cases which were irreducible when they applied to the London Truss Society, one hundred and fifty-six were reduced by the above treatment—fifty per cent. became reducible within three months.

Results.—It has been observed by those of the largest experience

that a large number of children are cured when suitable care has been taken to retain the hernia and to keep up a continual pressure over the internal ring, thus causing adhesions between the walls of the canal. It is not uncommon to hear an adult thirty-five or forty years of age state that he had had a rupture when an infant, that he wore a truss for a year, and was cured; and that the hernia had never returned until three or four days ago when he strained himself by lifting a very heavy weight. The fact that he was free from this hernia for thirty-five or forty years would indicate that a cure had occurred in infancy. Congenital cases are much more difficult to retain, consequently the prognosis for their cure is not good. They require specially close observation, and after five years, if they are no better, the Bassini operation is to be recommended.

In general, the prognosis is better the shorter the duration of the hernia; so while it is good during infancy it gradually diminishes up to puberty, after which period there is not much hope for a cure from a truss. It is much more favorable for girls than for boys because of the absence of the spermatic cord, but the prognosis for a femoral or for a direct hernia is bad because the adjacent tissues cannot be made to adhere under the pressure of a truss.

In adults the chance of a cure by the use of a truss is small; only a few remain ultimately cured. There is a large class who are said to be cured temporarily. The London Truss Society reports two hundred and forty cases in which the hernia did not reappear for an average of 22.5 years. One hundred of these cases were in children in whom the hernia had occurred during the first year. In only eighteen of these cases was the patient over twenty-five years of age. There were no reported cures in cases of femoral hernia. The prognosis, however, is most satisfactory and favorable for obtaining a symptomatic cure—that is, the hernia is well retained and the patients are relieved of all their unpleasant symptoms. Later they are able to wear a much lighter truss. It is very necessary that they should be fitted accurately and be kept under careful observation by one who understands their infirmity and who appreciates thoroughly the dangers which may befall them if they neglect themselves. The patient should also understand the principle and purpose of the truss, and appreciate the risks which may arise if the truss fails to continually fulfil its purpose.

Operative Treatment.

Attempts were sometimes made even in the time of Celsus to cure hernia by operation, and during the middle ages operations became quite frequent; but after a time, especially during the seventeenth

century, they fell into disfavor because of their high mortality and from the fact that there were so many relapses after the operation; furthermore, trusses were now so much improved that patients preferred to wear them rather than to risk the operation. At the beginning of the present century the new methods of subcutaneous operations seemed to lessen the dangers so much that operations began to be once more frequently undertaken. In 1835 Wätzer introduced a method of pushing up and invaginating the scrotum into the inguinal canal and then suturing or quilting through all the layers to keep it there, so as to close the canal. Wood introduced his operation a little later; it consisted in reducing the hernia and its sac, and then closing the rings and canal by a complicated subcutaneous suture of silver wire or animal tendons. In 1871, when Lister introduced the antiseptic methods and it became possible to operate safely through open wounds, the subcutaneous methods, were gradually abandoned. In 1876 Czerny published his method, with a series of cases, in which he had ligated the sac high up, and closed the canal and external ring with silk or catgut; he did not remove the sac below the ligature, but some later operators improved upon his method by removing the sac.

In 1886 Macewen presented a method which became very popular. He separated the sac very carefully from all adjacent tissues high up to the internal ring, but did not remove it. He then reduced it into the abdomen and folded it up so as to form a pad which would close and protect the internal ring; he finally closed the canal with chromicized catgut sutures. This method is less often performed at present because that of Bassini[2] is much more satisfactory. As it is the best method yet devised, its description will be given in detail.

Bassini Method.—1. The external incision begins at a point nearly or quite on a level with the anterior superior spine, continues obliquely downwards parallel and about one-half inch internal to Poupart's ligament, and ends at the centre of the external ring.

2. The incision is rapidly carried down until the aponeurosis of the external oblique is freely exposed for a distance of two and a half to three inches. A director is then passed through the external ring just beneath the aponeurosis, and the aponeurosis is divided one-half to one inch above the internal ring.

3. The cut edges of the aponeurosis are held up with forceps and dissected free from the underlying muscles as far as the edge of the rectus internally, and externally until the shelving portion of Poupart's ligament has been clearly exposed.

4. The sac and cord are then isolated *en masse*, which is best accomplished with the fingers and blunt-pointed curved scissors. If

the peritoneal layer of the sac is first reached the dissection is easy, rapid, and bloodless.

5. The cord and vessels are now separated from the sac, and this too is best done with the fingers. The separation is carried high up within the internal ring, and the sac is ligated or sutured at the place where it merges into the general peritoneal cavity, then removed below the ligation.

6. The cord is held up and the edges of the aponeurosis are rolled back, while five buried sutures of kangaroo tendon are introduced beneath the cord. These are best introduced from within outwards and should include the internal oblique and transversalis muscles, the transversalis fascia (and sometimes the edge of the rectus) on the inner side, and the deep shelving portion of Poupart's ligament on the outer side. The lowermost suture should embrace the conjoined tendon.

7. The cord is now replaced, and the cut aponeurosis is closed over it by means of a continuous catgut suture, extending as near to the os pubis as possible, care being taken not to cause undue constriction of the cord.

8. Closing the skin wound with interrupted catgut sutures, without drainage, completes the operation.

Another operation, that of Halsted,[3] is similar to Bassini's in many respects, with the exception of the treatment of the cord, and this peculiarity is not recommended by the writer.

Lucas Championnière and Kocher have each devised operations which have been very successful in their own hands, but the best for general use is the Bassini method, with the modification of Halsted of protecting the internal ring in weak subjects by placing a deep suture above and external to the ring.

The operation should be done under strict antiseptic precautions, with careful attention to every detail. The first incision should be rapidly carried down to the aponeurosis and then each bleeding point carefully clamped. When the sac has been freed as high up as possible it is better to suture the opening into the peritoneum rather than to ligate, for the ligature might slip off from the stump, and the peritoneal pouch can be more smoothly closed by a continuous suture than by a ligature. Catgut may be used here. The closing and strengthening of the posterior wall of the canal (by suturing Poupart's ligament to the internal oblique and transversalis muscles) must be completed with some suture which will remain unabsorbed until after the tissues are regenerated (from sixty to ninety days); the best suture for this purpose is kangaroo tendon or chromicized catgut. Some prefer silver wire or silkworm gut, but as

these are foreign bodies there is always danger that they may at any time irritate the tissues and slough out. The same suture material should be used to unite the divided borders of the aponeurosis. Care must be taken not to draw these sutures too tight, else the tendinous structures, of lower vitality than the muscular tissues, will slough. The superficial skin sutures are of catgut, which will remain unabsorbed about seven to ten days. Every bleeding point should be secured, as the wound must be perfectly dry in order to insure the primary union which is essential to a radical cure.

Complications.—The most frequent factor which is present to complicate the operation is a mass of omentum, which may be either reducible or adherent and irreducible; care in separating the adhesions and in accurately securing the stump will be all that is necessary.

The spermatic cord has been severed, but this should be avoided by a careful dissection. In some other cases the cord has been too much diminished in size by removing the adjacent veins, or the sutures at the internal ring have been too tightly drawn, and in either case atrophy of the testes will follow.

The operation has been complicated in a few cases by the presence of other organs—such as the appendix, the ovary, or the bladder. These exceptional cases will be separately considered later. The first dressing is changed at the end of a week when a lighter bandage is applied, and the patient is kept in bed for two weeks longer. It is best to wear an abdominal support for about three months, until the tissues are firm.

Benefits of the Operation.—Those of the largest experience claim that at least eighty per cent. of the operative cases remain cured. The mortality for the operation may be considered not higher than one per cent. in non-strangulated cases. Even in those cases in which a permanent cure does not result, the condition of the patient is improved, for the large scrotal hernia which could be controlled only by a strong and unpleasant truss may now be retained by a much weaker and more comfortable one; an irreducible and painful hernia has been removed, and a simple abdominal belt supports a weak abdominal wall and retains the hernia.

Indications.—In *children* the earlier the parts are returned to their normal condition the better, and as the chances of success are so much greater than those of failure, so the operation is advised whenever the hernia is congenital or when the truss has been worn more than five years and it is still unsatisfactory; when there is adherent omentum; when there is a reducible hydrocele; when the hernia is irreducible or strangulated; also in cases in which suitable treatment

cannot be carried out, when perhaps the child lives too far away, or when its guardians constantly neglect it.

In *adults*, when the hernia is irreducible, when it is complicated by an ectopic testicle, or when the truss does not prove satisfactory, the advisability of operation should be considered if there are not some special contraindications on account of the age or of some organic disease.

Contraindications.—No operation should be advised in children until a trial has been made of the truss. In adults past middle life with very large scrotal herniæ which have been outside the abdominal cavity for more than a year, operation is considered inadvisable, because there will probably not be room inside the abdomen for all this displaced hernia.

Strangulation.—In operating on a strangulated inguinal hernia it is well to avoid the risk of wounding the epigastric artery by cutting upwards parallel to the middle line one-fourth to one-half inch; if, however, it should be cut, the wound must be enlarged and both ends ligated. The artery generally skirts around the inner side of the internal abdominal ring and so is not apt to be wounded in the case of oblique inguinal hernia. In the case of a direct inguinal strangulated hernia this artery has been cut unawares, and death has followed.

In order to save time and to avoid accidents at such an operation, it will be advantageous to remember the arrangement of the principal coverings of the sac. An indirect hernia pushes before it a covering of peritoneum, and in passing through the canal and down into the scrotum it is further enveloped by the coverings of the cord, the fascia transversalis, and the cremasteric fascia. Most commonly the sac lies directly in front of the vessels of the spermatic cord, but if the advancing hernia enters the sheath of the spermatic cord these structures may become separated, altered in their arrangement, and so adherent that the individual layers are not easily recognized.

Properitoneal Hernia.

A properitoneal, or *interstitial* hernia is a rare variety of inguinal hernia in which the hernia occupies an abnormal position between the layers of the abdominal wall. It may occur between any of the layers, but it is most often situated between the external and internal oblique muscles, or external to the external oblique, and just beneath the skin and superficial fascia. Least often it is found between the peritoneum and the transversalis fascia. This form has been carefully studied by Krönlein.[1] It is very rare, being seldom discovered until after strangulation has occurred.

Etiology.

It occurs most often in males, and during the first year of life. In the largest number of cases it is upon the right side, thus showing a tendency to occur most frequently in that region in which defects of development in the descent of the testis are most common. It is usually congenital, and is generally associated with an ectopic testis which has been detained in the canal, or at least not far below the external ring. The hernia begins to descend through the canal until its further descent is obstructed by the undescended testicle or a hydrocele of the canal of Nuck, and then, if more pressure is applied from within the abdomen, the hernia must enlarge in the direction of the least resistance. Thus it is now forced upwards between the layers of the abdominal wall. Sometimes the hernia so increases in size that it projects downwards over Poupart's ligament. If, on the other hand, the hernia encounters a greater resistance above than below, it may begin to distend the external ring and may then descend into the scrotum.

Fig. 28.—Properitoneal Hernia.

Macready states that "in the majority of cases this hernia is in the processus vaginalis of an ectopic testis; hence there must be some causal connection." In some cases the properitoneal hernia is not of congenital but of acquired origin, and Krönlein states its cause to be mechanical, as when a poorly fitting truss allows the hernia partly to protrude under the pad into the canal above the external ring. If the pressure is now increased, the hernia is forced backwards and upwards between the layers of the abdominal wall. Again, repeated attempts to reduce a difficult hernia by ill-directed taxis (especially when the opening into the abdomen is narrow) may gradually dilate the sac just below the neck (at the internal ring). There is thus produced a pouching upwards, and so a protrusion is formed between the layers of the abdominal wall.

Diagnosis.

This hernia has its peculiar characteristics. It is generally found on the right side; it is flattened and not much raised from the abdominal surface; it is oval and parallel to Poupart's ligament; extends upwards and outwards towards the anterior superior iliac spine, and when very large may even overlap Poupart's ligament. The abdominal wall is often very thin over the tumor. The hernia

Fig. 29.—Properitoneal Hernia.

is frequently irreducible, wholly or in part, for the reason that the omentum is apt to have become adherent. If the hernia can be reduced one will generally find an undescended testicle underneath, either in the canal or at the external ring. This testicle cannot be pulled farther downward, nor can it be reduced inside the abdomen with the hernia. It is atrophic, and has usually lost its testicular feel and sensation. This side of the scrotum is empty and has not developed, while on the opposite side the testicle is considerably larger than normal.

Differential Diagnosis.

As this is an infrequent variety the diagnosis is not easy. A large psoas abscess, pointing just above Poupart's ligament, has been mistaken for a properitoneal hernia, so also has an abscess arising from the retroperitoneal glands, and again a tuberculous abscess of the abdominal wall.

Treatment.

In this variety of hernia it is best to advise operation, unless such treatment is especially contraindicated; for this hernia is very apt to become strangulated, and when strangulated it is most apt to be reduced *en masse*, so that while the tumor seems to have been completely reduced, the symptoms of strangulation remain, and if an incision be made, it will be found that the sac with its contents has been pushed either between the layers of the abdominal wall or inside the abdominal cavity, but that the constriction at the neck of the sac has not been relieved and that the intestine is still strangulated inside the sac. In case operation is contraindicated, a cup-shaped truss or an abdominal belt must be carefully adapted to the case.

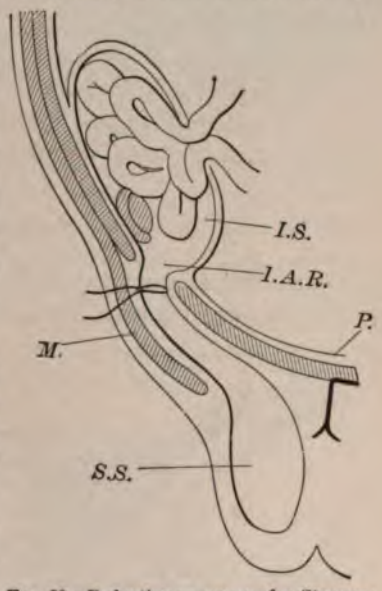

Fig. 30.—Reduction *en masse* of a Strangulated Inguinal Hernia. *I.S.*, Intraparietal sac; *I.A.R.*, internal abdominal ring; *P.*, peritoneum; *M.*, muscle; *S.S.*, scrotal sac. (After Heaton.)

At the time of operation, the treatment of the testis will depend upon its condition and the age of the patient. If the patient is not past the age of puberty, it is best to dissect the adhesions away from the cord and then anchor the testis as low down below the spine of the pubis as possible. In this case the testicle, being free from any pressure, may gradually develop its functions. If, however, the patient is past puberty, the testicle will probably have already become atrophic, and so, being unable to develop, is useless and may be removed. The wound will be closed in the usual way.

Inguino-Perineal Hernia.

This form is uncommon. In rare cases the testis, instead of descending as is normal into the scrotum, becomes deviated and is drawn downwards, backwards, and sidewise into the perineum by the pull of certain fibres of the gubernaculum testis. The scrotum on this affected side remains undeveloped, empty, and flat. The testis may be felt underneath the skin of the perineum, and it may also remain small and undeveloped. If now an inguinal hernia develops,

it will follow the course of the descent of the testis and so be found in the perineum.

In the female, if an inguinal hernia develops to any size it will descend behind the labium majus and so also be found in the perineum.

These forms of perineal hernia which emerge from the abdominal cavity through the inguinal canal must not be confused with those forms of pelvic hernia (page 326) which are found in the perineum after they have descended through the outlet of the pelvis behind the bladder and between it and the rectum or uterus. A much rarer occurrence is when the testis is drawn down into Scarpa's triangle and the hernia descends into this area. Macready collected sixteen cases.

Treatment.—This will be the same as in the other forms of inguinal hernia, mechanical or operative.

Femoral Hernia.

A femoral (*femur*, thigh) hernia escapes from the abdomen at the groin, passing beneath Poupart's ligament at the inner side of the femoral vessels. It takes a downward course through the innermost compartment of the femoral sheath till it reaches the saphenous opening, when it turns forward through this opening towards the front of the thigh and is then bent upwards towards the groin. This inner compartment is one-half inch long and is called the femoral or crural canal. The upper portion, which is called the femoral ring, is funnel shaped and large enough to admit the tip of the finger. Its size is variable and it is larger in females than in males.

Fig. 31.—Double Femoral Hernia. (Bourgery.)

At the outer side, external to the ring, is the femoral or external iliac vein. On the other three sides the ring is bounded by firm, unyielding structures; above is Poupart's ligament, behind is the hip bone, covered by the pectineus muscle, and at its inner side, the conjoined tendon and Gimbernat's ligament.

The *coverings* of a femoral hernia are as follows: the skin, superficial fascia, cribriform fascia, fascia propria of Cooper—a very thin layer made up by the union of the septum crurale and the sheath of the femoral vessels—and the peritoneum forming the sac.

Etiology.

This variety is always acquired and is never congenital. It may be due to some abnormality of Gimbernat's ligament, or to some relaxation of the same. If these tissues are lax, a long-continued intra-abdominal pressure will tend to pouch the peritoneum, and the lodgment of the bowel in this pouch will act as a wedge to dilate the canal. It is much less frequent than the inguinal form, because the canal does not normally remain open for the passage of an organ, such as the testis. It is more frequent in women, because in them the femoral ring is naturally larger and the closure of the ring is less firm. This is especially true in those who have been pregnant. It is uncommon before puberty, being rather an infirmity of adult years, since in early life there is practically no space between Gimbernat's ligament and the femoral vein, these structures being almost in contact.

Symptoms.

These are in general the same as those of the other forms of hernia—pain and swelling. When the hernia is reducible it disappears upon lying down, but returns somewhat upon standing, and is distended on coughing.

Diagnosis.

This is not always easy, and it is frequently difficult to distinguish a femoral from an inguinal hernia. In corpulent individuals, especially in stout women, one should first locate the spine of the pubis and the point of attachment of Poupart's ligament, and then trace the latter outwards to determine the exact position of the hernia. If it is below the ligament and external to the spine, it is a femoral hernia; if it is above the ligament and internal to the spine it is an inguinal hernia. In femoral hernia the external inguinal ring will be found empty while the tumor persists external to the spine and below the ligament. The sac is generally internal to the femoral vessels, but it is sometimes found external to or even behind them. The spine can generally be found by abducting the thigh, as this stretches the adductor longus and makes prominent its point of attachment to the spine. The right crural ring is said to be generally larger than

the left, and this may account for the more frequent occurrence of hernia upon this side.

The development of a femoral hernia is gradual. It appears as a small, tense, rounded swelling just below Poupart's ligament. Its upper surface touches the ligament and may sometimes turn upward in front of the ligament, but by manipulation it can be pressed

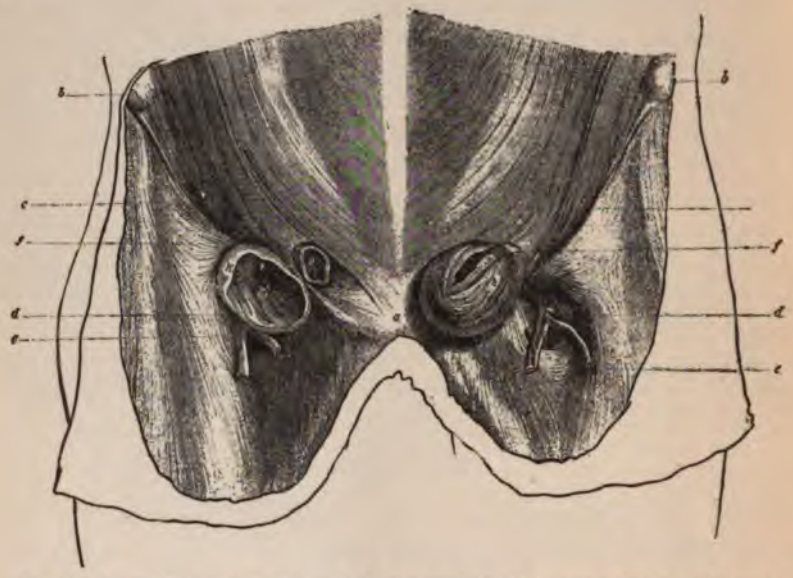

Fig. 32.—Dissection Showing Unilateral Femoral and Double Inguinal Hernia. (Cooper.)

downward. When the hernia is in the canal it is very small, but after it protrudes from the saphenous opening it enlarges, yet rarely becomes larger than a hen's egg.

Differential Diagnosis.

In general, tumors other than femoral hernia are rare in this immediate area. However, the following may occasionally be found.

Adenitis.—The enlarged gland is more superficial, while the hernia is deep seated, if small. The gland is spindle-shaped and more movable from side to side, and there are frequently other smaller glands in the neighborhood. In rare cases they may become inflamed (a suppurating bubo) and so simulate a strangulated hernia. These glands may be situated between the sac and the skin, or at the outer side of the hernia. There is no impulse on coughing In many cases the enlargement of the glands is secondary to a new growth in

the neighborhood (cancer of the rectum, sarcoma of the femur), and when this is found the diagnosis is more easily confirmed.

Lipoma.—This gives the sensation not of a fluid, but of a solid. It is irreducible, and gives no impulse on coughing. It is apt to be lobulated.

Varix of the Saphenous Vein.—This condition is found quite often and is accompanied by varicose veins lower down in the calf of the leg. It is usually about the size of a pigeon's egg, very soft and compressible, and when it is reduced and pressure is made on the femoral vein, the tumor fills up from below. Upon coughing there is a sensation felt by the finger somewhat like an impulse, but upon closer observation it is found that it is not a regular impulse, but more of a thrill, as the blood is forced down underneath and past the tip of the finger.

Psoas Abscess.—It occasionally happens in cases of disease of the lower vertebræ, when there is abscess formation, that the pus gravitates downwards underneath the iliac fascia and points underneath Poupart's ligament in the region of the femoral opening. This condition simulates some of the symptoms of a femoral hernia, for, upon coughing, an impulse is felt, and upon lying down the tumor recedes. It is, however, generally external to the femoral vessels, and may often be felt to be continuous with a tumor above Poupart's ligament. Fluctuation may also be made out, and upon thorough examination a hump or knuckle may be found on the back over the region of the diseased vertebræ. The patient may also give a tuberculous history.

Hydrocele of the sac occurs in a few cases in which the omentum has come down and has become irreducible, while at the same time the opening at the neck of the sac has been obliterated. The serous exudation accumulates and the hydrocele is formed. In this case there is no impulse, neither can the tumor diminish in size.

Cysts are sometimes, though rarely, developed in this region. Malignant growths arising from the fascia or from the bones have been occasionally reported, but their hardness and irregularity of outline and the history of their development would differentiate them from a hernia.

Treatment.

This is more difficult, and will probably be less satisfactory than in inguinal cases. Especial care is needed to see that the entire tumor is reduced, and in order to accomplish this best the thigh should be flexed, adducted, and rotated inwards. This will relax the

margin of the saphenous opening. If the hernia is difficult to reduce, taxis may be employed, and this should be very brief and gentle, for the edge of the ring is so sharp that the bowel is very readily injured. As a rule, reduction here is more difficult than in inguinal cases, because the ring is smaller and the canal is narrower, and also from the fact that the protruded portion is at right angles to its neck. Therefore one should press backwards before pressing upwards. Strangulation is much more apt to occur in these cases, because of the conditions described, since the opening is so small and the borders of the ring are so unyielding.

Mechanical Treatment.

In the femoral variety the French truss (Fig. 27), the one which is applied from the affected side, is probably the most satisfactory. If a truss similar to that shown in Fig. 25 or 26 be used, the shank must be longer, and it must be curved more towards the body. The pad should lie below the fold of the groin and should press backwards and upwards. The spring should be as light as consistent, and the pad quite small, for if it is large it must remain in the fold between the body and the thigh and cause pain when the patient flexes the leg in going upstairs or in sitting down.

In cases of temporary irreducibility the same treatment may be employed as in inguinal cases: the application of a spica bandage over a pad or a specially made cup truss as suggested by Macready.

The *prognosis* for a cure by wearing a truss to retain a femoral hernia is bad. Even those who believe that a truss may cure an inguinal hernia have very little hope for such a result in femoral cases. So while a truss may be advised as a support, still no cure can be expected. In a word, a femoral hernia is much more difficult to hold by a truss; very firm pressure cannot be applied on account of the femoral vein; the truss is always more or less uncomfortable and the hernia is always in greater danger of becoming strangulated. For these reasons an operation is advised in all suitable cases, in patients under forty years of age, in which it is not especially contraindicated. It is indicated in all irreducible cases, or whenever there has been a previous history of irreducibility or of strangulation. In non-strangulated cases the mortality is less than one per cent.

Operative Treatment.

One of the best of the various operations is that of Bassini.[5] An incision is made parallel with Poupart's ligament, and it should be

long enough to afford plenty of room for the deep dissection. Poupart's ligament is next clearly outlined and brought into view to serve as a landmark. The sac is clearly dissected away from the canal, pulled down, and freed from all adhesions high up about the ring. It is opened, and, if there is any adherent or irreducible omentum, this is liberated, and, after carefully ligating each bleeding point it is reduced into the abdomen. As a rule, there is not a large amount of omentum present, but if there is it is generally partly irreducible. If there are other structures present in the sac they are all reduced. The sac is now transfixed, ligated, and cut away, and it should at once retract high up through the ring. The ring and canal are now to be completely closed. Bassini employs three silk sutures to unite Poupart's ligament to the pectineal fascia. The first is placed near the spine of the pubis, the second 2½ cm. externally, the third 1 cm. from the femoral vein. These are not to be tied until three or four others have been inserted, first through the edge of the falciform fascia and then through the pectineal fascia, the lower suture entering just above the saphenous vein. The upper sutures, which draw Poupart's ligament backwards to the pectineal line, are then fastened. The other sutures which bring together the anterior and posterior walls of the canal are next tied. The skin incision is closed without drainage.

Marcy and Cushing follow in a measure the "purse-string suture" of Wood, who advised that the needle be passed through Poupart's ligament, the pubic fascia lata, and Gimbernat's ligament, then horizontally through the pubic fascia lata just below its insertion into the pectineal line; it is then tied. A second or third similar suture may be passed about a quarter of an inch below. Marcy prefers kangaroo tendon for the deep sutures, and the writer believes that it is better not to use silk or silver wire, but to employ a suture (such as kangaroo tendon or chromicized catgut) which will remain solid for about sixty days and then become absorbed. In many cases it may be better to make a vertical or oblique incision over the long axis of the hernia rather than the transverse incision of Bassini.

After the incision has been made, the bleeding points must be clamped, for the field of operation must be kept very clean and dry. Sometimes it may seem as if the hernia was immediately underneath the skin, but careful dissection demonstrates a layer of lymphatic glands between the sac and the skin. Again, there may often be found a hard, rounded mass of subperitoneal fat tissue covered with fascia and simulating the hernial sac with its contents. In a few cases cysts have been found between the hernia and the skin. Their

origin is uncertain; some have thought they were diverticula from the hernial sac, as in some cases they communicated with it by a small opening; but in other cases they have been free and independent. They may be single or multiple, and, when incised, they pour out a thin yellow serum; the true hernial sac will be found more deeply situated.

Strangulation.—In case of strangulation it is best to make an oblique incision in the direction of the long axis of the hernia. The starting-point should be about an inch above Poupart's ligament in the neighborhood of the spine of the pubis, and it should be sufficiently long for a very free and rapid dissection. If the patient is old and weak and it is not desirable to give ether, the incision can easily be made without pain under a two- or four-per-cent. solution of cocaine; then the only pain which will be felt will be that caused by the compression and ligation of the severed arteries, and which is probably due to the pressure upon the sympathetic nerve fibres in the arterial coat.

It will not be necessary to distinguish all of the layers over the hernia, but the incision must be carefully made in order that the sac may be recognized before it is opened, for the bowel might be adherent to the sac wall immediately underneath the line of incision, and the knife would then open the intestine. The interior of the sac will easily be recognized by its smooth, glistening surface. It should be split from end to end so that the neck can readily be inspected at its inner side, for it is here that the sharp edge of Gimbernat's ligament forms the usual seat of constriction. This can be recognized by passing the tip of the finger along the surface of the hernia upwards towards the spine of the pubis; in some cases it may be possible to insinuate the tip between the hernia and the ligament, but in many this will be impossible. The knife is carefully passed along the finger as a director, with its sharp edge directed towards the ligament, and the ligament is nicked by the tip of the knife. Scarpa advised that Gimbernat's ligament be nicked but not incised, and Hyrtl recommended that this be done not by drawing the knife across the fibres, but by pressing the tip of the knife against them. An attempt should now be made to stretch the ring, but if it does not yield sufficiently the knife is reinserted to cut inwards and upwards, care being taken not to go too far upwards in the male, as the spermatic cord, which is just above Poupart's ligament, might be injured. The location of the vessels is of great importance, and care must be taken to avoid the obturator artery if present. (The obturator usually arises from the hypogastric, but exceptionally may be given off from the epigastric or femoral.) The epigastric artery is close to the ring, lying above its outer border;

frequently an aberrant obturator artery descends into the pelvis at the outer side of the ring, or immediately behind it, and sometimes it passes over the ring and descends, curving along down the inner border of the ring and the margin of Gimbernat's ligament on its way to the obturator foramen. When this arrangement exists it is almost impossible to avoid wounding the artery, for the vessels surround the ring on three sides, the fourth side being the bone. If an artery is injured, the incisions must be so enlarged that the bleeding point can be secured. (It is said that in fifty per cent. of women the obturator artery is derived from the epigastric branch of the external iliac.)

After the bleeding has been stopped the sac can be opened and the contents treated in the manner described above. If the patient is in a satisfactory condition one may proceed with the radical operation for a cure, but, as a rule, the femoral cases are so much more severe than the inguinal that the primary operation must be as brief as possible.

Cæcal Hernia.

By this term is understood a hernia in which a part of the cæcum has descended, and this variety, though infrequent, still occurs quite often enough to demand one's attention. If it exists and is not recognized by the physician, it may become later a source of embarrassment in regard to its prognosis. Usually the hernia consists not alone of a part of the cæcum, but also of the vermiform appendix,[6] or a portion of the ileum may be included. Macready reported fifty-one cases, and among these thirty-six were of the right inguinal type. Brieger[7] tabulated forty-one cases in which the vermiform appendix was found to complicate the cæcal hernia.

Etiology.

As a rule, these cases are of congenital origin and are due to the fact that in the embryo there sometimes persists a peritoneal fold (the plica vascularis) which extends from the testis upwards to the cæcum, appendix, or colon. This fold contains some fibres of unstriped muscle, and if these persist and later contract, the cæcum or appendix is drawn down into the scrotum. When it occurs as an acquired condition, it is probably due to the fact that whenever the mesentery of the cæcum is so long that the latter is very freely movable, it may be dragged down into the sac by the agency or the pull of a portion of the ileum, which is quite commonly found in most cases of large herniæ.

Some writers have considered that the posterior portion of the cæcum was not enclosed in the peritoneum, so that when the cæcum descended it was behind, and not inside a regular peritoneal sac. Roser* gives a diagram of a case in which a portion of the cæcum was uncovered by the peritoneum and was behind the true sac. Bardeleben has studied this question quite thoroughly, and as the result of one hundred and sixty dissections concluded that "the cæcum, that is, the part below the ileocæcal valve, is as a rule surrounded completely by peritoneum, possesses a short mesentery, and lies so freely movable in the belly that it can be raised upwards and moved from side to side. A cæcal hernia always possesses a peritoneal sac." Treves has verified these conclusions from his own investigations, and Macready states that "the cæcum in a hernia will possess a perfect sac on the left side always, and on the right side almost always."

Fig. 33.—Cæcal Hernia with the Appendix. (Roser.)

Diagnosis.

When the hernia is reducible the symptoms are about the same as in the other forms of herniæ. Cæcal hernia occurs most frequently upon the right side and is more often inguinal than femoral. It is not possible to distinguish the cæcum itself unless the appendix is also in the sac and can be traced up to its origin in the head of the cæcum. In children, and in older patients who are very thin and spare, it is quite easy to detect the appendix through the thin tissues.

Treatment.

This will be the same as that described for the other forms of inguinal and femoral hernia. If an operation is indicated, one should be on the look-out for the exceptional cases in which the cæcum is not covered with the peritoneum, and here by a careful dissection the bowel may escape an injury. In Brieger's table of twenty-three operations for cæcal hernia complicated by the presence of the appendix,

the latter was found to be normal in only four cases. The appendix must not be returned unless it is surely normal. If it is wrapped up in the omentum, or so adherent that it cannot be well separated, it is best to resect it high up near its insertion in the bowel and bury the stump with Lembert silk sutures.

Sigmoid Hernia.

This variety is rare, but when it exists it may be, like cæcal hernia, either congenital or acquired, and also may be due to similar causes, for there is a similar fold of peritoneum (the plica vascularis) which connects the sigmoid flexure with the left testis in fœtal life. If this persists, it may pull the sigmoid downwards towards the testis in the scrotum, especially in those cases in which the bowel has a long mesentery.

Diagnosis.

This is most difficult unless the tissues are very thin and the appendices epiploicæ can be recognized. The rectal tube cannot be inserted to the normal distance, nor can more than a few ounces of fluid be injected.

The *treatment* will be similar to that of cæcal hernia.

Vesical Hernia.

A portion of the bladder wall may protrude through any adjacent hernial ring (the inguinal, femoral, or ventral) and the intra- and extra-pelvic portions may thus communicate by a narrow isthmus which passes through the hernial canal. This variety is of rare occurrence and happens more often in elderly men with larger hernia than in any other class. It is seldom diagnosed except at operation, and even then only after it has been incised. Cases have been more often reported within the last few years because there have been more operations for the cure of hernia, and also because the operators have made more extensive dissections.

Anatomy.

These cases may be grouped under three heads; first, those which are extraperitoneal; second, those which are intraperitoneal, and third, those in which both forms are combined.

The most common variety consists of a prolapse of a portion of

the bladder wall which is entirely extraperitoneal; here the hernial sac generally contains also omentum and bowel. The next most frequent variety is the combined form in which both intra- and extraperitoneal portions of the bladder are involved; and the rarest of all is the true intraperitoneal variety.

The bladder has been found more often in femoral than in inguinal hernia, and this may be explained by the fact that most cases of hernia of the bladder are to be found associated with strangulated hernia, and also that the majority of cases of strangulation are of the femoral variety.

Etiology.

In most cases the bladder has been found larger than normal. In elderly men who have had frequent attacks of retention of urine the bladder walls are so stretched that they become lax and yielding, and then bulging may occur laterally, adjacent to a hernial opening; later the bladder wall (just as any other viscus when it is empty and lax) may pouch into and descend through the hernial ring. Again, the bladder may have a diverticulum or be abnormally displaced, or the hernial ring may be unusually large. The bladder wall may be pulled down by the contraction of a fold of peritoneum running down to the hernial sac, similar to what occurs in the case of a cæcal or sigmoid hernia. This tendency may be increased by pulling down the sac for a high ligation of its neck in performing the radical operation, and it has been found in several cases in which relapse had occurred that the bladder wall had been pulled still farther down into the sac.

In the *intraperitoneal* variety a portion of the bladder wall, providing it is lax and movable enough, may prolapse the same as any other organ.

Symptoms.

These are vague, and when recognized are connected with difficulties in urination. The patient may have noticed that a tumor, which was present before urination, diminished or disappeared afterwards, and that pressure upon this tumor caused the desire to urinate.

Diagnosis.

This has rarely been made before operation. At operation one may sometimes find a small, flat, doughy tumor, a smooth yellow, tongue-shaped, flattened mass, feeling like a thickened sac or a piece of adherent omentum, also an unusual quantity of yellowish fat sur-

rounding the hernia and adherent to the bladder. The bladder wall is frequently so very thin that it easily simulates the hernial sac. Sometimes it may be traced back into the abdomen, and further assistance may be furnished by passing a sound through the urethra into the bladder and into the suspected pouch. One may also inject some colored fluid through the urethra into the bladder and watch for its appearance at the suspected wound. Fenger[9] writes, "to avoid opening the bladder, think of the bladder while operating."

Treatment.

The opening into the bladder (whether incised, lacerated, or punctured) should be at once carefully closed with fine silk sutures. The Lembert method should be used. The sutures should not penetrate the mucous membrane, and should be about ten to the inch; this first row of sutures is to be reinforced by one or two extra rows, each placed a little farther outside. A strip of iodoform gauze should lead from the sutured area of the bladder out through the wound, so that in case there should be any leaking of urine this drain would lead it outside. If there is no leaking, the gauze can be withdrawn after forty-eight hours. The external wound can then be closed except just at the lower end, where the gauze comes out. If a radical operation is performd it is best to follow the advice of Curtis[10] to employ the method of Macewen, rather than that of Bassini.

After the operation the bladder must not be allowed to become distended, but should be kept as empty as possible. It is better to pass the catheter every three hours for the following two days, rather than to leave the catheter in the urethra for continuous drainage.

The *mortality* in these cases is high, because they occur generally in elderly patients and often are complicated by strangulation.

Partial Enterocele.

The term partial enterocele, or Littré's hernia, is applied when there is a protrusion of a portion of the circumference of the intestinal wall. It may be just a nipple-like protrusion, a rounded oval prominence, or it may be a congenital pouch as in the case of Meckel's diverticulum.

The formation of the hernia is as follows: That portion of the bowel wall which is immediately over or behind the ring is forced as a pouch into the ring by some suddenly increased intra-abdominal pressure, and it is not the entire section of the bowel, but only a small portion of its circumference.

This formation is most apt to occur in a ring which is small and has firm edges, and where there is only a small space into which the hernia may protrude. Hence the largest number of cases occur at the femoral or obturator rings. The portion of the intestinal wall which has been nicked into the ring generally comes from the free edge, opposite the mesentery.

Fig. 34.—Littré's Hernia. (Treves.)

Symptoms.—These are those of incomplete strangulation. Pain is colicky in character, and, while it is referred to the region of the hernia, on inspection no hernia is generally recognized. Vomiting is usually constant and does not cease so long as the strangulation persists. Constipation may yield to treatment because the entire lumen of the intestine is not obstructed. Gas may also escape, but the local pain continues without the presence of any tumor, and for this reason the seriousness of the case is seldom recognized until perforation has occurred. When strangulation exists, ulceration with loss of the mucous membrane (corresponding with the area constricted) may occur, and if this is prolonged all the coats may be destroyed so that perforation follows. These cases are rare, and they seldom occur in infancy, but more often in old age.

Treatment.—The constriction must be relieved by an immediate operation, and the further treatment will be the same as that indicated in the section on strangulated hernia. It should be remembered that the constricted pouch has a special tendency to retract at once after the constriction is relieved, so it must be most carefully examined as to its vitality.

Umbilical Hernia.

This variety may be either congenital or acquired.

Congenital Umbilical Hernia.

This term designates a hernia which exists before the cord falls off. It is quite rare, and its origin may be explained as follows:

Etiology.

In the embryo the intestines are at first outside the abdominal cavity until about the third month, when they begin to enter and the anterior abdominal wall gradually closes over them to meet at the

linea alba. In some cases there is a defective development, so that a complete closure does not result, and an opening may remain at the umbilicus. This opening may be minute, in which case there is a slight bulging into the base of the cord, or it may be just large enough to admit a loop of bowel; or there may be a very large defect in the wall—an eventration—as is sometimes seen in monsters in which the cavity and the hernia are covered merely by the fœtal membranes—the sac wall—which are continuous at the edge of the opening with the adjacent skin.

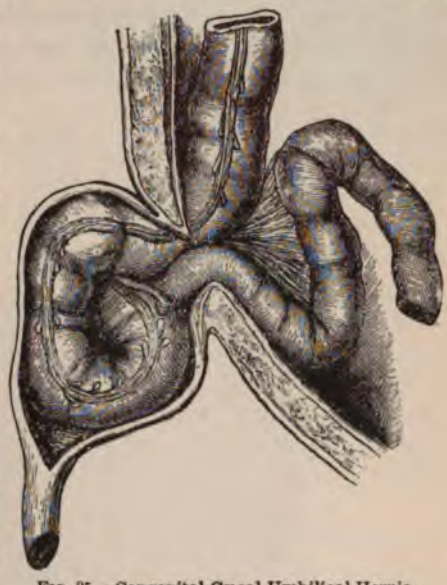

FIG. 35.—Congenital Cæcal Umbilical Hernia. (Albert.)

TREATMENT.

This may be mechanical or operative. If the opening is very small it may be covered with a pad and held in position by strapping: but when the opening is large, the soft, thin coverings will not endure the pressure of a pad, and suppuration is apt to develop and terminate in a fatal peritonitis. If the condition of the child is good an operation may be performed, but this has rarely been successful.

The *prognosis* is favorable only when the opening is small.

Acquired Umbilical Hernia.

ETIOLOGY.

The linea alba is formed by the decussation of the fibres of the oblique and transversalis muscles. As the fibres of one side cross over to join with those of the other side, there are sometimes left small elliptical gaps or interspaces, which close later. The umbilical is the largest gap and the one latest to close. As this opening is not so firmly nor so completely closed in the infant as in the adult, it is much more apt to stretch and yield in the former, and therefore the largest number of cases of umbilical hernia occur during the first year of life. The acquired form develops after the umbilicus has once been closed and after the cord has fallen off. The majority of

cases develop during the first year and are about equally divided between males and females up to the fifth year; from then until twenty years they rarely develop. Some which had developed earlier and were so small as to escape attention may, however, be noticed for the first time during this period, although many which had developed earlier became cured before puberty. The next period of frequency is between thirty-five and fifty years. Seventy-three per cent. occur in females; twenty-seven per cent. in males. The exciting cause is an increase in the intra-abdominal pressure. In infancy the common causes are crying, constipation and straining, whooping-cough, etc. In the adult, severe strains; in men, hard lifting, obesity with large pendulous abdomen, sometimes the presence of ascites which distends the abdomen and so stretches all the hernial openings; in women, frequent and difficult pregnancies, the pressure of ovarian and other tumors, straining.

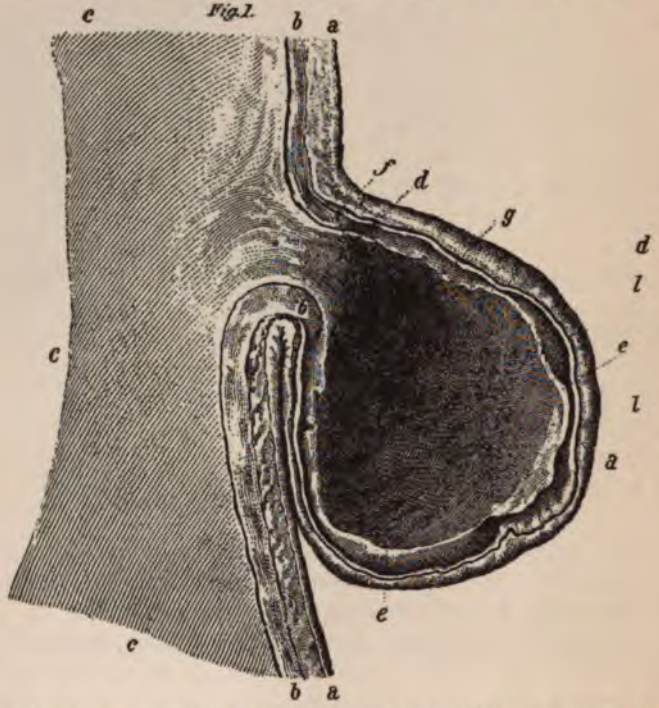

FIG. 36.—Umbilical Hernia. *a*, *a*, *a*, Integuments; *b*, *b*, abdominal muscles; *c*, *c*, *c*, peritoneum; *d*, mouth of the hernial sac; *e*, *e*, hernial sac; *f*, termination of the linea alba around the mouth of the sac; *g*, fascial layer between the integuments and the sac. (Cooper.)

Symptoms.

The onset is usually gradual rather than sudden, unless it is acquired at the moment of some great violence. Symptoms which may be referred to the stomach are more apt to be present here than in the other varieties of hernia, but the general symptoms are the same as those of the other forms.

Diagnosis.

This hernia is always direct. In the child it has more of a conical shape, "like the finger of a glove." It always contains intestine, seldom omentum, and is generally easily reduced. There is scarcely any canal, and the opening is almost always circular, with a sharp thin edge. In the adult, the protrusion is more hemispherical in outline and generally contracted at its neck; it contains omentum and frequently small intestine. If the hernia has existed for some time it will undoubtedly have been irritated and inflamed, and so there are generally some adhesions which render it more or less irreducible. In some cases in which a belt or truss has been worn, the skin will have been irritated and a slight ulceration may exist.

Occasionally in the adult a lipoma may be present and simulate a hernia, but it is generally harder, more mobile, and situated a little above or below the umbilicus and at one side of the median line.

Prognosis.

In young children this is good. In a large number of cases a spontaneous cure results. Strangulation is of rare occurrence. In the adult a spontaneous cure rarely occurs. It is not common for a cure to follow the use of a belt, although, if the hernia be kept always reduced and the opening constantly closed, marked relief will usually follow. In those cases in which the protrusion is very large (the size of an infant's head or more) reduction is painful, is generally difficult, and is often impossible. There is a constant tendency for the hernia to become irritated, inflamed, and more or less irreducible; obstruction and strangulation not uncommonly follow.

Treatment.

Mechanical Treatment.

In *children* it is well to begin with the mechanical method, for it has been uniformly successful and at the same time is free from danger to the patient. Macready advises this method, and believes that

a cure can be promised in nearly every case. A strip of adhesive plaster, two inches wide and long enough to encircle the child, is laid upon the table. The child is then placed upon its back upon the middle of this plaster. The two ends are brought forward around the child and over the pad, which has been placed over the umbilicus. In this case the pad cannot slip, whereas when the plaster is put on in front and the ends are carried backwards it is more apt to slip. The pad must not be too conical, but rather elliptical. The band must be kept firm and is to be changed every ten days. If there is any excoriation it should be carefully treated. In some cases a belt may be advised rather than the strap, but only when very careful daily attention will be given to keep the pad and belt exactly in place; as it does not stick to the skin, it slips about and very frequently leaves the hernia unprotected, so that it is not to be advised except with caution. Trusses are not to be employed in these conditions. In favorable cases a cure may be expected in about eighteen months, but sometimes the treatment is continued for several years without success. If it has been carefully carried out, and the hernia persists at the age of puberty, it is advisable to consider the radical operation, which, if performed at this age, is quite free from danger and reasonably sure to be successful.

In the *adult* the treatment may be adapted either to relieve the conditions by mechanical means or to attempt to cure the hernia by the operative method. The best palliative measure is the use of a well-made abdominal belt with a suitable pad which will support the heavy abdomen and thoroughly retain the hernia. This belt is generally better tolerated than a stiff truss, but each may have its own individual advantages in special cases. If the hernia is small, reducible, and manageable, the belt will probably succeed in relieving the symptoms, but, of course, will not produce a cure. In old, long-standing cases, in which the abdominal walls are very thick and the neck is small, it is most difficult to retain the hernia in the abdomen, as a certain amount of the omentum will slip down into the canal and lie under the truss. The hernia may now become inflamed and the resulting adhesions will

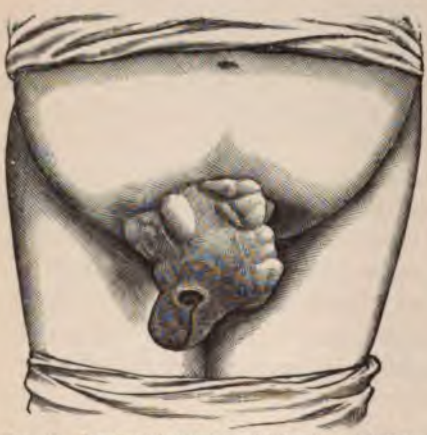

FIG. 37.—Irreducible Umbilical Hernia, with Excoriation over the Sac.

render it more or less irreducible. In order to care for this condition a large, concave pad, formed to fit over the mass, must be worn inside the belt. Considerable pressure is required, so that it occasionally happens that the friction of the pad over the skin, which is often moist with perspiration, produces excoriation and ulceration. This in turn renders the wearing of the belt impossible for several days, and, unless the patient can remain in bed, the hernia still further increases.

As it is believed that in adults, with a moderate-sized umbilical hernia, the belt cannot cure the hernia, and that, as time advances, there is a constant tendency for the conditions to become more complicated—because the hernia may become larger or adhesions may form and render it partly irreducible—so in suitable cases it may be well to consider the question of operation. There are many cases, however, which are unsuitable on account of their age and condition, and in these the best that can be done is to make the patients as comfortable as possible with suitable belts.

Operative Treatment.

The dangers which are associated with the operation may be due to the prolonged anæsthesia which is required, to the difficulties of dealing with the mass of firm adhesions, to the fact that the enormous hernia has been so long outside the abdominal cavity that there is no longer room inside, and to failure to secure primary union, owing to the thick layer of subcutaneous fat. While a radical cure cannot be expected so confidently in the umbilical as in the inguinal or femoral varieties of hernia, still the patient will be relieved from the danger of irreducibility and of strangulation; also a much easier belt can be worn.

A large elliptical incision is made to excise the surplus skin about the umbilicus, and it is carried down through the superficial and deep fascia and the subserous tissue to the peritoneum at the mouth of the opening; the thin sharp edges of the ring are excised and the sheaths of the recti muscles are opened. The peritoneum is lifted up between two pairs of forceps and carefully incised. A director is introduced and the sac is opened; if there are any adhesions they are gently separated and ligated. If there is a large amount of omentum in the sac it is transfixed and ligated in small bundles. Care is to be exercised that the stump is entirely free and not adherent at the edges of the ring. If it is necessary to enlarge the opening it should be enlarged upwards, and not downwards, for there is a greater tendency for the hernia to relapse if the lower margin of the opening is incised. If the bowel protrudes, it must be reduced by taxis; in some cases the walls are so lax that this may be difficult,

but some assistance may be gained by raising the borders of t opening with blunt hooks or retractors. After reduction, the ed of the peritoneum are brought together in the median line with f catgut sutures; this produces less puckering and pouching of peritoneum than when the opening is closed by a single ligat through all the layers. Silkworm-gut sutures are next passed throu all the tissues except the peritoneum, but are left untied until other sutures have been inserted; these are passed about three-qu ters of an inch apart. The edges of the muscles are then clea separated from the fascia, and are brought well together with str kangaroo tendon interrupted sutures. Next the edges of the apon rotic fascia are united with finer strands of kangaroo tendon. T silkworm-gut sutures are now drawn tight and tied. If the edges the skin gape, fine catgut sutures may be used. No drainage is e ployed. The wound is covered with sterile gauze, and straps of hesive plaster two inches wide and about eighteen inches long passed over and across the abdomen, to support the walls and lessen the tension on the sutures. An abdominal swathe is put over the dressing, which is left untouched until the tenth day, wh the silkworm-gut sutures are removed. The patient remains in b about three weeks. An abdominal belt is worn in all cases for year, and, when there is reason to fear a relapse, its use is continu

Ventral Hernia.

This term is applied to a hernia traversing the anterior abdomi wall at any point other than the groin or umbilicus.

Etiology.

It may be due to defects of development when the hernia protru through the lax spaces between the aponeurotic fibres. It occ most frequently in the linea alba, midway between the umbilicus the ensiform cartilage, or in the linea semilunaris, or in certain w gaps between the iliac crest and the false ribs. It may occur a sequence to an operation for an abdominal tumor, appendicitis, This is especially the case when the entire incision has not healed primary union; the weak cicatrix yields, bulging occurs, and a p trusion follows. It may be due to traumatism, a stab wound, rupture of muscular fibres, or the spontaneous opening of an scess.

The exciting cause is an increased abdominal pressure from a cause occurring in connection with the presence of a weakened a

in the abdominal walls. It occurs most commonly between thirty-five and fifty years, and somewhat more frequently in females than in males.

Symptoms.

These are not generally recognized until after they have existed some time. The patient complains of an indefinite burning or tearing pain in the abdomen upon sneezing or coughing, or upon violent exertion. At first the pain is not localized; later, as it increases, he finds a small protrusion on exertion, and there is localized pain. If hernia is due to a defect of development it is usually small, round,

Fig. 38.—Ventral Hernia Following Incision for Appendicitis. Side view. (Bull.)

Fig. 39.—Ventral Hernia Following Incision for Appendicitis. Front view. (Bull.)

and reducible; while if due to operation it will be larger, and there will be a broader area of bulging along the line of the cicatrix. This will be stretched out, and often so thin that the outline of the intestines can be recognized. They may be adherent to the skin. If due to the first cause, the ring will be firmer and smaller, whereas when due to operation the opening is less sharply outlined and is considerably larger.

The contents may consist of the intestine and omentum, or of anything else which could be protruded. A hernia in the linea alba is apt to contain masses of subperitoneal fat.

Separation of the Recti.—This is a variety of ventral hernia, and while found occasionally in children, it most frequently occurs in women over thirty-five years of age who have had numerous and frequently difficult labors. It occurs between the umbilicus and the symphysis pubis.

Prognosis.

When the hernia is reducible, with constant and careful attenti[on] and by the use of a suitable belt, the larger number of patients m[ay] expect relief, and that the hernia will not become much larger or ir[re]ducible and strangulated; this immunity is due to the large size [of] the opening and to the neck not being subjected to constriction. [If] however, these cases are neglected, the hernia will become very lar[ge] and painful and may endanger the life of the patient.

Treatment.

This is very similar to that advised for the umbilical variet[y]. A well-fitting belt, with a suitable pad, can be recommended for t[he] majority, while for the younger or for those in whom there are no contraindications, an operation will probably offer the best opport[u]nity for a complete cure.

Lumbar Hernia.

This variety is very rare, and is more apt to occur as a sequen[ce] to an abscess (for which it is sometimes mistaken) or to an inju[ry] than to develop spontaneousl[y]. The hernia protrudes through t[he] triangle of Petit, which is situat[ed] above the iliac crest and is limit[ed] anteriorly by the border of the e[x]ternal oblique, posteriorly by t[he] border of the latissimus dorsi mu[s]cles. As this space is larger in t[he] adult than in the child, so this for[m] may be expected to occur more oft[en] later in life.

Fig. 40.—Congenital Lumbar Hernia. (Bull.)

Treatment.

A special truss or belt must b[e] devised to encircle the body so a[s] to apply pressure over this area. If not especially contraindicated a radical operation, as in umbilical hernia, might be undertaken.

Internal Hernia.[11]

This term includes in general all cases of hernia in which the pro[-] trusion occurs through an opening in the interior of the body. The principal varieties are diaphragmatic and retroperitoneal. More

rarely the protrusion may occur through the foramen of Winslow, or through a defect or tear in the mesentery or in the peritoneum.

Diaphragmatic Hernia.

In this rare variety the hernia protrudes through an opening in the diaphragm into the pleural cavity. It may be either congenital or acquired.

The *congenital* cases are uncommon, but it sometimes happens that a child is born with a large congenital defect in the diaphragm through which varied portions of the abdominal viscera have protruded into the pleura. These children usually die.

The *acquired* cases are due to some traumatism, as a fall, or some great violence. The opening is generally on the left side, for the right side is protected by the liver. In the majority of cases there is no sac; Tillman collected 279 cases, and in 248 the sac was absent. The hernia consists of small intestine and some portion of the stomach wall, though at times other organs may be included. This hernia is seldom recognized before death; in Leichtenstern's 250 cases only 5 were diagnosed.

If the diagnosis can be established, a median incision is to be made below the xiphoid cartilage, and the hernia is to be reduced if possible, the opening in the diaphragm being then sutured. The prognosis is unfavorable.

Retroperitoneal Hernia.[12]

This variety is often designated "*hernia Treitzii*" in honor of Treitz, who was the first one to collect a number of these cases and to accurately describe them; he reported eight cases between 1847 and 1854. To conform to his definition a "hernia Treitzii" must consist of a sac with an opening, and containing a portion of the intestine. This sac must be formed of peritoneum and be buried in true retroperitoneal connective tissue. The hernia may occur when the peritoneum forms a groove or furrow, the hernia pushing its way onwards and forcing the fold of peritoneum forming the fossa before it, generally in one of the following localities. The most frequent and important is the *fossa duodenojejunalis*, which is situated in the left hypochondriac region, posterior to the point where the duodenum passes into the jejunum. It is bounded above by the pancreas, on the left by the kidney, and on the right by the aorta. There have been sixty-six cases of this variety reported. The second most frequent locality is the region of the cæcum, the *fossa subcœcalis*. The third, the *fossa intersigmoidea*, is in the left iliac region. The hernia occurs between

the tissues of the mesocolon of the sigmoid flexure. This is the rarest form, and only three cases have been reported.

Symptoms.—These are of acute strangulation, obstruction, distention, severe pain, vomiting, and collapse. The *diagnosis* has never been made during life, with but a single exception, the case of Staudenmayer. This patient died.

FIG. 41.—Hernia into the Fossa Duodenojejunalis. The hernial orifice is displaced to near the cæcum. The colon has been drawn aside to show the sac which contains all the small intestines. (After Treitz.)

Treatment.—The patient should be operated upon as soon as a diagnosis is made.

Pelvic Hernia.

Under this term may be grouped the different varieties of hernia which protrude through the lower outlet of the pelvis—between the symphysis in front and the coccyx behind.

ETIOLOGY.

The causation is not definitely known, but it is supposed to be due to the gaps or defects in the levator ani muscle or its fascia; or, perhaps, more often it is of congenital origin in cases in which the low attachment of the peritoneum about the fossa of Douglas produces an exceptionally deep pocket between the rectum and bladder or uterus. This form of hernia is rare, owing to the marked elasticity and resistance of the muscular floor. (As a rule, herniæ are uncommon through muscular planes.)

The cases may be classified according to the position at which they emerge at the surface. In man they may be present in the *perineum* or in the *ischiorectal* space. In woman they may occupy the *labium majus*, the *vagina*, or the *ischiorectal fossa*. The hernia generally protrudes at the side, and not in the middle line of the perineum, or it may protrude into the vagina through an opening high up in the lateral or posterior wall.

They are more apt to occur in adults than earlier, and more often in females than in males. Macready found forty cases, thirty-four in females, and six in males. In men the *perineal* form is most often found, while in women it is the *labial* form. Here it comes down usually on the right side along and outside the lateral wall of the vagina, where it may be felt, and emerges in the posterior portion of the labium majus. It has been mistaken for a large cyst, and has also been incised for an abscess. When it protrudes into the vagina it is pedunculated in form, and so may be mistaken for a polypus.

Symptoms.

These are the same as in other forms. There may also be some difficulty in walking, in urination, or in defecation; local pain, or a sense of weight in the perineum. Colics are quite apt to occur.

Treatment.

Reduction with careful taxis is generally easy. If the opening is high up it may be well to introduce the fingers in the rectum or vagina to compress the sac. In some cases it will be advantageous to place the patient in the knee-elbow position.

A pessary can usually be inserted to retain the hernia. It is sometimes more satisfactory to employ a pad, which can be held in position by a perineal band, which in turn is supported by a belt around the body above the hips.

Strangulation is rare, but the possibility of it should be kept in mind in old cases of prolapse. If it should be found, care must be taken to avoid the bladder, which is quite often prolapsed.

Rectal Hernia.

This variety is so rare that it is not usually described, but it may be defined as a protrusion of a portion of the pelvic contents through the anus included in a pouch formed by eversion and prolapse of the rectum, the innermost covering of the hernia being the peritoneal coat of the rectum.

Diagnosis.—It must be distinguished from a simple prolapse of the rectum by the impulse on coughing and by the feel of the tissues when the hernia is reducible.

Treatment.—The simplest method is to support the hernia with a suitable pad held up by a perineal band. If this is unsuccessful, an operation should be performed, unless it is especially contraindicated.

Ischiatic Hernia.

Etiology.—This is a very rare form of hernia, and is supposed be due to a congenital defect. It protrudes through the upper pa of the great sacrosciatic notch.

Symptoms.—When the hernia is small and there can be found n swelling, it is difficult to detect symptoms. When the swelling large it may be confused with a lipoma, a cyst, an abscess, a hæm toma, or an aneurysm. In infancy, a spina bifida may simula it. If it becomes strangulated, localized pain in connection with tl general symptoms may assist in the diagnosis. The hernia usual consists of intestine, but the omentum or ovary may sometimes l found.

Treatment.—On account of the location and of the mobility of tl buttock, it is almost impossible to wear a truss or a bandage. A operation should be advised unless especially contraindicated by ag etc. The incision should be carried down through the gluteus ma imus muscle in the most direct manner. After the hernia has be reduced the incision is to be closed by the usual series of sutures.

Obturator Hernia.[13]

Etiology.—In this variety the protrusion emerges from the pelv cavity through the obturator foramen, together with the obturat nerve and vessels, the nerve being at the outer side and the arte generally also, though at times it is behind the hernia. The area the opening is about 9x14 mm. The direction of the hernial tract downward, forward, and inward, and the hernia protrudes general under the obturator externus muscle, although at times in front that muscle, and is covered by the pectineus and adductor longu The hernia pushes before it in its descent the peritoneum and tl strong pelvic fascia to form the sac. Its contents are usually inte tine alone; the omentum is present only rarely.

This form of hernia is rare. It is an affliction of advanced ag and seldom occurs under fifty years. It occurs more often in wom than in men. Macready collected sixty-three cases, among whi were fifty-nine women and only four men.

Symptoms.—The patient may complain of attacks of colic ar nausea without vomiting, or of an indefinite pain along the course the obturator nerve, at the inner side of the thigh from the gro down to the knee, which is sometimes due to the pressure of tl

hernia upon the nerve in the foramen or canal. But inasmuch as the hernia is so deeply situated and concealed the symptoms are seldom recognized until strangulation has occurred (there may be now a very tender area), and often even when this is present the case is not diagnosed until after death.

A swelling may sometimes be felt. It is large high up, just behind and underneath the adductor longus, but it is not accessible from in front or in Scarpa's triangle. In order to make it most evident the thigh must be flexed, rotated outwards and carried inwards to relax the adductor muscles, but sometimes even this is not sufficient. To obtain the fullest information the finger should be passed inside the vagina or rectum, and then swept around the obturator foramen.

Treatment.—When the hernia is detected early enough it is generally possible to reduce it by pressure upwards, outwards, and backwards, but, because of the deeply situated location of the opening, it is impossible to apply a bandage or a truss. Therefore we should advise an operation, unless it is contraindicated by age or other unsuitable condition. If neglected, strangulation is most apt to occur, and eighty-five per cent. of these patients die.

Operation.—A longitudinal incision is made over the upper part of Scarpa's triangle, internal to the femoral vein, and the tissues are divided until the pectineus muscle is exposed. The inner border of the pectineus is separated from the outer border of the adductor longus, and, when these edges are drawn apart, the hernia will be found in front of the obturator externus and the adductor brevis muscles.

As in these cases the apex of the wound is so deep and dark that it is difficult to get a good view of the parts, it is considered rather better to operate from above through the abdominal wall. The incision is made best through the lower part of the linea semilunaris, as thus a good view of the region of the foramen is obtained. The constricting ring can now be divided with greater safety, for, if the vessels are injured or if the bowel is nicked, the injury can be readily seen and remedied from above. In order to escape the artery it is best to divide the ring downwards and to the inner side. Inasmuch as these cases are generally not operated upon until late, the process is apt to have advanced to gangrene, so that special care must be taken to protect the abdominal cavity from any contact with the extravasated matter. The further treatment must follow the methods described in the section on strangulated hernia.

Irreducible Hernia.

This term is applied to any form of hernia which cannot be reduced, but in which the functions are still carried on in the bowel. The symptoms of strangulation are absent.

Etiology.

A hernia which was at first reducible becomes later irritated and inflamed, plastic material is exuded and adhesions are formed between the different portions of the hernia itself, or between the hernia and the wall of the sac. If these adhesions are low down in the sac the hernia may be partly reduced by invaginating the sac, but on pulling down the latter the hernia descends. If, however, the adhesions are at the neck of the sac the hernia cannot be reduced in the least.

Fig. 42.—Irreducible Omentum. (Bull.)

There are various causes which render a reducible hernia irreducible; such are the failure to wear a proper truss, fæcal impaction, an increase in the size of the omentum in the sac through thickening and agglutination of its folds, in cæcal and sigmoid herniæ an adherence of the peritoneal fold to the bottom of the sac; finally, in certain old cases in which an enormous hernia has been down in the sac for a long time, there ceases to be room inside the abdomen for the return of the extra-abdominal portion.

The way in which the ill-fitting truss produces the irreducibility is as follows: At first the truss may retain the hernia perfectly, but gradually it slips or yields a little on motion of the patient, and a small portion of the hernia protrudes underneath the pad. As it is usually omentum there is not much pain, and so the patient does not keep it reduced, but continues to allow the truss to press on the

otrusion. The friction and irritation from the pressure slowly ovoke an inflammatory process of low grade, which results in the oduction of adhesions, and these render the hernia irreducible.

This condition is uncommon before puberty; it increases in fre- ency from then up to thirty-five years, and is most frequent from t age to sixty years. The reason it is infrequent before puberty that these early herniæ are chiefly of intestine and not of omen- n, and adhesions are not so apt to arise from the intestines as from omentum, which is the chief element in the majority of cases irreducible hernia. A less frequent condition is when the irreduci- omentum is combined with the reducible intestine, generally the all intestine. It occurs most often in umbilical, less frequently in noral, and least often in inguinal hernia.

Diagnosis.

The history of the case will assist one very much in making a ferential diagnosis. The patient states that the hernia has been ning down for a long or a brief period, and that he has always been e to put it back, until now he finds it irreducible. There is an im- lse on coughing, and palpation confirms the diagnosis. There is vomiting, constipation, or fever, and but little pain. This may colicky in character, and is often referred to the lower part of the lomen.

Treatment.

There are a number of *acute* cases of short duration which may en become reducible after a few days' rest in bed. An ice bag is plied, and gentle taxis is performed daily. A spare diet is main- ned, and the bowels are thoroughly moved. As the bowel is seldom herent, it will often go back quite readily, but the trouble comes th the omentum. If the adhesions are not too strong, they may come separated and allow the omentum to be reduced, otherwise it mains adherent and irreducible.

In *children* with irreducible hernia it is sometimes possible to ord relief by holding them head downwards for one or two minutes; e intra-abdominal pressure is thus removed, the rings are relaxed, e fluid contents in the bowel run back into the abdomen, and the pull traction which the viscera exert reduces the protrusion. Compres- on may be applied by a spica bandage, or by a firm rubber bandage a pad which fits closely over the protrusion; often this constant essure is of much value.

If the hernia has been irreducible for some time, for seve
months at least, it may be termed *chronic*. In this class of cas
rest in bed, with the ice bag and taxis, would be contraindicated.
the irreducible portion is not large and the symptoms are not urge
a truss with a special concave pad may be tried, or if the protrusic
is in the scrotum, pressure may be made by a firm leather sac whic
laces up. Macready has found this method very satisfactory, so muc
so that he believes a hernia which has been irreducible for sever
years may, under proper management, be made again reducible.
some cases, when forcible taxis has been employed, an effusion deve
ops. This also, he claims, may be reduced by suitable pressure (
fact he treats hydrocele in children with success by pressure).
some few cases, in which the irreducible portion attains a large size
the size of an infant's head—neither the rest nor the taxis nor t
compression will be of use, but something must be done to relie
the patient of the weight and to prevent any further protrusion. T
best relief is afforded by a well-fitting canvas sack which supports t
hernia and is suspended by straps which cross over the shoulders a
go around the waist.

There are many persons who have become burdened with an i
reducible hernia and who have been unable to find relief from any
the previous methods. Their affliction is a constant source of dang
to themselves, for so long as the hernia is irreducible the ring ca
not be closed, since the protruding part acts as a wedge to still fu
ther dilate it. At any time a loop of intestine may be forced dow
and it may easily become inflamed and strangulated, through even
slight accident. Under these circumstances we must consider t
advisability of an operation, unless it is contraindicated by the a
and condition of the patient. In the young and robust the operatic
is indicated; but in elderly and flabby patients, when the parts a
deeply situated and when there are probably dense adhesions, it
contraindicated. It is probably inadvisable in cases complicat
with chronic bronchitis or asthma, for the coughing and strainin
would possibly act very unfavorably. When the operation is to
performed upon a fat patient with a large irreducible hernia, it
best to prepare the patient by a week's rest. The diet should
regulated and the bowels must be thoroughly cleared out with abu
dant cathartics.

Operation.—This will be that performed for the radical cur
Special attention must be given to insure a firm ligation of ea
bleeding point after the adhesions have been carefully separate
The omentum usually presents the most difficulty, and if the stum
is too large to be easily reduced, it is better to diminish its si

rather than to increase the size of the abdominal opening, for this would weaken the wall and might predispose to a relapse.

Prognosis.

An irreducible hernia when neglected tends to increase, and there is a predisposition to strangulation.

Reduction without an operation succeeds more often in the inguinal variety than in the femoral variety. In the young an operation is successful in the larger number of cases. In middle life a relapse is more apt to occur, but if a suitable truss or belt is worn a hernia should not develop. In old age a truss with a concave pad or a sack is to be advised rather than an operation.

Obstructed Hernia.

Etiology.—This variety is also called *incarcerated hernia* (*incarcerare*, to imprison), and the term designates a large hernia which previously might have been reducible or irreducible, but which is now irreducible because its lumen is obstructed by the presence of fæces and gas. There is no interference with the circulation in the wall of the bowel. This form occurs generally in old people, and is usually of the umbilical or inguinal variety.

Symptoms.—The hernial tumor swells, becomes hard and irreducible; there is some impulse on coughing, but not much pain. The bowels become constipated and the abdomen is distended. There may be slight fever. Vomiting may occur, but it is not severe. These symptoms usually last for two or three days; then the bowels move and the hernia is reduced.

Treatment.—Reduction of the hernia may be brought about by means of large high enemata of sweet oil with a little turpentine or oxgall, or by the exhibition of saline cathartics and calomel in small and repeated doses. Local hot or cold applications may be made. Gentle taxis may stimulate the peristalsis and so force the contents along. If these means fail to produce relief by the end of forty-eight hours, the symptoms gradually increase in severity, the obstruction becomes complete, and the vomiting persistent, with a soft, rapid pulse. *Acute strangulation* has now developed, and the prognosis is grave, for an aged patient with a large strangulated hernia is most unfavorable for an operation.

Inflamed Hernia.

Etiology.—Inflammation may attack any portion of a hernia, the intestine, the omentum, or the sac. It may be due to the pressure of an ill-fitting truss, to prolonged and forcible taxis, or to contusions, or it may exist as the result of acute strangulation. When it is due to truss pressure, the omentum is most commonly involved, and its effects are usually limited and do not extend to the abdomen; it is generally of low grade, and results in the formation of adhesions between the protruded portions and the sac. It is in this way that an ill-fitting truss may cause a reducible hernia to become ultimately irreducible.

When it is due to violence, the intestine suffers most, and the inflammatory process often extends to the general peritoneal cavity, but if it remains local, adhesions result as in the former case. Inflammation occurs most frequently in femoral hernia, and next in the inguinal and umbilical forms.

Symptoms.—The swelling is red, hot, and tender. Impulse is present, but there is not much tension. There is some fever, and there may be a little vomiting and constipation. There are no general constitutional disturbances as in strangulated hernia.

Treatment.—The foot of the bed and the hips should be elevated, and an ice bag or coil should be applied. Saline cathartics and enemata are to be given, and if the symptoms persist for forty-eight hours, an exploratory incision should be made.

Prognosis.—In young individuals it is good, but in the old it is unfavorable.

Strangulated Hernia.

A hernia is strangulated (*strangulare*, to choke) when its neck has become so compressed that it can no longer be reduced into the abdominal cavity, and when at the same time there occurs an arrest of the circulation in the bowel and in its wall. It is a little more frequent in females than in males. It is rare before five years of age and unusual before twenty; then it gradually increases in frequency up to fifty, and from fifty to seventy the largest number occur. Many observers have not seen cases of strangulation in young children, but at certain hospitals in which a large number of children are examined it has been found occasionally." It is more frequent in femoral than in inguinal hernia, in old than in recent cases, and in congenital than acquired inguinal hernia. In fifty per cent. of the cases the contents

are small intestine, in five per cent. omentum, and in thirty per cent. intestine and omentum.

Mechanism of Causation.

The earliest theory to explain this condition was the one of Celsus, which dated from about four centuries before Christ and was widely accepted until the last century. Celsus believed that the arrest or obstruction was produced by an accumulation of hardened fæces in the protruded loop of intestine. Accordingly, the old methods of treatment were to soften this mass of fæces by taxis, warm baths, clysters, and poultices. Sharp (1750), and later Malgaigne, demonstrated that this theory was wrong, because it was generally the small, rather than the large intestine which was strangulated, and its contents were usually fluid.

Many different theories have been advanced from time to time, but the following are the most satisfactory:

Elastic Compression.—First, a loop of intestine is forced through a small hernial opening by some increased intra-abdominal pressure, either some violent strain, as in coughing, lifting, defecation, or a difficult labor, or an increased peristaltic action; then, during the passage of the bowel through the hernial orifice the latter yields a little and is dilated to its utmost, but as soon as the pressure from above diminishes, the elastic ring retracts, and constricts almost completely the lumen of the intestine. It thus becomes strangulated on account of the tight grasp of the constricting bands. The loop of the intestine may be empty or it may contain liquid fæces.

Fæcal Occlusion.—A loop of intestine already in a hernial sac becomes suddenly distended by a gush of intestinal contents, forced in by some suddenly increased intra-abdominal pressure. As the intestinal wall dilates on account of its elasticity, a little more of the bowel wall and its mesentery is drawn into the sac. In consequence of this addition the sac becomes still further distended, while the constriction becomes correspondingly tighter. The enclosed loop of intestine is now so overdistended with its increased contents of fæces and gases that it is strangulated.

Fig. 43. — Diagram Showing Theory of Strangulation. (Lossen.)

It is probable that these two conditions act together in most cases. In some cases there are bands (the results of old adhesions) be-

tween different portions of the intestine and adjacent structures, and a loop of intestine may be forced behind or underneath the band, and so become strangulated. At first the constriction may be sufficient to compress the veins only. The arterial walls being more elastic, the blood is still forced under the constriction and into the walls of the intestine, while its return through the veins is arrested at the seat of the constriction. Congestion results, and the swelling increases below the constriction until the circulation entirely ceases. Strangulation is produced through venous congestion. As the circulation is not suddenly but gradually cut off, the rapidity with which the strangulation becomes complete will depend upon the completeness with which the hernia occupies the ring.

There has been considerable discussion as to the cause and seat of this constricting ring, and it is now determined that its seat is at the neck or narrowest portion of the sac, and is either formed of the fibrous rings of the hernial orifice which are outside the neck of the sac, or is the narrowed neck of the sac itself. This narrowing may have been congenital, as in that class of inguinal herniæ which descend into an unobliterated tunica vaginalis; or it may have been due to the irritating pressure of a truss; or to the fusing together of the different folds or pleats of a large, thin, and flexible sac, as it is gathered and pressed together in the hernial orifice or canal. Macready says: "It is probable that the seat of the stricture is more often at the neck of the sac in inguinal hernia, and at the fibrous ring (Gimbernat's ligament) outside the neck in femoral hernia."

Changes in the Intestine.

These depend upon the completeness of the compression and the length of time it has existed. As the circulation becomes more and more completely arrested, the *color* of the intestine changes, varying from a faint blue to a reddish-brown, and then to black, when the strangulation has become complete. At the same time there occurs an exudation of serum into the lumen of the bowel and into the sac outside the bowel, together with an infiltration of serum between its coats, the latter being the cause of the increasing thickness of the walls. The strangulated loop feels full and tense, a condition due partly to the thickened walls and partly to the contents of the bowel. If the bowel be opened gas will escape, together with the exuded serum and fæcal contents. This may vary in color (depending upon the severity of the strangulation) from a pale, thin, yellowish fluid to one blood-stained, and the odor may be very foul.

Changes at the Seat of the Constriction.—In most cases in which

the constricting ring has been severed there will be found immediately underneath it on the intestine a grooved furrow, more or less deeply indented. This may be a quarter of an inch in length or it may extend entirely around the intestine. If the bowel still retains its vitality this furrow will probably fill up; if it does not, the prognosis is bad.

If the constriction has been firm and continuous, ulceration should be suspected. It begins at first in the mucous membrane, extends to the muscular coat, and later to the peritoneal coat, often causing perforation at the seat of the constriction. The greatest danger exists where the ring is sharpest. This is especially seen in femoral hernia at the inner side, where the constriction is caused by the sharp edge of Gimbernat's ligament. The opening varies in size from a pinhole upward, and it may be so situated that the extravasation of fæcal contents is either into the abdominal cavity or into the hernial sac.

As the strangulation becomes more severe, the circulation is more completely arrested, and so the nutrition of the bowel is more diminished. This altered condition of the bowel favors the escape from it of certain bacteria which are presumed to cause inflammation, in particular the bacillus coli communis, which is the most important, although streptococci and staphylococci are also found. The surface of the bowel may become covered with fibrin and lymph. It has a velvety appearance and becomes cloudy from loss of endothelium. This inflammatory process may extend to the abdominal cavity and set up a general peritonitis, or it may remain local and result in abscess formation. Adhesions may now be formed between the different portions of the contents and the sac wall. These at first are slight, and are easily separated, but later they become so firm as to bind the adjacent portions inseparably and they must then be reduced *en masse*.

Gangrene usually occurs when strangulation is severe, and follows the septic infection of the bowel wall. It may be limited to a small area, or may involve the entire strangulated mass. These areas become changed in color, presenting a marbled appearance, and the dead ashen-gray spots contrast markedly with the adjacent living tissues. The dead areas are cooler than the living portions. The peritoneal coat loses its lustre, the walls are softened and collapsed, and there is a penetrating cadaverous odor. As the gangrene advances gas is formed and the parts crackle. One coat only may suffer, but generally the entire wall is involved.

Changes due to Taxis.—These are common, inasmuch as taxis is so frequently misapplied; either the individual periods are too frequent and too prolonged, or too much force is employed. The su-

perficial tissues and the wall of the bowel are bruised, and it is said that the darker color may be due more to unskilled taxis than to the strangulation. The wall becomes weakened and more friable as the result of the inflammation, so that even moderate taxis sometimes causes a rupture. In that case the intestinal contents escape, and the serum becomes stained with blood. The bowel would be more often seriously injured were it not that in a large number of cases the omentum surrounds the neck of the bowel, and so relieves it from severer pressure. The exuded serum also acts as an elastic cushion to reduce the pressure.

Changes in the Omentum and Mesentery.—These may be due partly to the pressure of the constriction and partly to the taxis. The compression does not appear to injure the omentum so severely as it does the intestine. Moderate constriction causes at first a darker color, but as the process advances the dark color may become lighter, owing to the loss of coloring matter. If inflammation develops, it is apt to extend into the abdomen. Gangrene is less apt to occur owing to the absence of bacteria.

Fluid in the Sac.—The amount and character of this depend upon the severity of the strangulation. At first clear serum is transuded from the veins, but this may be tinged with blood from taxis, and it may become serofibrinous or seropurulent as the process advances.

Bacteria are seldom found in the fluid in non-gangrenous hernia, but as the strangulation becomes more severe, they increase rapidly with the production of the gangrene. The fluid now becomes turbid and much darker in color, and produces a very offensive odor.

Symptoms.

Pain is the earliest and most constant symptom. It is generally most marked at the hernial region, but may be referred to the umbilicus, and radiate from there over the lower abdomen. It begins as a series of attacks of severe colic, with paroxysms of a cutting, burning character.

As the strangulation is usually not complete at first, the arrest of the circulation is gradual and the onset of the different symptoms is variable.

Vomiting is a most important symptom, and usually begins soon after the strangulation occurs. It may be due to two causes. First, when it occurs soon after the strangulation; here it is set up by the reflex irritation of the solar plexus, produced by the sudden constriction of the intestinal nerves in the strangulated portion. Second, when it occurs later; the peristalsis forces the intestinal contents

downward until they are obstructed by the stricture, when regurgitation follows. This is made more marked by the powerful contractions of the abdominal muscles. The amount of the vomitus depends upon the amount in the stomach and intestine above the stricture. All food is vomited at once undigested. Later it may be mixed with mucus and stained with bile, and still later, the fæcal contents of the small intestine are ejected. Vomiting may continue with longer or shorter remissions until death. In rare cases there is no vomiting, but nausea is present.

Constipation is not always present at first, because the constriction may be high up in the small intestine, so that there may be a considerable amount of fæces below the stricture. After this has been evacuated the constipation is complete and no gas passes. In the uncommon cases of partial enterocele (Littré's Hernia, see page 315), constipation may be incomplete, because the constriction does not obstruct the entire lumen of the bowel. In that case, only a piece of the wall of the bowel is strangulated.

The pulse is small and rapid. It may become softer on account of the reflex paresis of the heart muscle, which is due to the irritation of the solar plexus. The temperature is slightly elevated, but this is an unreliable factor, and does not correspond to the degree of danger, even when gangrene is present. When there is prostration, however, the temperature may become subnormal.

At first the tongue is moist, but soon it becomes coated and later, with the peritonitis, quite dry. Hiccough is one of the later symptoms associated with peritonitis.

Tympanites gradually develops as the bowel above the constriction becomes more and more distended by its contents of fluid and gas. This meteorism increases more rapidly after peritonitis has developed. It is a dangerous sign, for it indicates a paralysis of the coats of the bowel. It may increase so much as to press the diaphragm upward, and so interfere with the respiration.

Locally the skin may not denote the condition underneath. If there has been extensive taxis it may be reddened or ecchymosed, but gangrene may be present without any inflammation of the skin. Some swelling will be present, and a hard, tense, tender, and irreducible tumor can be felt just outside the hernial orifice, with its neck running up into the canal. There is no impulse on coughing. There may be some tympanites over the tumor if any gas is present, and sometimes pressure will reveal the cracking of emphysema in the subcutaneous tissues.

In some cases marked prostration and severe collapse may develop rapidly at the very outset. The eyes are dull, the face is ashy,

gently, but surely, removed from this ring, or else the ring must be cut.

Taxis.

To accomplish this reduction taxis may be tried, and by taxis is understood the method of reducing a hernia into the abdominal cavity by the continued pressure of the palm of the hand, combined with a constant kneading or manipulation of the hernial tumor by the fingers.

The patient must be placed in a position most suitable for the relaxation of the constricting fibres; he must be in bed on his back, with his hips raised and his knees flexed. The tumor is now lifted up, and firm pressure is carefully made in the direction of the axis of the canal. One hand grasps the fundus of the tumor, and the other manipulates the neck, kneads the contents, directs the return of the protrusion, and prevents a further reprotrusion. Care must always be taken not to press upward too strongly, for this mistake sometimes forces the tumor to overlap the external ring. If the omentum in the neck is too much crowded together it may become too thick and larger than the opening. In that case, instead of pressing upward, the tumor is to be pulled downward in order to lengthen and to thin the neck. Then the tissues about the neck are gently pressed back, the operator remembering that the part of the bowel which came down last must be replaced first, and that the parts in the sac (the omentum) which first descended are in front, while those which came down later were added from behind.

The taxis must be begun very gently, so as not to pain or alarm the patient, and also in order to gain his confidence, otherwise he will hold himself rigid and will not relax his muscles. It should be continued for not more than five to ten minutes at a time, and then repeated after an interval of thirty to forty minutes up to five or six hours after the onset of strangulation. During the intervals a warm bath and a hypodermic injection of morphine (one-quarter grain) may be given, and in some cases when taxis is next applied the hernia will be easily reduced. Some have advised pouring a tablespoonful of ether over the swelling every ten to fifteen minutes, on the ground that the intense cold produced by the rapid evaporation would relieve the engorgement. This may be tried during the intervals of taxis, but no time should be lost with these experiments, for they seldom succeed. From time to time the abdominal wall immediately above the hernial opening should be firmly pressed deeply into the pelvis by the ends of the fingers, which are carried just over the brim of the pelvis. This movement displaces the viscera from the immediate vicinity of the internal hernial opening, and so causes some traction upon the

bowel from within. By this traction the vessels are straightened out and are relieved from their kinking and their engorgement. Gas and fluids may also escape from the loop back into that part of the bowel which is inside the abdomen.

Some writers have thought that one of the obstacles to the reduction of the strangulation was the effusion in the sac, and so have advised aspirating this fluid. If this is done, it should be under thoroughly aseptic conditions; it will probably not accomplish much. Others have advised aspirating the bowel itself to remove the fluid and gas, but this should not be done except in an open wound and when the opening can be closed by a Lembert suture.

Signs of Reduction.—The intestine suddenly slips back with a gurgle into the abdomen and the tumor disappears; the ring is empty and the finger readily enters and explores the canal; the pain disappears; the symptoms gradually subside; the distention goes down, and gas and fæces are passed from the rectum.

A spica bandage and pad or a truss is applied at once. The patient should remain in bed for several days, on a mild, low diet, the bowels being moved regularly with gentle cathartics.

Dangers of Taxis.—According to those authorities who have enjoyed a large experience, taxis is a means for good in skilful hands, but also a power for much harm in the inexperienced, for a considerable force is being applied to tissues the pathological conditions of which are unknown. The bowel walls may be inflamed or gangrenous, and a little too great or too prolonged force may cause a fatal rupture. All operators of experience report numerous cases in which the bowel has been bruised and many cases in which it has been ruptured. It is said that the black color of a strangulated bowel is due more to taxis, too prolonged and forcible, than to the strangulation itself.

In a doubtful case it is much safer to secure the assistance of an expert rather than risk injuring the sac or its contents, for in this class of cases injuries generally are followed by a fatal peritonitis.

After the strangulation has persisted for twelve hours taxis should not be attempted without an anæsthetic; and it is often remarkable how readily the hernia is reduced under the complete relaxation produced by the anæsthetic. All preparations should have been arranged to proceed at once with herniotomy if the taxis should be unsuccessful.

The bowel may have been bruised so much that gangrene develops even after the taxis has been successful, and a fatal peritonitis results. The contents of the gut may be squeezed back into the portion of bowel inside of the abdomen, while the intestinal wall remains unre-

duced. Again, the bowel wall may be ruptured just underneath the constriction and its contents will then escape into the abdominal cavity, while, at the same time, there is no decrease in the size of the strangulated portion.

One of the most deceptive and at the same time gravest conditions is present in those cases in which the symptoms are not relieved after the tumor has apparently been reduced. If the swelling has disappeared, if the ring is open and the canal is empty, but the symptoms persist, one should think of an incomplete reduction or of a reduction *en masse*. The protruded, strangulated loop of bowel has been pushed back out of sight, either between the layers of the abdominal wall or into the abdominal cavity, without having been freed from its constricting ring. This may happen either in the hands of the surgeon, or, occasionally, when the patient himself tries to effect the reduction. It occurs more commonly in inguinal and umbilical than in femoral hernia, and most often in the interstitial or properitoneal variety (see Properitoneal Hernia). In these cases immediate operation is demanded, and it may be well to consider the advisability of an incision through the abdominal wall in order more rapidly and thoroughly to examine and treat the prolonged strangulation.

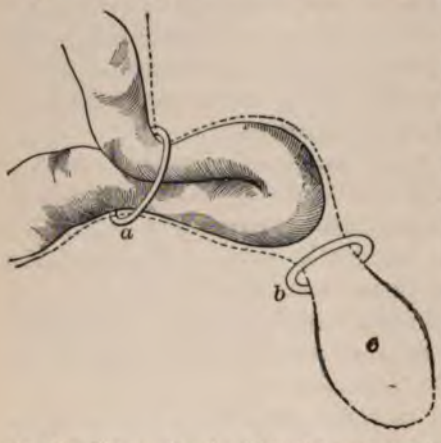

FIG. 44.—Diagram Showing Reduction *en masse* of a Strangulated Hernia. *a*, Internal abdominal ring; *b*, external abdominal ring; *c*, empty scrotal sac.

Contraindications to Taxis and Indications for Operation.—It should not be employed when the strangulation has persisted for twenty-four hours; when there is stercoraceous vomiting; when the bowel is thought to be inflamed or gangrenous; when the strangulation occurs with an old, irreducible hernia; when there is the history of previously prolonged taxis by other men. Nor should this treatment be used in Littré's hernia (partial enterocele), for the small portion of the bowel is so minute that it cannot be grasped thoroughly, and so firmly constricted that taxis will probably not reduce it. The number of favorable results continues to increase when herniotomy is performed early, and that method will gradually supersede taxis, the latter being limited to the earlier hours of strangulation.

Herniotomy.

A careful study of various records taken from a large number of operators has demonstrated that the success of herniotomy is directly proportionate to its early performance, and that when performed before a patient has become exhausted by continued pain, loss of sleep, and lack of nourishment, the danger will be much diminished. One should not wait, in employing taxis, for more than twelve hours after the onset of strangulation, and the increasing severity of the symptoms may demand an earlier operation, especially if the patient is vomiting, and collapse has occurred. Herniotomy should also be performed when it is presumed that there has been an incomplete reduction, or whenever taxis is contraindicated. Again, in certain suspected cases a definite diagnosis cannot be made from the local symptoms, as in the case of an old irreducible hernia, an inflamed gland, or an obturator or internal hernia. In such cases it is safer to operate, for whenever the hernia is found a life will probably be saved, whereas if no hernia is found, the incision has not added much to the danger already present. More patients with obstruction die because they are not operated upon early enough than in consequence of the operation.

Before the operation is begun the preliminaries should be carefully completed. The area of the operation must be shaved and made thoroughly clean, and then covered with a large sterile dressing. The patient should be well protected from any loss of heat by suitable flannel garments and blankets. The bladder is to be emptied, and, if there has been much fæcal vomiting, the stomach must be washed out, the temperature of the water being about 100°. If the condition of the patient is satisfactory ether may be given; but if there is much prostration, it is best to use a two- or a four-per-cent. solution of cocaine for producing local anæsthesia.

Incision should be made parallel with the long axis of the tumor, beginning well above the tumor and ending well below, so as to have the incision amply large, for it will save much time when the constriction is reached. It is to be carried rapidly down to the sac without losing any time in attempting to distinguish the different anatomical layers, for they are seldom recognized. It is often difficult to know just when the sac is reached, since its character varies so much. Sometimes it is thick and dark and again thin and grayish; it may be adherent to the bowel or omentum, or it may glide easily over the bowel; it may contain fluid, and so fluctuate. In many cases one will be assisted by recognizing the mass of subperitoneal fat just before

reaching the sac. If uncertain, one should grasp the tissues with two "rat-tooth" forceps, lifting only a very thin layer each time and nicking between the forceps with the point of the knife. After several trials the sac is opened and a few drops of serum ooze out of the minute opening. A director is now passed in and moved around the bowel, and the incision is enlarged. Maisonneuve said that if the operator was in doubt whether he had opened the sac, he could be sure he had not opened it. In some cases in which the bowel is adherent to the sac, it may be possible to move the sac about until an area is found which is non-adherent, and the opening is made here. The fluid which escapes may give some information about the condition of the bowel. If it is clear and free from odor, the bowel may be only congested; if it is turbid, then the bowel is inflamed; if thick and with fæcal odor, the bowel has become gangrenous.

After the sac is fully opened, the index finger, which acts as a most intelligent director, is inserted toward the constricting ring. It should be determined now whether this ring is made up of fibres outside the sac or is due to a constriction of the sac itself. Next, the operator should try to slip the tip of the finger or the nail underneath the constricting band, for sometimes it can thus be stretched without the aid of the knife; but if this is not possible, then he should pass the knife along on the palmar surface of the finger (the back of the finger pressing the bowel away), and incise in a direction according to the variety of the hernia. The incision should not be a free one, but rather a nick with the point of the blade, an eighth of an inch at first, for often just this little nick will cut several fibres and so relieve the constriction; it may be further dilated by the finger or a pair of forceps.

The bowel should be immediately examined at the seat of constriction to determine if it has been wounded by the point of the knife, or if a perforation has occurred from the effects of the constriction. If even a minute opening exists, bubbles of gas with fæcal odor or a thin yellowish fluid will escape.

The bowel and omentum must always be drawn down until healthy portions appear, and both ends of the loop should be examined for several inches above and below the constriction. It is to be at once covered with hot towels or sponges wrung out in hot sterile salt solution, and then very minutely inspected inch by inch as regards its vitality. If its serous surface is smooth and glistening; if its color is red or dark, but not black, and it improves quickly after the constriction is cut; if it feels warm, and bleeds when it is pricked; if, when gently stroked, the blood which has been expelled returns

quickly, then its circulation and its vitality are good and its reduction is indicated. But if, on the other hand, it is black or brownish-black, lustreless, cool, does not bleed when pricked, its color does not improve, its wall is infiltrated and thickened, and its serous coat peels off readily, then gangrene has begun to develop and simple reduction is contraindicated.

When the bowel is merely congested it can be reduced, and if the condition of the patient is satisfactory, the operation for the radical cure can be completed.

Omentum is present in about half the cases, and when strangulated should always be removed. The stump should be spread out as thin as possible, then very carefully transfixed and tied in small sections (not more than a quarter of an inch in diameter) with fine catgut. These ligatures must not be nearer than one inch to the transverse colon, or they may damage the bowel, and the stump must project one-half inch beyond them, for the stump is apt to contract and allow the ligatures to slip off, causing a fatal hemorrhage. Further, the stump must not be too long or too large, because it may necrose and form an abscess and result in a fatal peritonitis. All adhesions are to be carefully separated and ligated; the recent ones may give way easily with gentle traction, but the old ones must generally be divided or dissected.

Reduction will be best accomplished by putting back the portion which yields first, pushing up a small portion at a time, and repeating this procedure. The contents of the bowel can usually be squeezed back before pushing up the bowel itself. When all of the hernia has been reduced the finger should follow up the stump into the abdomen and feel the posterior surface of the symphysis and the adjacent regions to determine that there are no adhesions about the openings and that the reduction has been complete.

In some cases there may be great difficulty in accomplishing the reduction, but the opening should not be enlarged unless all the other methods fail, for the longer the incision the weaker will be the wall and the greater the tendency to relapse. At times the intestine may be greatly distended above the constricted loop, more from meteorism and the temporary paralysis of the bowel walls than from a peritonitis. In these cases Greig Smith advises that the protruded loop be pulled out from the wound and that, after the wound has been thoroughly protected from infection by pads or sponges, an incision be made in the most dependent portion to liberate the gas and fluid contents; the incision is then closed with a Lembert suture. In certain cases reduction may be impossible, as in the case of a long-standing irreducible hernia in which the intestines have been so long outside the

abdominal cavity in the sac that they have become firmly adherent in a mass which is too large to be reduced. The constriction must be relieved and the bowels protected until a secondary resection can be performed. Tait has recommended in some cases that a ventral incision be made and that the bowel be reduced by pulling from inside. This would be rarely necessary, and probably would need to be combined with the usual method of herniotomy.

If the bowel is only moderately inflamed (if there is no ulceration) and if the affected area is small, the hernia may be reduced and retained by packing directly at the bottom of the wound. A gauze drain must protect the adjacent tissues and the wound must be freely open. If there is a minute pinhole perforation and the adjacent parts are absolutely healthy, this small portion of the bowel can be tucked in and the healthy bowel sutured over it. It can then be reduced as described above. If one is uncertain about the condition of the bowel, if it is a little too dark or the area is too extensive, it must not be reduced, for it is never safe to take for granted that the bowel is all right. There have been many cases in which the bowel was thought to be sound and was reduced and gangrene has followed. The bowel should be protected by hot, dry towels and inspected every six hours. If the improvement is not satisfactory at the end of twenty-four hours, the bowel must be treated as gangrenous.

In rare cases, when the constriction has been relieved, the improvement in the circulation may become so marked that the entire strangulated loop can be returned after an exposure of from twenty-four to thirty-six hours. Recovery followed this method in a case of one of the writers,[6] in a woman of sixty-seven years, who had suffered from strangulation of a cæcal femoral hernia for more than one hundred and five hours.

Two methods of procedure may now be considered, namely, to leave the intestine protruding and to establish an artificial anus; or to resect the gangrenous portions, to close by suture or the Murphy button, and to perform the radical operation.

Primary resection would be preferable if it were possible, because the patient could be relieved of the strangulation and have the radical operation performed with the prospect of an early recovery. If the patient is young and robust; if the strangulation is of short duration, and there is no collapse and no peritonitis, and the tissues are absolutely healthy; if the operator is skilful in this variety of work, and if the patient is in condition to endure the prolonged operation, then primary resection may be indicated. Its chief disadvantage is the length of time which is required. This has been somewhat shortened by the use of the Murphy button. Its greatest danger is

due to the fact that the resection may not be made through absolutely healthy tissues, so that gangrene occurs later at the line of the sutures. All sutures should be of fine silk. If the condition of the patient is satisfactory and there is no peritonitis, then the wound may be closed as is usual after the radical operation.

When the strangulation has been prolonged the patient is usually depressed and gangrene has already developed, so that the patient is not in a physical condition to endure the prolonged operation, nor are the local tissues suitable for primary resection. The operative procedure must be brief, and the bowel must be rapidly and completely emptied. In these cases it is best to make an artificial anus; and in general, it may be said that this is the safest method for the general practitioner in the largest number of cases. The bowel is drawn out of the wound until the diseased portion is free, so that if a perforation occurs the contents cannot pass into the abdominal cavity. Sutures are then passed from the skin at the edge of the wound through the serous and muscular coats of the healthy portion of the bowel to retain it in position. It is surrounded with gauze packing to protect the adjacent tissues and to prevent fluid running back into the abdomen, and is then incised. A large drainage tube is inserted into the opening, so that it may not become obstructed, and a large absorbent dressing is applied. This must be changed as often as it becomes wet through. The skin must be protected by some simple ointment to prevent a troublesome eczema, or even sloughing, from the irritation of the fæces. Later the anus may be closed by a secondary resection or a plastic operation; sometimes it gradually contracts down to a small fistula and closes spontaneously. In case the anus has been made in the jejunum or in the upper part of the ileum, considerable food may escape unabsorbed. We must therefore pay especial attention to the nutrition of the patient, and it may become necessary to give nutrient enemata through the artificial opening.

After-Treatment.—If much prostration is present, external warmth with hot bottles will be needed, combined with the appropriate stimulation. It is better to give only a small amount of fluid during the first twenty-four hours; morphine may be allowed in small doses. The pulse should be carefully watched for the onset of peritonitis. The bowels should not be moved before the fourth day, and then with an enema rather than by cathartics.

Complications.—In some cases the symptoms may persist after the herniotomy has been performed. The vomiting may continue for several hours after the reduction has been accomplished, but failure to diminish later is a suspicious sign that there is a paralysis of the

intestinal walls; or these symptoms may be due to the fact that the injured bowel has become gangrenous or that there is some leakage at the seat of suture, and for this reason it is wise to leave a gauze drain down to the area involved. If all goes well and no leaking occurs, this drain may be pulled out after forty-eight hours. The local peritonitis may have extended to the general cavity; or there may be a second strangulation, caused by an adhesion, a band, an internal hernia, or a kinking of the intestine; or there may be a neglected reduction *en masse* or a twisting of the bowel. In any case the wound must be reopened and a careful search made for the cause. Sometimes the accidents may be due to the resection of the omentum. The ligature may be inefficient, or it may become loosened and slip off the stump, and so hemorrhage occurs. It may be tied too near the edge of the adjacent intestine so as to interfere with its circulation, and ulceration follows. When the omental stump is ligated in too large masses, the end may slough and then an abscess develops.

Remote Consequences.—Treves has shown that intestinal obstruction is more apt to follow in patients who have suffered from strangulated hernia because of the old adhesions.

Cicatricial stenosis may occur as the result of sloughing in the injured bowel within a few weeks or not until months afterwards.

Prognosis.

If a strangulated hernia is unrelieved by reduction or by operation, the termination is generally fatal. In very rare cases spontaneous recovery has occurred through the perforation of the bowel and of the sac and the formation of a fæcal fistula. Lind collected 155 cases of strangulated hernia in which taxis was successful in only 10 per cent., and 2 of these patients died later of gangrene. Henggeler[13] collected 276 cases in which taxis was successful in only 5 per cent. The largest number of these successful cases occurred when the strangulation had lasted less than 12 hours. The mortality increases in a direct ratio to the duration of the strangulation. A careful study of the best statistics shows that 8 per cent. of cases terminate fatally when the operation is performed within 24 hours after the onset of the strangulation; 22 per cent. when the operation is within 48 hours; 45 per cent. within 72 hours, and 60 per cent. when 96 hours have passed. An average duration of strangulation after which the intestine may recover is from 48 to 60 hours.

The age of the patient is of much importance. Under five years the mortality is high, but it becomes lower between five years and thirty years, then rapidly increases up to sixty years, after which

time it is highest of all. The mortality also depends upon the variety of the hernia, being least in the inguinal, more in the femoral, still more in the umbilical, and most of all in the obturator and the other concealed forms, because in these infrequent forms it is seldom discovered early enough for a successful operation. In Henggeler's 276 cases there occurred 111 inguinal cases with 21 deaths (18 per cent. mortality); 159 femoral, with 38 deaths (23 per cent.); 2 umbilical, with 1 death (50 per cent.); and 4 obturator, with 4 deaths (100 per cent.). It has been thought that the aseptic methods were more favorable than the use of antiseptic solutions, for it was shown that during the years 1881 to 1885, when carbolic acid was used, the mortality was 38 per cent.; between 1885 and 1893, when bichloride was employed, 21 per cent.; but since then, under the aseptic methods, the mortality has become 16.3 per cent.

The cause of death in fifty per cent. of the cases is gangrene or peritonitis. Gangrene of the intestine is more frequent in femoral hernia, while ulceration at the line of the constriction is more common in inguinal hernia. The prognosis of strangulated omentum is more favorable, inasmuch as gangrene is seldom present, and the inflammatory process usually terminates in the formation of adhesions between the omentum and the sac wall.

The prognosis will be best when no active cathartics have been given, when taxis has not been prolonged, and when an early operation is performed under aseptic methods.

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DISEASES OF THE SPLEEN.

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DISEASES OF THE SPLEEN.

Anatomy and Physiology.

The spleen is practically a complicated lymphatic gland, placed in the circulatory system instead of in the lymphatic channel. It is enclosed by a fibrous elastic capsule from which trabeculæ enter into the substance of the organ, and giving off subdivisions unite and form a more or less delicate framework in which the lymphoid tissue is embedded. The splenic artery enters at the median side where the peritoneal reflexion separates, leaving a hilum, and it subdivides into numerous branches which traverse the trabeculæ. Lateral branches are given off from these trabecular arteries and penetrate the splenic pulp. The latter consists of aggregations of lymphoid cells more or less separated by stellate cells united by their prolongations to form a loose meshwork. Denser aggregations of lymphoid cells surround the smaller arteries, and on section through the organ these are recognized as rounded or somewhat elongated, light-colored spots, the so-called Malpighian corpuscles. On microscopical examination the latter are found to consist of densely packed round cells, in the centre or towards one side of which may be seen a small arterial twig. The Malpighian corpuscles correspond with the lymphoid follicles of the lymphatic glands, and the looser pulp substance between the Malpighian bodies is analogous with lymph sinuses of the glands. It forms a sort of channel containing lymphoid cells and red corpuscles more or less abundantly.

The weight of the spleen in the normal adult varies from 140 to 200 gm. According to Birch-Hirschfeld the measurements in normal individuals are on an average 13x8x3 cm. Considerable variations, however, occur in different individuals, and the size of the organ is relatively larger at birth and in early life than subsequently. The weight in proportion to the body weight is about 0.26 per cent., but it decreases considerably in old age.

The spleen occupies a position at the fundus of the stomach and lies between the left kidney, the diaphragm, and the tail of the pancreas. It extends from the eleventh rib, beginning 1 or 2 cm. from the spine, downwards and forwards to within 4 cm. of the point of the eleventh rib, according to Luschka. Distention of the stomach or of

the splenic flexure of the colon tends to displace the organ still farther backwards and upwards, while descent of the diaphragm by thoracic or pleural affections or traction from below causes a descent to a more anterior and inferior position.

The function of the spleen still remains doubtful in many respects. Unquestionably this organ bears an important relation to the manufacture of blood corpuscles. This has already been referred to in the discussion of blood formation (Vol. VII., p. 234), but a brief recapitulation will not be out of place. During early embryonal life, after the hæmogenic function of the primary mesoblastic columns and that of the liver have been successively passed, the spleen becomes an active organ in the formation of red blood corpuscles. Towards the end of fœtal life the bone marrow assumes greater activity and the spleen becomes relatively less important.

In post-fœtal life the importance of this organ in hæmogenesis remains more or less in doubt. Bizzozero, Foà, and Salvioli found that nucleated corpuscles are abundant in the splenic pulp after hemorrhage, while Tizzoni and others have shown that the removal of this organ causes at least a temporary decrease in the number of erythrocytes. Others, notably Löwit, Wertheim, and Müller, hold that this organ plays a part in hæmogenesis throughout life comparable to that of the bone marrow and the lymphatic glands. It would seem undoubtedly true that the spleen is less important than the bone marrow, though the position of Neumann and others who deny entirely that it is concerned with blood formation after birth cannot be maintained. In cases in which hæmolytic substances are injected into the circulation, or in which destruction of the red corpuscles has followed the introduction of distilled water into the blood or transfusion of heterogeneous blood, the spleen is found to become enlarged in proportion with the degree of blood destruction, and microscopic examination of the pulp shows the presence of pigment masses, red corpuscles of small and large size, nucleated erythrocytes and large cells containing small or fragmental erythrocytes.

A study of these changes and a consideration of the ultimate fate of hæmoglobin after its solution in the circulating medium indicate that this organ is actively concerned with the reproduction of hæmoglobin and thus takes a part in the final preparation of the corpuscles. Whether the corpuscles originate within the splenic tissue or whether merely the completion of the process occurs in that situation must for the present remain an open question.

Undoubtedly the spleen is active in the production of leucocytes. These are formed within the Malpighian bodies or "germ centres" and subsequently make their way into the splenic pulp and even—

tually into the venous circulation. An evidence of this proliferation is found in the abundance of mitotic figures within the Malpighian follicles in conditions in which hæmogenesis is active.

In a certain sense the spleen is an unessential organ. It has been repeatedly proved by experiments upon the lower animals and by surgical operations in man that the organ may be removed without serious detriment to the health. At first there may be considerable decrease in the number of corpuscles, which, however, to a certain extent must be ascribed to the seriousness of the operation itself, and there is usually a primary stage of leucocytosis. Subsequently the condition of the blood is restored to the normal and the individual apparently enjoys perfect health. It is quite likely that other organs compensate for the loss of the spleen, though the evidence of this is scanty. Several experimenters have found that the thyroid gland undergoes enlargement after splenectomy, but it has been proved beyond doubt that this organ is not hæmogenic in function and the enlargement is, therefore, in no way compensatory. The experiments of Vulpius, moreover, show that lymphatic glands present no enlargement or other evidence of abnormal activity, though further investigations of an histological character are needed upon this point. The action of the bone marrow, and the changes which it undergoes after splenectomy, have not been sufficiently investigated.

Reference has already been made to the fact that the destruction of red corpuscles in the circulating medium leads to swelling of the spleen and accumulation of fragmented degenerating corpuscles within its substance. Furthermore, Ponfick, von Hofmann, and others have shown by the injection of pigment substances into the blood that the cells of the splenic pulp appropriate the pigment particles, while the spleen undergoes more or less enlargement. Birch-Hirschfeld, Wyssokowitsch, and subsequently many other investigators have found that bacteria injected into the circulation are similarly arrested in the spleen, and the bacteriological study of this organ in various diseases shows that it is a frequent point of arrest of micro-organisms. These facts have been used in support of the theory that the spleen is the seat of bacterial destruction and that it occupies an important position in the process of immunization. Metchnikoff in particular has insisted upon the importance of these facts as a support to his phagocytic theory, and Bardack has found that injections of cultures of anthrax bacilli of a low grade of virulence were more toxic in animals deprived of the spleen than in normal animals. On the other hand, Tizzoni and Cantanni, Kurlow, Kanthack, Martinotti, and Barbacci have found no diminution in the resistive powers after the desplenization of animals. The general

opinion that this organ is unimportant in immunizing is, therefore, to a certain extent justified, though the experiments thus far made have not been entirely conclusive. It is possible, as Birch-Hirschfeld suggests, that the bone marrow or other structures may compensate for the spleen, and the strength of the cultures injected has frequently been such that little difference could be expected between the animals having a spleen and those without such. It is certain that the organ exercises an eliminative function, removing from the circulation, for a time at least, foreign bodies or degenerated cells. What further activity it may possess, however, must be determined in the future.

Congenital Defects.

Complete absence of the spleen may occur in association with other malformations of the abdominal organs and is a frequent condition in acephalic monstrosities. Occasionally it is found in embryos or in infants presenting no other striking abnormality, as in the case of Birch-Hirschfeld, in which a mature infant without other defect excepting asymmetrical development of the liver—one lobe lying on the left side, the other on the right—presented complete absence of the spleen. Death occurred in a few days. A similar condition was observed by Robert, the child living to the third year. The function of the spleen in these cases is probably conducted by the lymphatic glands and bone marrow, as may be assumed from the case of Arnold, in which the lymphatic glands of the abdomen were enlarged, and also from experimental and surgical experiences in which heterotopic splenic tissue has been found in the peritoneum or omentum after splenectomy.

More frequently than complete absence of the spleen, *rudimentary formation* or hypoplasia has been found to occur. Sometimes the organ in these cases weighs but a few grams, as in the instance reported by Savill, the weight in this case being 3.63 gm. (fifty-six grains) and the measurements $1\frac{3}{8} \times \frac{1}{8} \times \frac{1}{8}$ inches.

Supplementary spleen tissue is very frequently observed, the supernumerary spleen in these cases usually occupying the gastrosplenic omentum. Occasionally a number of such supernumerary spleens may exist. Minor defects, such as abnormal lobulation and irregularity in shape, are not unusual.

These defective developments and abnormalities of the spleen are unattended by symptoms referable to this organ, and even in cases in which the spleen is entirely absent there are no functional disturbances.

Abnormal Location.

Of the congenital abnormalities of the position of the spleen, the most important and interesting in a diagnostic sense is that known as transposition, in which the organ occupies a position on the right side, corresponding to that normally assumed upon the left side. The liver in these instances is found upon the left side, and other transpositions, such a dextrocardia, left-sided position of the cæcum, and consequent reversal of the position of the other intestinal parts, may occur. The transposition may, however, affect only the liver and spleen.

Downward displacement may occur in the new-born in consequence of umbilical hernia, or as the result of other abdominal deformities. Congenital diaphragmatic hernia may cause an upward displacement.

Movable Spleen.

Movable spleen (*lien mobile, wandering spleen, wandermilz*) may be the result of congenital conditions, or may be developmental. In the former instances there is abnormal elongation of the gastrolienal ligament and the organ may be freely movable throughout a considerable part of the abdomen. Elongation of the ligament, however, may result from pressure upon the splenic region, from blows, from the carrying of heavy weights, from continuous coughing, traction made upon the organ by peritoneal adhesions, or from increase in the size of the spleen itself. Enlargement of the spleen is by far the most important cause, and in every instance of this condition, unless secondary adhesions have occurred or the size of the organ is unusually great, mobility is observed.

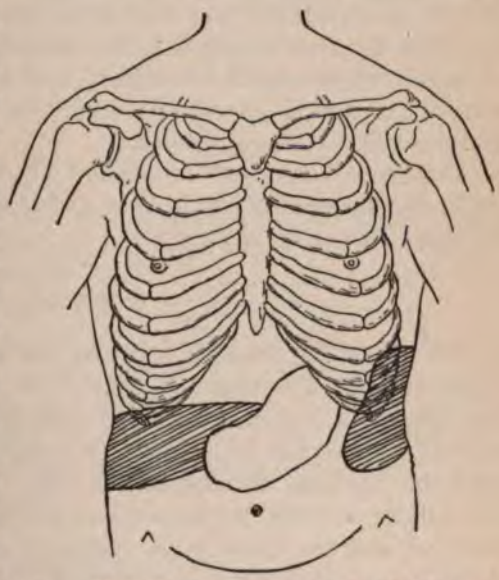

Fig. .—Movable Spleen Associated with Enteroptosis.

Considerable interest attaches to the cases in which movable

spleen is associated with descent of the stomach, the liver, and th other abdominal organs (enteroptosis). Cases of this kind are me with in individuals in whom the abdominal walls are lax, or the fat o the omentum and peritoneum is small in quantity. Sometimes, o the other hand, the condition results from excessive adipose deposi within and upon the abdomen. A case of the latter description i illustrated in the accompanying diagram. It is difficult to determin in some of these instances which organ was the first to become dis placed. Occasionally descent of the spleen may lead to secondar mobility of the stomach, but more frequently the organs are affecte coincidently.

Morbid Anatomy.

The spleen descends in a diagonal direction downwards and fo wards and in extreme cases may be found below the umbilicus an even within the pelvis. In its descent the gastrosplenic ligamen becomes elongated and with the splenic artery and vein and th pancreas, which is usually dragged downwards at the same time forms a slender and sometimes twisted pedicle.

Secondary changes in the organ are not unusual. The obstruc tion of the circulation may lead to enormous distention of the spleni vein, the diameter of this in some instances becoming as great as tha of a coil of the small intestines; and the circulation may be ob structed to such an extent that nutrition of the organ gradually fail and secondary atrophy results.

Complete torsion of the pedicle may lead to strangulation an rapid degeneration of the spleen, and this in turn may occasion rup ture and secondary peritonitis.

Symptoms.

The most important symptoms are those obtained by physica examination. The organ is usually felt with ease below the edge o the ribs on a line passing diagonally from the lower costal borde toward the symphysis pubis. In exceptional cases the spleen ma be visible through the abdominal wall. Palpation discovers the in dentations or *crenæ* on the median border, while the outer border i rounded and the lower edge more or less sharp. A difference be tween this condition and enlargement of the spleen without mobilit is found in the fact that the upper end of the organ is frequentl below the edge of the ribs and may therefore be palpated. This however, is not invariably the case. The organ usually moves les decidedly with the respiration than the normal spleen, or one en

larged but not movable. On percussion the normal splenic dulness is wanting in the left hypochondrium and especially posteriorly.

Subjective symptoms may be absent; indeed in many instances no suspicion of the disease exists until a physical examination has revealed the abdominal tumor. Sometimes, however, distinct and even distressing symptoms are met with. The patient complains of a feeling of weight or pain in the left side, or of a constant dragging sensation. At times attacks of sharp abdominal colic attended with violent retching or vomiting may denote the traction exerted upon the fundus of the stomach or upon the intestines. Pressure upon the latter structures may lead to obstinate constipation and occasionally to complete ileus. Dysuria is sometimes complained of when the organ occupies the pelvis and presses upon the bladder; and paresis of the limbs with formication and other paræsthesiæ have sometimes resulted from compression of the nerves.

Associated Conditions.—The stomach frequently suffers from the descent and traction exercised upon the fundus or from contraction and bending at the pylorus due to the descent of the pancreas. In consequence of these disturbances dilatation of the organ may occur and the symptoms of this condition may complicate those already existing. Associated mobility of the kidney, the liver, and of the stomach, has already been referred to. Twisting or torsion of the pedicle may lead to intense abdominal pain and to evidences of local or even of general peritonitis.

Diagnosis.

There is usually but little difficulty in diagnosis. It is sometimes hard to determine whether an enlarged spleen is movable or not when the degree of mobility is slight, but in cases in which the condition is at all pronounced little difficulty is experienced. The tumor might be mistaken for various abdominal growths, but a consideration of its position, of its shape, and of the absence of splenic dulness usually makes the diagnosis easy. Cases in which descent to the true pelvis has occurred may be mistaken for ovarian or uterine tumor, as in the instance reported by Gustav Klein in which it had been supported by a pessary.

Treatment.

In moderate cases the use of the abdominal binder or support is all that will be required. In more pronounced instances operative interference may be necessary. The only operation advisable in such instances is complete removal or splenectomy. Dendalo has recently

collected seventeen cases from the literature in which this operation was performed and only two of the cases ended fatally. Vulpius collected forty cases of movable spleen, including idiopathic enlargement, and among these there were thirteen deaths, or a mortality of 32.5 per cent. Rydygier adds eight further cases with two deaths, making the mortality 31.2 per cent. The condition of the patient after the operation has generally been good, though temporary disturbances of the health are usually observed.

Atrophy of the Spleen.

Congenital hypoplasia or defective development has been referred to. Secondary atrophy the result of the involution of advancing age or of distinct disease may, however, occur. Anatomical statistics show that the organ decreases progressively in relative size from birth until old age. More decided diminution in size occurs in certain diseases, notably in some cases of arteriosclerosis affecting the splenic arteries. In these instances the spleen becomes small and hard, the capsule is thickened and frequently much wrinkled, and on section the trabecular tissue is prominent, while the pulp of the spleen is seen to have undergone more or less complete atrophy. Another form of atrophy is that known as cyanotic induration. This will be referred to below.

Atrophy is unattended by clinical symptoms and has therefore merely a pathological interest.

Acute Congestion and Inflammation.

Synonym.—Acute splenic tumor.

Definition.—It is impossible to draw a sharp line between acute congestion of the spleen and acute inflammation. The limitations of these processes are difficult to establish in any part of the body and are particularly obscure in the spleen on account of the peculiarities of the circulation. Whether, as most histologists maintain, the blood circulates directly through the splenic pulp, or whether, as has been claimed by others, such as Thoma, Sokoloff, and Panski, the circulation is confined within vascular channels throughout this organ as in other places, it is certain that the blood more readily gains access to the splenic pulp than could occur if the channels were as resistant and as well developed as in other situations. In consequence of this fact the mildest grades of congestion are followed by extravasation of blood corpuscles almost as marked as that which occurs in cases of inflammation. Clinically distinctions between congestion

and inflammation are even less readily established, and it is advisable, therefore, to consider all cases as inflammatory processes having different degrees of intensity.

Etiology.

A certain amount of enlargement of the spleen occurs during the digestive period, as is readily demonstrated in animals killed some time after the ingestion of food. Exercise and other causes affecting the circulation similarly produce moderate increase in the size of the spleen; but these changes are entirely unimportant. Distinct increase in the size of the organ may occur in consequence of traumatism; blows, contusions, and pressure upon the left hypochondrium being not rarely followed by a certain amount of congestion. Usually this is moderate, but occasionally it becomes extreme and the lowered vitality of the organ resulting from the injury may establish a suitable soil for the destructive activity of micro-organisms already within the splenic tissue or entering it later from the circulation. Embolism in cases of valvular disease of the heart, of thrombosis of the cardiac chambers, or of arteritis and atheroma, not rarely leads to congestive enlargement of the spleen secondary to the hemorrhagic infarction. This form, however, remains for consideration under a separate head. Circulatory disturbances resulting from cardiac or pulmonary disease, and particularly such as affect the portal area in hepatic disease or in cases of thrombosis of the portal veins, may cause considerable congestive enlargement. In the instances, however, in which the circulation in the inferior cava is interfered with, as in pulmonary and cardiac disease, congestive enlargement of the liver is generally concomitant and in all of these conditions the splenic disease is more frequently lasting or chronic than acute.

The most important group of cases of acute inflammation or acute splenitis is that in which the condition is secondary to toxic and infectious processes. For a long time it has been recognized that the spleen undergoes enlargement in the infectious fevers, and the enlargement may be sufficiently pronounced to justify the term *acute splenic tumor*. Among the conditions of importance are typhoid and typhus fevers, relapsing fever, malaria, pneumonia, smallpox and the other exanthematous fevers, anthrax and pyemic infection, including malignant endocarditis, puerperal sepsis, and the like.

Acute splenitis, however, is not confined to the severer infections but occurs in such apparently trivial conditions as acute pharyngitis, coryza, tonsillitis, bronchitis, and the like, though the degree and the frequency of the occurrence are decidedly less marked. Among other

infections of a more serious character in which moderate and occasional splenic enlargement occurs are acute miliary tuberculosis, secondary syphilis, meningitis, and infectious jaundice.

In typhoid fever, splenic enlargement occurs in the overwhelming majority of the cases. Murchison found it absent in but one instance. Hofmann observed it in ninety-five of one hundred and seventeen autopsies, while other writers have placed the frequency on an average at from eighty-five to ninety-five per cent. of the cases. It is somewhat difficult to estimate precisely on account of the variability in the normal size and of the difficulty of determining lesser grades by the appearance of the organ. In addition to this, post-mortem contraction probably leads to the return of the normal size in certain instances, in which enlargement had been suspected during the life of the patients. The frequency in typhus fever is scarcely less than that in typhoid, and in relapsing fever splenic enlargement is almost invariable.

The regularity with which the spleen is enlarged is, however, greatest in malaria, Collin having found it present in every one of four hundred and ninety-one cases. In the other conditions to which allusion has been made splenitis is less frequent and less important.

Pathological Anatomy.

In the earlier stages the spleen is simply congested and enlarged, the substance being dark red in color and firm, and the capsule more or less distended. The Malpighian bodies are usually obscure and there may be visible areas of hemorrhagic extravasation. The size of the organ varies from a little beyond the normal to the extremest grades of hypertrophy, instances occurring in which it is four, six, or ten times the normal size and weight. Microscopically, at this stage the blood-vessels are found overdistended and the spaces within the splenic pulp contain masses of white and red blood corpuscles and very soon degenerated erythrocytes in the form of fragments or of masses of pigment.

In the later stages the spleen undergoes hyperplasia and degeneration. The former process affects the cells of the Malpighian bodies as well as those of the stroma of the organ, and leads to still greater increase in size and perhaps increase of consistence. Coincidently, however, degenerations usually occur and the substance becomes softened and somewhat lighter in color. Hemorrhagic extravasations are more abundant and on microscopic examination granular degeneration of the cells and fragmentation of the nuclei are observed, while large phagocytic cells containing pigment bodies or broken-down cor-

nucles may be abundant. In other cases the necrotic processes are localized and are particularly prone to begin in the Malpighian bodies. Spots of light color visible to the naked eye, and presenting somewhat the appearances of minute tubercles, may be seen on the surface of the section, and microscopically these are found to consist of masses of degenerated cells. Similar prominence of the Malpighian bodies, however, occurs in infective cases in which simple hyperplasia without degeneration of the Malpighian bodies is an early and pronounced condition.

In favorable cases the splenic enlargement subsides and is entirely removed by gradual resolution. In many instances, however, notably in typhoid fever, malaria, and relapsing fever, a certain amount of the hyperplasia remains for a time, or may be permanent. At the same time pigmentation may occur, the latter being particularly prominent in cases of malaria. In unfavorable instances a termination in diffuse necrosis of the organ or in suppuration may be observed, and particularly is this the case when traumatic injuries are sustained during the time in which the spleen is diseased. Actual rupture of the organ may occur spontaneously or as a result of injury. Repeated acute splenitis, as in cases of malaria, frequently leads to a chronic enlargement.

A peculiar feature has been observed in relapsing fever by Ponfick and others. This consists in the formation of areas having the appearance of hemorrhagic infarctions which may be scattered through the organ or may be closely grouped and involve a considerable part. Ponfick asserted that in these cases the infarction is the result of thrombosis of the veins rather than of embolic occlusion of the arteries, the latter being found entirely patulous.

Symptoms.

The subjective symptoms are absent in the majority of cases, no symptomatic indication of a splenic affection occurring during life. Occasionally, however, there are manifestations calling attention particularly to this organ. In most of these cases the patient complains of a feeling of weight or a dragging sensation in the left hypochondrium and it may be difficult for him to rest upon the left side. At the same time pressure over the lower ribs of that side may occasion discomfort, though this is often absent even in cases in which the enlargement of the organ is considerable. Severe pains are unusual and are more frequently due to extension of the process to the capsule giving rise to perisplenitis than to the uncomplicated disease. Stabbing or lancinating pains may pass directly through the left side

of the body or may radiate upwards to the left shoulder, and even into the left arm or downwards to the left leg. These may be aggravated when the patient lies upon the right side in consequence of the traction exerted upon the attachments of the organ. Usually the pain is of short duration, subsiding after a day or two, but occasionally it persists, notably in malaria, for a considerable length of time and sometimes even after the original disease has subsided.

Cough, dyspnœa, and palpitations of the heart may occur, though the connection of these with the disease of the spleen may generally be questioned. Vomiting may result from direct pressure upon the stomach and from extension of the inflammation to the capsule of the organ.

Physical Signs.

More important symptoms of acute splenitis are obtained upon physical examination. In very rare instances the enlargement may be such that the left side of the abdomen is somewhat swollen and it is sometimes possible to see the outlines of the lower part of the spleen through the abdominal walls, particularly after a deep inspiratory effort.

Palpation.—This furnishes the only reliable method of determining enlargement of this organ. The patient should be placed in a semi-supine position, midway between the dorsal and right lateral position. The head should be supported by a pillow and the thighs flexed slightly towards the abdomen. The examiner, standing at the back of the patient, places the extended fingers of the left hand upon the abdominal wall just below the margin of the ribs in front of and above the end of the eleventh rib. The patient is then instructed to breathe slowly and deeply with open mouth while the fingers of the examiner's hand are pressed firmly but steadily upwards beneath the margin of the ribs, the right hand being used posteriorly so as to displace the spleen as far to the front as possible. It is generally easy to palpate the lower end of the spleen by this method even in cases in which the enlargement is quite trivial. The organ is found to descend with each inspiratory effort, and in cases in which the enlargement is at all considerable, it may be readily palpated. The notch or notches at the anterior edge of the organ, the so-called *crenæ lienales*, are occasionally palpable.

Actual pulsation of the organ has sometimes been detected (Gerhardt, Drasche, and Pryor), but is extremely rare.

Percussion, though it is frequently relied upon, is most deceptive. The normal position of the organ has been referred to, but attention is again called to the fact that the position is more posterior than that

which is frequently assumed for it. Undoubtedly considerable enlargements may be determined with ease by percussion, but the method is unreliable in cases in which the stomach is tensely distended, or when the colon contains solid contents. Mistakes are easily made when the left kidney is enlarged and unusual thickness of the respiratory muscle and of the diaphragmatic attachments may lead to confusion.

Recently I have employed the method of *auscultatory percussion*, checking it by careful palpation, and have found this method more reliable than ordinary percussion, though in any case too much reliance cannot be placed upon the results.

Auscultation sometimes aids in the physical examination. Griesinger first noted the occurrence of intermittent or continuous bruits somewhat resembling the uterine souffles in instances of malarial enlargement. Subsequently Mosler detected the same in malaria and in relapsing fever, finding a bruit almost regularly during the period of chill in intermittent fever. The explanation given by Griesinger was that the pressure of the enlarged spleen upon the abdominal veins produced the murmur in question, but Bouchard has shown that the bruit is quite as distinct when the patient lies upon the left side and when the pressure referred to would be much less likely to occur. Mosler explained the murmur by assuming that contraction of the blood-vessels of the abdomen and particularly of the splenic artery occurs during the chills of malaria. The murmur may be heard in a limited area over the spleen, but occasionally is transmitted upwards towards the chest and downwards along the left side. Cardiac murmurs must be carefully excluded, as these are sometimes transmitted to the splenic region.

Course and Duration.

The clinical course of acute splenitis depends altogether upon the primary disease. Fluctuations, however, are common. At times the enlargement begins immediately after the onset of the original disease, but subsides or grows less marked within a short time. More commonly the splenic condition remains throughout the disease and not rarely persists for a time after the latter has subsided. Chronic hypertrophy may result. Occasionally the splenic enlargement antedates the other symptoms of the disease to which it owes its origin, as Birch-Hirschfeld observed in the case of typhoid fever occurring in his own person, and as Friedreich and others have maintained for the same disease. Spontaneous and traumatic ruptures have been referred to.

Extension of the inflammation to the surrounding tissues, espe-

cially to the capsule of the organ, may occur, and inflammatory adhesions may attach the organ to neighboring structures.

Diagnosis.

The existence of splenic enlargement is easily determined in cases in which the enlargement is considerable in degree. In cases of slight hypertrophy the diagnosis is sometimes very difficult. The only method upon which reliance may be placed is that of palpation, deductions from percussion being notoriously unreliable, the error amounting to as much as forty per cent. as compared with twenty per cent. when palpation is the method employed. If the lower end of the spleen is felt during inspiration in the course of any febrile or infectious malady, acute splenic enlargement may be assumed with considerable certainty.

Malposition of the organ may lead to mistakes, but in this condition a large part of the organ, or even the upper end, may be palpable and the mobility in the longitudinal or lateral direction may be striking. Enlargement or displacement to the left of the left lobe of the liver may occasion a palpable mass in the left hypochondriac region which is distinguished from the spleen only by careful examination. It should always be suspected in women who have been accustomed to tight lacing, or in persons in whom the lower thoracic outlet is contracted.

Tumors of the cardiac end of the stomach may occasion some difficulty as may also fæcal accumulations in the colon, but the associated symptoms, the shape of the tumor, and the less pronounced movement with respiration, usually suffice to distinguish these cases.

Prognosis.

In the immense majority of cases acute splenitis subsides after the disease which has occasioned it has been overcome and it exercises no influence upon the course of the disease. The accidents which may occur have already been noted.

Treatment.

Pain may call for the application of ice-bags, or of cloths wrung out in ice water, or for other measures of treatment, such as tincture of iodine, hypodermic injections of morphine, and the like.

Splenic Infarction.

Etiology.

Embolism of the splenic artery is very common in cases of endocarditis as well as in thrombosis of the left heart, or in atheroma of the aorta. Portions of the diseased valves may become detached or small parts of the fibrinous deposits upon the valves or upon the endocardium or the lining membrane of the arteries may be carried in the circulation and lodge in the terminal arteries of the spleen. This organ stands next to the kidney in the frequency with which embolism and infarction occur, the statistics of Sperling showing that the spleen was affected in 46.5 per cent. of the cases, while Leuch observed it in 50 per cent. The result of obstruction of the terminal vessels is nearly always a hemorrhagic infarction.

Pathological Anatomy.

Splenic infarcts may be single but are usually multiple. They may be small in size or they may involve the greater part of the organ. The infarct presents itself as a wedge-shaped area, the base being beneath the capsule and the apex towards the hilum of the organ. In the earlier stages the infarcts are dark red in color and are quite hard, resembling a densely hepatized lung. The capsule of the spleen covering the infarcted area may be uninvolved, but frequently shows localized fibrinous inflammation. The subsequent changes vary considerably in different cases. Usually the infarct grows lighter in color, eventually becoming quite gray or white, while it is surrounded by a zone of intense congestion or hemorrhagic infiltration. Softening may occur or gradual cicatrization leading to the formation of a pigmented scar. In some instances the spleen is found to be greatly distorted by numerous cicatrices of this character. If the embolus is of infective character the infarct tends to undergo rapid softening and abscess formation results. Occasionally small infarcts are completely absorbed, leaving scarcely any trace of their existence.

Symptoms and Diagnosis.

When in the course of a case of acute or chronic cardiac disease or of atheroma, a chill followed by sudden pain in the left hypochondriac region occurs, and the spleen is found to be enlarged, embolism and infarction may with considerable probability be assumed to have occurred. Frequently, however, embolism is unattended by distinct

clinical manifestations, and not rarely the spleen is found to be of normal size. In severe instances the onset of the condition may be marked by a protracted shaking chill, and the pain attending the obstruction of the vessel may be of extreme severity. Vomiting, depression, and even collapse are occasionally observed. The diagnosis is made more certain when embolism of the kidneys occurs and leads to severe pain in the loins and albuminous or blood-stained urine.

Prognosis.

In most instances the prognosis is favorable. In cases of acute and especially malignant endocarditis the condition may terminate in abscesses. In other cases gradual absorption or cicatrization is the rule.

Treatment.

Injections of morphine may be necessary when the pain is severe, or cold applications may be made to the side.

Suppurative Splenitis.

Synonym.—Abscess of the spleen.

Etiology.

Suppurative splenitis results from direct infection of the organ with pyogenic micro-organisms. A distinction may be made between the *non-embolic* and the *embolic* or *metastatic* forms. In the former class are included cases in which the infection of the spleen occurs by direct introduction of the micro-organisms through wounds, or by extension of infective processes in the vicinity, and cases in which the micro-organisms have gained access through the circulation, the suppurative processes being spontaneous or dependent upon secondary traumatism (idiopathic abscess). Thus in instances of infectious fever, like typhoid, malarial, and relapsing fevers, the micro-organisms may be present in the spleen with no other result than the development of acute splenic tumor until some traumatism, perhaps trivial, has initiated a more active inflammatory and suppurative condition. Extension of the inflammation in cases of peritonitis, perirenal abscess, pancreatitis, or ulcerative conditions of the stomach and intestines sometimes leads to the development of abscess of the spleen. Embolic or metastatic abscesses occur in cases of ulcerative endocarditis as well as in consequence of puerperal sepsis and vari-

ous other forms of intra-abdominal suppuration in which the micro-organisms have gained access to the blood. In a recent case under my own observation the splenic abscess was secondary to suppurations of the glands of the neck and was dependent upon the simple pyogenic micrococci.

Pathological Anatomy.

In the non-embolic cases the abscess is usually single. It varies in size from a walnut to enormous collections distending the splenic capsule and sometimes filling a large part of the abdomen. Cases have been recorded in which the size was such that the condition was supposed to be one of ascites. In the metastatic forms the abscess cavities are usually much smaller and are generally multiple, occurring more frequently near the surface than in the deeper parts of the organ. In the earlier stages they may present the appearance of hemorrhagic infarcts, beginning to soften at the apex or in the centre. Subsequently all appearance of infarction is lost and a simple abscess cavity is formed.

The final termination of abscesses varies in different cases. When small the pus may gradually become inspissated and eventually the abscess may be reduced to a cheesy mass surrounded by a hyperplastic connective-tissue wall. Larger abscesses tend to rupture into the peritoneum, causing peritonitis, or into the intestines, stomach, pelves of the kidneys, pleura, or into the bronchial tubes after penetrating the lung tissue. Very rarely they rupture externally.

Symptoms.

The onset of the condition may be marked by decided symptoms in cases of metastatic abscess, particularly such as are due to embolism in the course of cardiac disease. In these instances the symptoms are the same as those of hemorrhagic infarction: The spleen becomes enlarged and is often tender, the patient complains of pain in the side, subsequently irregular fever develops, and finally the signs are those of a profound septic condition.

Idiopathic abscess and sometimes metastatic forms begin more gradually, the onset rarely being distinguished by striking symptoms. The growth of the abscess, however, is usually rather rapid and pain may be complained of when the capsule becomes distended or secondarily inflamed. Pressure upon the diaphragm may occasion coughing and dyspnœa; disturbances of the stomach are frequently met with, and fever of irregular type is usually observed. Pressure upon the vena cava may occasion congestion and œdema of the feet, and ascites has been frequently reported.

Physical examination reveals the lower end of the spleen projecting beneath the ribs in the earlier stages and subsequently there may be a tumor of considerable size. The shape of the spleen may be preserved, but not rarely irregular projection is discoverable. In the later stage fluctuation is detected and in rare instances, such as that of Grisolle, a large part of the abdomen may be filled with a fluctuating mass simulating ascites.

Rupture of an abscess may be denoted by sudden pain and collapse. The pus may be discharged through the bowel or by vomiting, and more rarely through the urinary tract or the bronchi.

Prognosis.

The prognosis is always grave, though cases have been recorded in which recovery has taken place after spontaneous discharge of the contents of the abscess, externally (Wardell, Sandler, Zweifel), through the bronchi (Nasse), through the bowel (Webb), or in other ways. A favorable termination by inspissation has already been referred to.

Treatment.

General depletion of the portal system in all cases of acute splenitis in the course of infectious disease has some value as a prophylactic measure. After the formation of the abscess surgical treatment may be advisable in idiopathic or traumatic cases and drainage may be followed by a favorable outcome. Operative interference is, of course, less advisable when the splenic condition is merely a metastatic lesion in the course of a primary infective process.

Chronic Congestion and Inflammation.

Synonyms.—Chronic hypertrophy; Congestive hypertrophy.

Definition.—Under this head are included enlargements of the spleen, characterized by proliferation of the trabeculæ and other fixed elements of the splenic tissue, and to a lesser extent by hyperplasia of the true parenchyma of the organ. Distinct degenerative conditions and infectious or malignant new growths are excluded. Some cases begin as manifest inflammatory processes from the first; other cases have their origin in simple passive congestion.

Etiology.

Not rarely chronic enlargement of the spleen follows acute splenitis, and is therefore due to infectious agents. Of these forms malarial hypertrophy is by far the most frequent and important

is may occur in cases in which repeated attacks of malaria have urred, or it may be noted in persons who have lived in a mala- us country, but who have never presented distinct evidences of larial intoxication. This is well illustrated in Pantjuchow's sta- ics upon the weight of the spleen among the residents of Tiflis. ong the Russians the average weight was 285 gm., while the rage in various native peoples was 505 gm. The average weight persons dead of malignant acute malaria was 543 gm., and in those o had suffered from chronic malaria 790 gm.

Among the chronic infections syphilis and tuberculosis are of at importance. Syphilitic enlargement of the spleen may be te in the secondary stage, as has already been noted. Chronic argement is more frequent in congenital and hereditary syphilis in ich, according to Parrot, the spleen stands next to the bones in nt of the frequency of involvement. Syphilitic gummata of the een are rare. They may be solitary or multiple, large or small. e spleen may be coincidently enlarged by inflammatory hyper- sia and congestion, or more rarely the gumma is embedded in erwise unaltered splenic tissue.

Tuberculosis is only an occasional cause of splenic enlargement of form under consideration at present. Cases, however, are met h in long-standing pulmonary or visceral tuberculosis. Amyloid argement and acute splenic enlargement are more frequent. Allu- n may be made at this point to the occurrence of tubercles in the een, the latter, like syphilitic gummata, occurring in association h true splenic congestion or hypertrophy, or independent of such. acute miliary tuberculosis the splenic tissue may be densely dded with minute gray granules, while the splenic tissue itself is tely congested. In chronic miliary tuberculosis small tubercles y occur without other disease of the organ. Particular interest, wever, attaches to another form of tuberculosis, that in which large lules, sometimes the size of a bean or even of a walnut, stud the face and are embedded in the deeper portions of the organ. This been referred to by the Germans as *Affentuberculose*, on account he frequency of the form in monkeys. It is occasionally met with scrofulous children, and sometimes presents the external appear- es of secondary malignant tumors. The spleen may be consider- y enlarged.

In certain chronic nutritional diseases, particularly in rachitis, argement of the spleen has been met with. It is difficult, how- r, to estimate the exact frequency on account of the difficulty of tinguishing cases due to congenital syphilitic infection, and those which rachitis alone is of etiological importance.

A separate description is given in the article upon "Diseases of the Blood," in Vol. VII., of an affection recently described by von Jaksch under the name of anæmia infantum pseudoleukæmica. The pathological changes in the spleen in these cases differ from those which are found in pseudoleukæmia or true leukæmia and correspond very closely with simple hyperplastic enlargement affecting the connective tissues in particular. It is probable that various underlying diseases may occasion the splenic hypertrophy and the anæmia in these instances, rather than that the disease is primarily an affection of the spleen. Many of the instances reported as cases of splenic anæmia may be disposed of in a similar manner, as may also cases described by French writers under the title *splénomégalie primitive.*

Among the causes of congestive enlargement of the spleen, obstructions of the portal circulation by various hepatic diseases, such as cirrhosis, hypertrophic cirrhosis, carcinoma, or echinococcus cysts, or by venous thrombosis take the first rank. In these instances the return circulation from the spleen is impeded, the venous channels are overfilled, and reactive hyperplasia ensues. Undoubtedly, however, in certain instances of hypertrophic, or even of atrophic cirrhosis of the liver the primary irritation which has led to the disease of the liver may also affect the spleen with the production of hyperplastic changes. The splenic hypertrophy may actually antedate the cirrhotic process in the liver; and enlargement of the spleen may be absent in certain instances of cirrhosis, even though the circulation be considerably obstructed. The explanation of the latter fact in most cases is that the splenic tissue has already undergone fibroid changes which prevent distention and increase in size.

Chronic congestion and enlargement of the spleen is less frequent and less pronounced when the entire venous circulation of the inferior vena cava is obstructed by cardiac or pulmonary disease, or by thrombosis, and in these instances congestive enlargement of the liver occurs at the same time, while evidences of congestion of the kidneys are obtained in the examination of the urine.

Continuous pressure upon the lower thoracic region has, in certain cases, been supposed to exercise an influence in bringing about splenic hypertrophy, and at times the disease seems to follow traumatic injury.

Finally, there are cases in which a minute examination of all possible causes of splenic enlargement fails to discover any condition adequate to produce this result. These instances have been at times referred to as *idiopathic enlargement.* An interesting series of cases was reported a few years since by Claude Wilson, embracing six cases

affecting the members of a single family in three generations. He was unable to decide as to any cause, and suggested inherited susceptibility to slight malarial influences, or the existence of some undiscovered disease. It is likely that chronic gastrointestinal irritations, in which acute manifestations have never occurred, but in which a constant irritation of the spleen exists, may lead to such results. This explanation, however, is purely speculative.

Pathological Anatomy.

The spleen may be increased enormously in size. Sometimes it is ten or twenty times the average weight. Cases have been recorded in which it has been actually forty times the normal; in one reported by Monro the weight was 25 kgm. (55 lb.). The surface of the organ may be entirely smooth, the capsule being somewhat thickened. This hyperplasia of the capsule may be simple and fibrous in character, but at times it assumes a somewhat hyaline appearance, and the areas of thickening may have an almost cartilaginous hardness. In other cases there is considerable hyperplasia of the capsule, which may occur as a uniform process or in lines or patches; and adhesions to the diaphragm, the stomach, or other surrounding structures are not unusual.

On section through the organ the appearance of the splenic structure differs somewhat in individual cases. As has already been said in the definition of the condition under discussion, hyperplasia of the stroma and fixed connective tissues is the essential process. Sometimes this reaches very considerable degrees of intensity and leads to the formation of dense trabeculæ, while the proper substance or splenic pulp is relatively much less conspicuous.

In other cases the connective-tissue hyperplasia is less striking, and the disproportion between the trabecular tissue and the pulp is therefore less marked. Occasionally considerable hyperplasia of the Malpighian bodies or of the pulp substance may occur and may give to the surface of the section a somewhat mottled appearance. Friedreich called attention to the fact that in some instances such hyperplasia may occur in localized areas and lead to the formation of projecting granules somewhat comparable to the granulations of a cirrhotic liver. In malarial cases the spleen is usually intensely pigmented, being dark red or almost black in color and of quite a uniform or homogeneous structure. The name "ague-cake" spleen has been frequently applied. In cases of chronic congestive splenitis the appearances in the earlier stages do not differ from those seen in the instances due to other causes, excepting that the enlargement of

the venous channels and consequent dark coloration of the spleen is perhaps more striking. In the later stages there is a tendency to atrophic contraction, and the development of cyanotic induration or cyanotic atrophy of the organ.

Symptoms.

In many cases chronic enlargement of the spleen manifests itself by no discoverable subjective symptoms, the patient being entirely unaware of its existence. Usually, however, in cases in which the spleen is considerably enlarged, the patient complains of a feeling of weight, or of a dragging sensation in the left side, and sometimes there is actual pain. The latter may be of a dull, indistinct character, or may be neuralgic in type, radiating upwards along the side of the chest or forwards over the abdomen. Firm attachments to the diaphragm or to the stomach may cause functional disturbances of a pronounced character. In the former case, dyspnœa sometimes becomes pronounced, and the patient finds it impossible to lie upon the right side without serious discomfort in breathing. In the latter case disturbance of digestion, anorexia, and even obstinate vomiting may occur.

The enlarged spleen may press upwards upon the diaphragm and heart, causing severe palpitations. Pressure upon the vena cava and the other veins of the abdomen is not unusual in instances in which the size of the organ is very considerable. The consequence of this may be pitting about the ankles, enlargement of the superficial veins of the legs, and even extensive œdema, the condition of the blood (anæmia) aiding very largely in the development of the latter symptom. Attacks of abdominal colic due to pressure upon the colon, or to attachments between the spleen and the stomach or colon, sometimes add considerably to the patient's distress.

The general appearance of the patient is often striking. It must be remembered, however, that this is more frequently the result of the original condition which has occasioned the splenic enlargement than of the disorder of the spleen itself. The experimental evidence referred to in the discussion of the functions of this organ shows that the abrogation of the splenic activity is not attended by serious disturbances of health, while the diseases to which chronic splenitis is due are most of them such as lead to considerable anæmia. Pallor or an ashen hue of the skin is frequently observed, particularly in cases of chronic malaria with splenic enlargement. Occasionally the skin has a decidedly greenish or even a brownish discoloration, and there may be distinct pigmentation somewhat resembling that of Addison's disease, though more diffuse in character. The patient

usually complains of symptoms directly dependent upon the anæmia. Among these palpitation of the heart and dyspnœa take the first rank, and on physical examination enlargement of the cardiac chambers and anæmic murmurs at the base of the heart or in the vessels of the neck may be discovered.

Hemorrhages may be due to extreme anæmia or to obstruction of the circulation. In the latter case considerable hæmoptysis may occur. In one instance under my own observation this symptom was the first indication of a chronic enlargement of the spleen, and the hemorrhage recurred repeatedly during the life of the patient.

Physical Examination.

The methods of examination are the same as in cases of acute enlargement, but the hypertrophy of the organ is usually much more easily determined, and indeed may be visible through the abdominal walls. The left side of the abdomen is enlarged and sometimes distended by a large tumor. On palpation the lower end of the spleen, the convexity of the left border, and the indentations or crenæ on the right border are often plainly palpable. Cases in which the enlargement is not very great and in which attachments have not formed may be distinguished by decided mobility of the organ (see Movable Spleen). On auscultation friction rubs may be discovered, but bruits, such as those heard in acute enlargement, cannot be detected (Mosler).

Course and Prognosis.

Chronic splenic enlargement may persist for many years without influencing the general health. Occasionally spontaneous disappearance takes place, and Gastanelli and Henoch have in particular called attention to the subsidence during pregnancy. The prognosis is entirely favorable *quoad vitam*.

Diagnosis.

Among the conditions to be distinguished are movable spleen, leukæmic and pseudoleukæmic hypertrophy, and tumors of the kidney and of the cardiac end of the stomach. The distinction from movable spleen is rarely difficult. It must, however, be remembered that in the majority of instances movable spleens are primarily enlarged. The cases in which a normal spleen is abnormally situated or movable are to be distinguished by the fact that the mobility is often very great, and the size of the organ can usually be determined through the abdominal walls. Leukæmic enlargement may be recognized by the condition of the blood. Great difficulty, however,

attaches to the differential diagnosis between splenic pseudoleukæmia or Hodgkin's disease and chronic splenitis. Undoubtedly in many of the instances of the former disease recorded in literature the actual condition was really that of chronic hypertrophy. The coincidence of enlargements of the glands would make the diagnosis more easy, but the history of the case is of greater importance. In instances in which a distinct history of infection of a more or less protracted character is obtained, and in cases of congenital syphilis or rachitis the diagnosis of pseudoleukæmia must be made with extreme reserve. After all, however, it is sometimes impossible to determine during life the sort of hypertrophy existing in a patient. Other tumors of the spleen are usually distinguished by the profounder disturbance of the general health, by the irregularity of the organ, by the absence of suggestive history, and finally by the occurrence of metastases. In cases of hydatid disease or of abscess, the rupture of the sac into the intestines, into the peritoneum, or externally may establish the diagnosis, or the existence of fluctuation, of fever, and of other symptoms may distinguish the condition present. Tumors of the kidney are usually differentiated with ease. They are much less movable, are situated farther to the left and usually lower, so that the upper margin may be felt in most instances; whereas in splenic hypertrophy the lower end of the mass alone is palpable. The condition of the urine may give important indications. The frequency of enlargements of the kidney by neoplasms in children, and the occurrence of hydronephrosis and pyonephrosis with distinct evidence of urinary obstruction in later life should be remembered. Tumors of the cardiac end of the stomach or of the transverse portion of the colon and of the omentum are rarely difficult to distinguish. The movements of the enlarged spleen synchronous with the respiratory movements, the absence of any distinct disturbances of the stomach or of the intestines in most instances of this condition, and the results of physical examination are the important indications in establishing the diagnosis.

Treatment.

The general condition of the patient requires attention in all cases. The associated anæmia calls particularly for the administration of tonics, and the diet should be suitably arranged according to the condition of digestion. Malarial enlargements sometimes subside quite promptly when the patient removes from the climate in which he has acquired the disease.

Many remedies have enjoyed a more or less extensive reputation as having power to reduce splenic enlargements directly. Among

these quinine and arsenic are prominent, and in every case of malarial enlargement they should be employed. Quinine may be given by the mouth or by injection. No advantage, however, seems to attach to the hypodermic administration in the splenic region, and the operation is always painful. Large doses should be given for a short time, and should be followed by smaller doses combined with arsenic in doses ascending to the point of tolerance. Under this treatment malarial enlargements frequently subside.

In cachectic individuals, and especially in rachitic or syphilitic children, iodine is of advantage. It may be administered in the form of Lugol's solution well diluted, or as the iodide of iron or potassium, the iron salt being especially useful in cases marked by anæmia.

Among other remedies advocated for reduction of the size of the spleen ergot, piperin, eucalyptus, salicylic acid, and pilocarpine may be mentioned. It is doubtful, however, whether any of these exercises decided influence.

In congestive cases the condition of the spleen may be relieved from time to time by the administration of salines, which serve to deplete the portal circulation and thus relieve the splenic engorgement. Care, however, must be taken lest the strength of the patient be too greatly reduced.

Various methods of local treatment have been recommended; among them massage systematically employed, and supplemented by cold douches or the application of ice-bags to the left hypochondriac region, may be of advantage. Faradization, vesication, and local applications of iodine may be employed, but offer little hope of beneficial effect.

Mosler has strongly recommended parenchymatous injections of arsenic or carbolic acid, injecting these remedies directly into the substance of the spleen. Other writers, among them Murri, Boarri, and Nigras, found that the beneficial result is due rather to the mechanical effects of injection, and have found distilled water as useful as the remedies named.

Feletti has practised acupunture with successful results in two malarial cases. Thoroughly aseptic needles are inserted directly through the abdominal walls into the substance of the spleen and allowed to remain a few minutes, the patient being absolutely at rest. This operation, however, as well as parenchymatous injection, is attended with a certain amount of danger, as fatal hemorrhages have sometimes followed puncture of the spleen.

Splenectomy may be a final resort in certain instances. In cases in which the size of the organ is not excessive little advantage is to be obtained from this operation, and it should be reserved for in-

stances in which the tumor is so large as to cause decided mechanical disturbance. The operation in itself is attended with considerable danger, but has been successfully practised by a number of surgeons.

Perisplenitis.

Synonyms.—Capsular splenitis; Local perisplenic peritonitis.

Etiology.

Inflammation of the splenic capsule may occur in association with disease of the spleen itself, notably with acute splenitis, infarction, and abscess. In other cases it results from extension of inflammations from the surrounding structures. Thus in ulcers of the stomach affecting the greater curvature or fundus, adhesions are frequently formed between the spleen and stomach, while in inflammatory affections of the pancreas, intestines, peritoneum, or perirenal tissues, simple inflammatory conditions of the splenic capsule or suppurative collections involving the capsule of the spleen may occur. Chronic hyperplastic thickening of the capsule has sometimes been ascribed to alcoholism and to syphilis, but is rather the result of chronic hypertrophies of the spleen occurring in the patients presenting the histories of these conditions. In arteriosclerotic atrophy of the spleen considerable fibrous thickening of the capsule is not unusual.

Pathological Anatomy.

In acute cases the capsule is coated with a fibrinous deposit of yellowish or whitish color and moderate in amount. In infective conditions purulent collections may occur in the vicinity of the spleen; while in subacute or chronic cases fibrous attachments are formed between the stomach, kidney, pancreas, intestines, diaphragm, or other adjacent structures, and the spleen. Capsular thickening in the form of diffuse or localized hyperplasia may occur, and sometimes in the most chronic instances the areas thus formed become exceedingly dense or pseudocartilaginous.

Symptoms.

The symptoms of perisplenitis are those of a localized peritonitis. Excessive pain, remaining fixed in the side or radiating throughout the abdomen, tenderness, swelling, and fever are the important indications. Subsequently general peritonitis may develop or in favora-

ble instances adhesions may form. The latter cases are characterized by symptoms resembling those met with in chronic enlargement of the organ.

Treatment.

Cases of simple or circumscribed capsular splenitis, if recognized, would call for local sedative treatment. The pain may be controlled by the application of ice-bags or injections of morphine. If perisplenic abscess formation occurs surgical treatment becomes necessary.

Rupture of the Spleen.

Etiology.

Rupture of the spleen may occur spontaneously or after traumatism.

Spontaneous rupture never occurs when the spleen is healthy. It is met with, however, in instances in which the organ has become softened or degenerated by acute splenitis in the course of the infectious fevers, notably typhoid, malarial, and relapsing fevers. Even in these cases, however, the rupture is not often entirely spontaneous, but on close questioning may be found to have followed an attack of coughing or vomiting or some strain in moving about too actively or in lifting weights. Rupture has occurred in a number of cases during the act of parturition.

Traumatic rupture is more likely to take place when the spleen is diseased, but may occur with a perfectly normal organ in consequence of severe blows, contusions, or crushes, or in consequence of direct penetrating wounds. In two instances under my own observation the organ was entirely normal and the compression of the lower part of the thorax—in one case by the wheel of a cart, in another by falling earth—had been attended by no serious external injury. An interesting case may be referred to in which the rupture was due to a varicose condition of the splenic veins (Cohnheim).

Morbid Anatomy.

In the mildest cases the capsule of the organ is merely severed as if by overdistention, and slight hemorrhage may occur upon the surface. In severe cases considerable laceration or multiple fractures may be noted. Hemorrhage is almost always abundant, and large collections of blood may be found around and behind the spleen. The abdomen may be filled with blood.

There is very rarely any evidence of secondary peritonitis.

Symptoms.

The symptoms are those of sudden internal hemorrhage. At the moment of rupture the patient frequently complains of severe stabbing or lancinating pain or there may be merely the feeling of a sudden giving way or rupture of an internal structure. The severity of the hemorrhage determines the character of the subsequent symptoms. When it is abundant the patient grows pallid and exsanguinated; the feet and hands become cold; the peripheral circulation fails so that the pulses become almost impalpable; giddiness, syncope, and sighing respiration are observed; and the abdomen becomes rapidly swollen. The pains which at first are localized in the splenic region may subside entirely or may give place to diffuse abdominal distress. Physical examination may detect splenic enlargement or dulness in the flanks due to accumulations of blood.

Diagnosis.

The occurrence of splenic rupture may be recognized by the localization of the pain, the increase of the splenic dulness, and the evidence of internal hemorrhage occurring in cases in which enlargement of the spleen may be assumed to have existed or in which direct traumatism to this region of the body has occurred.

Prognosis.

A fatal termination may be expected in practically all instances. The duration of life varies with the extent of the rupture, death having occurred within half an hour in one case (Darwin), and not until ten weeks after the injury in another (Powers). In the latter instance there was found at the autopsy a dense white scar partially filling the wound and the actual cause of death was not determined. In my own cases death occurred within a few hours. Complete cure by cicatrization of the wound has occurred in a number of instances recorded in the literature, even where the injury was due to gunshot wounds.

Treatment.

The patient must be placed absolutely at rest and morphine should be administered in liberal doses. Cold applications or ice-bags should be applied to the splenic region and internal hemostatics, such as ergot, are advisable. Diffusible stimulants will be required in the stage of collapse. Among these, injections of ether, of ammonia, and of camphor dissolved in olive oil are particularly valuable.

Amyloid Degeneration.

Etiology.

Amyloid degeneration or lardaceous disease of the spleen occurs under the conditions which lead to amyloid disease elsewhere. Among these causes chronic suppurations, particularly such as affect the bones in coxitis, Pott's disease, and the like, chronic pulmonary or other forms of tuberculosis, syphilis, and occasionally other cachectic conditions such as chronic malaria are the potent causes. In these cases amyloid disease is prone to occur in the spleen, the kidneys, the intestinal mucosa, and in the liver. The spleen is more frequently affected than any of the other structures named, Hoffman having found this organ the seat of amyloid change in seventy-four of eighty cases, or 92.5 per cent. In instances in which all of the organs are affected it is usual to find the process more advanced in the spleen than elsewhere, and it probably begins in this organ in most instances. The degeneration first manifests itself in the Malpighian bodies, affecting the walls of the blood-vessels, and later the lymphoid elements themselves. On section through the spleen at this stage the pulp substance is seen to contain numerous small pearly, gray, transparent bodies about the size of a currant seed. These have been likened to grains of boiled sago, and the term *sago spleen* is therefore quite appropriate. In other instances the amyloid disease is more uniformly distributed, the organ becomes enlarged, the capsule distended, and on section the substance has a homogeneous, dark-red color which has been likened to that of boiled ham or dried beef.

In the earlier stages it may be difficult to determine the existence of amyloid disease by naked-eye examination, but the reaction to certain coloring agents readily establishes the existence of the degeneration. If the freshly cut surface is treated with Lugol's solution the degenerated areas assume a mahogany red color while the normal portions are but little stained or at most become slightly brownish. Sections prepared for the microscope may be stained with solutions of methyl violet, and in such the diseased areas become light pink in color, while the healthy parts are blue.

Symptoms.

The onset of this condition is unattended by any symptoms. The first evidences of the disease are usually those discovered by physical examination, the organ in the later stages becoming enlarged and forming a splenic tumor of extreme hardness which pro-

jects more or less considerably below the margin of the ribs. Associated amyloid disease of the liver, kidney, and intestines aids in establishing the diagnosis. The liver becomes greatly enlarged, and the lower edge may be felt as an indurated sharp border. Albuminuria and especially the occurrence of globulin in notable quantities in the urine are significant symptoms; involvement of the intestines leads to protracted diarrhœa.

The general condition of the patient is usually one of cachexia, which in most instances is due to a large extent to the primary causes of the disease. The skin presents a clear whiteness and transparency which is almost characteristic in certain instances, while in other cases the appearance is rather that of a sallow cachexia. Examination of the blood may show more or less advanced secondary anæmia, but in a few instances in which I have made examinations of the blood the degree of anæmia was surprisingly slight in comparison with the pallor and apparent bloodlessness.

Diagnosis.

The recognition of amyloid disease is rarely difficult when it has advanced to grades of considerable severity. The existence of causes likely to produce the condition and the occurrence of enlargement of the liver and spleen are as a rule sufficient to establish the diagnosis. The discovery of marked globulinuria is of very great importance.

Prognosis.

The prognosis is unfavorable on account of the underlying conditions, though an arrest of the process may sometimes occur.

Treatment.

Various tonics have been recommended in the treatment of amyloid disease, although it is doubtful whether they exercise distinct influence. Among other remedies iodine, iron, or the iodide of iron may be administered. Arsenic may be useful in cases marked by decided anæmia, and in cases of syphilitic disease specific treatment should be employed.

Tumors of the Spleen.

In a certain sense the splenic enlargement of leukæmia and Hodgkin's disease may be included among the new growths of this organ.

In their structure they represent diffuse round-cell hyperplasia, or, according to the views of some, diffuse lymphosarcoma. Localized tumors of the spleen, however, are occasionally met with. They are more frequently secondary than primary. Among the primary forms attention has been called by Lancereaux and others to localized hyperplasias within the splenic substance of normal splenic tissue, and the term *splenic adenoma* has been applied. In a few instances *fibroma* has been observed.

Sarcoma, however, is the most frequent and the most important of the primary tumors, and doubtless many of the instances recorded by the older writers as primary carcinoma or cancer were instances of sarcoma. The round-cell variety is the most frequent, but occasionally primary melanotic sarcoma has been reported. *Cystic formations* have been frequently observed. Some of these are doubtless cavernous lymphangiomata; others are instances of cystic degeneration of the Malpighian bodies or of portions of the splenic pulp. Cystic tumors of this kind may be small but are sometimes of considerable size, and very frequently they are multiple. The cases described as primary *endothelioma* and primary *epithelioma* of the spleen have not been sufficiently studied. Absence of epithelial structure in the normal spleen makes the occurrence of this form of tumor as a primary affection most unlikely in view of the present knowledge of the pathology of tumors. It is possible, however, that fœtal inclusions of epithelial tissue may serve as a starting-point for carcinomatous new growths.

Among the secondary tumors *sarcoma* and particularly the melanotic form is most important. Secondary carcinoma is occasionally met with.

The primary tumors are usually single and may be partially encapsulated. Occasionally they reach considerable size. Secondary tumors appear in the form of multiple nodules.

Symptoms.

The existence of tumors of the spleen is usually determined by physical examination rather than by the existence of distinct clinical manifestations. Rapid growth may occasion considerable distress by increasing the weight of the organ or by the distention of the capsule, but pain is rarely a pronounced manifestation. The diagnosis is rendered certain when metastatic nodules are discovered in the abdomen or elsewhere.

Treatment.

Surgical procedures are the only hope of relief and must be undertaken with a full understanding of the gravity of the operation. Statistical evidence, however, is wanting upon the comparative danger of splenectomy in cases of tumors of this organ and in cases of simple enlargement or of movable spleen.

Parasitic Diseases.

Among the parasitic diseases invasion of the *Pentastoma denticulatum* requires only to be mentioned. It leads to the formation of small cysts lying immediately beneath the capsule of the spleen and often showing advanced calcification of their own capsules. The *Cysticercus cellulosæ* is very rare. A more important parasitic affection in point of frequency and of severity of its manifestations is *hydatid disease*, due to the invasion of the larvæ of the *Tænia echinococcus*.

Echinococcus.

The Tænia echinococcus is a parasite occurring in its adult form in the canine species, especially in the dog, and invading the human family in its larval condition.

Etiology.

The echinococcus disease is especially frequent in Iceland, Australia, and other localities, but is among the rarer affections in America. In a recent (1895) compilation of the literature Sommer was able to collect 67 instances in the United States, and Osler previously (1882) collected 12 instances in Canada. Of all of these cases the spleen was implicated in but 5. Of 1,681 cases summarized from Davaine and other authorities by Sommer the spleen was affected in 37. It is likely that the disease has been frequently overlooked in this country, and that numerous cases have been mistaken for other conditions or not recorded. I have myself seen 4 instances within the last several years in Philadelphia, only 1 of which, a case of S. Solis Cohen, has been reported in the literature. In 2 of these the spleen was not examined; in the other 2 autopsies were performed, and the liver alone was diseased. In the majority of cases the spleen is involved with other organs; occasionally it is the only part affected.

Pathology.

Single or multiple cysts may occur. In the former case there is a cyst of greater or less size with a firm fibrous capsule and a more delicate layer lining the inner side. The sac is filled with clear liquid of low specific gravity, generally non-albuminous and rich in sodium chloride. The multiple cysts occur in the form of a mother cyst with numerous daughter cysts springing from the inner layer. Secondary changes are not unusual. Injury may lead to rupture and discharge into the peritoneum, or spontaneous rupture may occur into this cavity, into the stomach, the bowel, the pleura, or even into the pelvis of the kidney or the ureter. Inflammatory changes may occur within the cyst and abscesses may result; or finally, in small cysts, gradual absorption of the contents may follow death of the parasite, and contraction and thickening of the capsule or calcification may then ensue. The situation of echinococcus cysts is most frequently the gastrolienal space, but in some instances the disease originates within the pulp of the spleen itself.

Symptoms.

The enlargement of the spleen may persist in a gradual and painless manner, the patient being totally unaware of any disease and presenting no appearance of illness. In other cases, however, pain may occur from the very first and sometimes is severe in character. The enlarging mass frequently produces severe pressure symptoms by upward displacement of the diaphragm or by compression of the stomach, intestines, or even of the bladder. Palpitations, dyspnœa, retching and vomiting, constipation, and dysuria or retention of urine are among the symptoms noted. The disease persists and progresses without fever or notable disturbance of the general health. Fever may, however, occur in some instances and should always lead to the suspicion of suppurative inflammation.

Physical examination in the earlier stages may establish merely enlargement of the spleen. As soon as the tumor has reached considerable proportions, however, it will be noted that the normal shape of the organ has been lost and that there is an adventitious, rounded mass projecting from one portion. Fluctuation may be detected in this upon palpation, and in rare instances a peculiar thrill or fremitus (hydatid thrill) may be discovered. Fluctuation and thrill, however, are by no means frequent. Auscultation may reveal distinct rubbing or friction and occasionally this may be palpable.

Diagnosis.

Aspiration with a fine needle serves to establish the diagn positively, the chemical character of the liquid being usually tinctive and the diagnosis being absolute when the hooklets are covered under the microscope. It is to be remembered, however, t inflammatory changes will often lead to admixture of consider albumin with the liquid and the hooklets may be completely abse

Hydatid cysts must be distinguished first of all from absc The latter condition is usually more rapid, is attended with fe and generally with marked evidences of inflammatory disturba The history of the case and the associated conditions (endocardi pyæmia, etc.) may finally furnish evidence which will point to nature of the disease. Exploratory puncture may be used as a resort and is generally absolute in its results. Peritoneal c rarely occasion difficulty. They are usually painful, are atten with fever or other evidences of peritoneal inflammation, are m fixed in position, and often deep seated. Finally, their position the discovery by percussion or palpation of the spleen in its nor situation establish the diagnosis.

Prognosis.

The prognosis is always grave. Rupture may occur at any t and may lead to death by shock, hemorrhage, or peritonitis. O sionally instances are observed in which discharge through the bo or through the stomach has been followed by recovery (Hub Small cysts may subside spontaneously by inspissation and calci tion after death of the parasite.

Treatment.

The only treatment advisable in these cases is surgical in cha ter. Repeated aspiration may be performed, but the present s of surgical technique warrants more radical procedure in most stances.

DISEASES OF THE LIVER.

BY

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AND

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NAPLES.

DISEASES OF THE LIVER.

INTRODUCTION.

History.

THE disorders of function of the liver were noted by Hippocrates as far as it was possible for him to do so in the complete absence of all anatomical and physiological knowledge of those times. To Galen is due the credit of having formulated a very complete theory of the functions and pathological relations of this large abdominal gland. This centre of nutritive activity of the organism, which stores up all the products of intestinal absorption and transforms them into the sanguineous fluid, was regarded by this great founder of medical science as the organ of sanguinification and calorification. He held that a disturbance of its function generated a great number of diseases, especially those having to do with an altered composition of the blood; that anæmia, plethora, and dropsy depended upon changes in this organ; that the bile was the result of this great vital activity, yellow bile being produced in acute febrile diseases, black bile in chronic affections, in mental disorders, etc. Thus, even in the time of Galen, the hæmatopoietic and calorifacient function of the liver was recognized better than would have been supposed from the absolute ignorance of physiology which then prevailed; and this knowledge, accepted without discussion and popularized by the Arab physicians, would have soon led to great advances had those theories not been opposed by Vesalius and Argentarius, who denied all value to the intuitions of the pathologist of Pergamus. Nor did the discoveries of Harvey and the arguments of Riolano and Bils bear any fruit here, for Glisson and Bartholin denied that the liver was of any importance as regarded the composition of the blood and accorded to it solely a bile-forming function. From that time the liver was looked upon as an intestinal gland whose only function was that of collecting the bile for purposes of digestion and of absorption of nutritive material.

This period of complete stagnation in the physiological conceptions of the liver was due to the fact that the pathology of this organ,

which had fallen into the hands of the iatrochemists and iatrophysicists, also remained stationary, and even the studies of Fernel, of Baillon, and of Sydenham failed to bring back a discussion of the Galenical theories, so pregnant of future progress. But the anatomico-pathological school was already forming which sought the cause of disease in the changes occurring in various organs, and a whole army of anatomists, histologists, physiologists, and pathologists were placing the milestones along the road leading to a true and complete pathology of the liver. Bartholin, Baillon, Glisson, Vesalius, and Bonnet, who gave an embryonic description of cirrhosis; then Morgagni in his immortal work, "De Sedibus et Causis Morborum"; Haller, Andrée, Saunders, Andral, Laennec, Cruveilhier, Rokitansky, and others all made serious contributions to the pathology of the liver. The most important labors of Magendie, Tiedemann, Gmenin, Reichter, Claude Bernard, and Lehmann have explained the hepatic functions, recalling and demonstrating experimentally the Galenical theories of sanguinification and calorification. Meissner, Bouchardat, Murchison, and Charcot discovered the urogenetic function of the liver; Heger, Schiff, Lussana, Legry, and others studied the depurative function; C. Bernard, Heger, and Schiff brought into prominence the glycogenic function; Laennec, Annesley, Haspel, Dutroulan, Frerichs, and Murchison explained the origin of certain special clinical phenomena; and Budd, Charcot, Kölliker, Harley, Ponfick, Schüppel, Kelsch and Kiener, Kupffer, and Pflügger showed what a great help may be derived from a study of the normal and pathological histology of the liver in the interpretation of the various diseases of this organ.

In recent times these interesting subjects have attracted a great number of students, and a host of experimenters have contributed in an anatomical and physiological as well as in a pathological and clinical sense to the progress which has been made in this most important chapter of medical pathology. A very great light has been cast upon hepatic diseases by chemical studies of the urine, and we may now say that it is possible to arrive at a correct diagnosis of diseases of the liver even more certainly by a functional examination than by a direct examination of the organ itself.

Anatomy.

The liver is the largest gland in the human body. Lying in the left hypochondrium, immediately below the diaphragm, it occupies a large space which takes from it the name of hepatic region. It is of a marbled-reddish brown color, of rather firm consistence, and

weighs, when removed from the body (cadaveric weight), on an average of 1,450 gm., or about fifty-one ounces. But if we calculate the great quantity of blood which is lost when the gland is removed from the body its weight may be estimated at about 2 kgm., or four and one-half pounds; this was called by Sappey the physiological weight. Its specific weight has a ratio to that of water of 15:10. Its surface is smooth, shining, and invested with peritoneum.

The anterior superior aspect is divided by means of a peritoneal fold, extending antero-posteriorly between the liver and the diaphragm (falciform ligament), into two parts, called the right and left lobes respectively, the right being much larger than the left. This is, however, only apparent, for there is no trace of such a division in the parenchyma of the organ.

The posterior inferior surface, slightly concave, is not regular like the upper one, but is divided into four parts by two parallel fissures running antero-posteriorly and by a third running transversely, connecting these two near their central point, forming approximately a letter H. The first portion, that on the right, has three small depressions: one anterior, the colic impression, caused by the hepatic curvature of the colon; one median, the renal impression which receives the upper end of the right kidney; and a posterior smaller one, the suprarenal impression, receiving the right adrenal. The right antero-posterior fissure is not complete; it has an anterior portion receiving in its somewhat wide concavity the gall-bladder, is interrupted just behind the transverse fissure, and reappears near the posterior margin to receive the ascending vena cava.

The left portion, representing all the inferior surface of the left lobe of the liver, is bounded by the left antero-posterior fissure, in the anterior part of which is found the fibrous cord representing the umbilical vein, and which receives in its posterior part the remains of the ductus venosus. The median portion is bounded by the two parallel fissures and divided into two parts by the transverse fissure, which represents the hilus of the liver and contains the portal vein, the hepatic artery, the bile ducts, the nerves, and the lymphatics. The anterior portion of this is called the quadrate lobe or anterior portal eminence, and the posterior portion the lobe of Spigelius or posterior portal eminence; the latter is united to the left lobe of the liver by means of a large ridge of hepatic substance which interrupts the right antero-posterior fissure. The inferior surface of the liver may also present other fissures (rimæ cæcæ) which bound accessory lobes. The antero-inferior margin directed obliquely upwards and to the left presents two depressions: one deep, corresponding to the fissure containing the umbilical vein, the other in the right lobe, wide, shal-

low, and rounded, corresponding to the fundus of the gall-bladder. The postero-superior margin, blunt and rounded, gives origin to the coronary ligament at the level of the lobe of Spigelius, and presents a wide and rather deep depression which is sometimes converted into a canal in which lies the ascending vena cava.

The liver is held in place chiefly by the other viscera which surround it and by the intra-abdominal pressure. Certain folds of the peritoneum, called ligaments, assist in this. Of these there are five, of which two hold the liver against the diaphragm and three connect it with neighboring organs.

The falciform or suspensory ligament is of triangular shape and contains within its folds the umbilical vein. Starting from the umbilicus and spreading out like a fan it passes to the right and left as far as the right longitudinal fissure of the liver where it divides into two parts, one of which accompanies the umbilical vein and the other passes upwards between the diaphragm and the superior surface of the liver as far as the coronary ligament, and joins the liver to the vault of the diaphragm.

The coronary ligament formed by a very wide peritoneal fold joins the posterior margin of the liver to the diaphragm, and widening out at its right and left extremities forms the right and left triangular ligaments.

The hepaticorenal ligament unites the inferior surface of the right lobe of the liver and the kidney. The hepaticogastric ligament, or lesser omentum, extends from the transverse fissure and hilus of the liver to the small curvature of the stomach and duodenum. The hepaticocolic ligament, which is the continuation of the preceding, is inserted into the superior portion of the ascending colon.

Although the first two of these ligaments may be regarded as suspensory ligaments of the liver, the other three serve rather to sustain the other organs and to maintain fixed the relation of contiguity between the liver and the splanchnic organs. We see, therefore, how slight are the means of support of the liver and how easily, when the other important factors in its topographical relations are changed, that is to say, the position of the other organs and the intra-abdominal pressure, the topography of the liver will itself undergo changes.

The Excretory Apparatus.—The liver possesses an entire system of excretory apparatus, composed of (1) Bile ducts, which join to form the hepatic duct; (2) a reservoir, the gall-bladder, which is connected with the preceding by the cystic duct; (3) a common canal, the ductus choledochus, formed by the union of the hepatic and cystic ducts. From the interlobular biliary canaliculi arise conduits, the biliary ducts, which anastomose and unite, becoming less

and less numerous but of larger size, as they approach the hilus of the liver, until near the transverse fissure of the inferior surface they are reduced to two, a right and a left, which immediately unite to form the hepatic duct. This duct, having a length of 2 cm. ($\frac{4}{5}$ in.) and a diameter of 6 mm. ($\frac{1}{4}$ in.) joins with the cystic duct to form the common duct or ductus choledochus. It is important to note that there are occasional depressions in the walls of the hepatic duct and of the large branches of the bile ducts, irregularly distributed in the former but arranged in two longitudinal rows in the latter.

From the mouth of the cystic duct to its entrance into the duodenum the bile duct is called ductus choledochus. Following the direction of the hepatic duct, of which it is merely a continuation, this passes downwards to the right and backwards, and at a distance of 6 cm. ($2\frac{3}{5}$ in.) from its origin unites with the pancreatic duct, pierces obliquely the inferior and internal wall of the second portion of the duodenum for a space of 15 mm. ($\frac{3}{5}$ in.) and opens into Vater's ampulla. Like the hepatic duct, the common duct has a very large number of depressions in its internal wall.

The cystic duct, starting from the union of the hepatic with the common duct, passes upwards and to the right for a distance of 3 cm. ($1\frac{1}{4}$ in.) and empties into the gall-bladder, which, lying in the anterior portion of the right longitudinal fissure, half covered by peritoneum, is pear-shaped, having a fundus, a body, and a neck of S-shape separated from the cystic duct by a constriction. The capacity of the gall-bladder is from 30 to 35 c.c. (a little over an ounce). At the junction of the cystic with the hepatic duct, which forms a very acute angle, the mucous membrane forms a spur of variable size which projects into the ductus choledochus.

Vessels and Nerves.—The hepatic artery lying behind the vena porta and to the left of the hepatic duct arises from the cœliac artery and gives off lobular, canalicular, and capsular branches, the latter supplying the serous membrane and anastomosing with neighboring branches of the mammary, phrenic, and other arteries; and vascular branches to the walls of the portal vein (vasa vasorum).

The portal vein, formed by the union of the superior and inferior mesenteric and the splenic veins, divides in the transverse fissure into two branches for the two lobes of the liver and then ramifying in the organ dichotomously but without anastomosing, and accompanied by prolongations of the capsule of the liver (Glisson's capsule), terminates in the interlobular veins.

Sappey describes under the name of accessory venæ portæ other venous canals which are distributed through the liver and which he divides into five groups: (1) branches coming from the lesser omen-

tum and from the lesser curvature of the stomach; (2) twelve to fifteen branches coming from the fundus of the gall-bladder; (3) branches from the walls of the portal vein, of the bile ducts, the hepatic artery, and Glisson's capsule; (4) slender veinlets descending to the liver from the diaphragm in the suspensory ligament; (5) branches coming from the abdominal walls and the anterior portion of the falciform ligament and distributed to the liver. These accessory portal veins possess a very great importance in the symptomatology and in the interpretation of many pathological changes in the liver. It is a well-known general law that, when the escape of blood through a vessel of a certain calibre is interfered with by any obstacle, the collateral vessels in consequence of internal pressure undergo a certain degree of dilatation; and in this way a collateral hyperæmia is produced which tends to compensate for the circulatory and nutritive disturbances caused by the original morbid condition. It is by such a mechanism that, when the portal circulation is impeded by any cause, by increased intrahepatic resistance or by pressure along the course of the vein, collateral hyperæmia arises in the accessory portal veins of Sappey, and results in a dilatation of these vessels which is to be regarded as compensatory.

Among the different groups of accessory portal veins above mentioned, the fourth and fifth are the most important, since they include vessels which are not derived from the digestive organs, but wholly from the abdominal walls, and also because they are capable of greater dilatation. Sappey has observed that among these the venule whose mouth corresponds to the left branch of the portal vein takes on the greatest increase in size, and this following the course of the umbilical vein has been mistaken for it by some; but he has shown, contrary to what was formerly believed, that the umbilical vein never remains pervious, nor does it ever become pervious in consequence of an obstruction in the portal circulation. The para-umbilical veins (Schiff), however, anastomose with the abdominal subcutaneous veins, and when the latter undergo a varicose dilatation, the so-called caput medusæ is formed about the umbilicus.

Aside from these accessory portal veins, the collateral circulation may be established in a certain way by other possible communications between the portal and the systemic venous systems, as by the œsophageal, coronary, and inferior hemorrhoidal veins and by the veins of Retzius, which pass from the intestine to the vena cava.

The hepatic and suprahepatic veins are formed, to the number of two, by the union of the intralobular veins, emerge at the posterior margin of the liver, and empty immediately into the ascending vena cava.

The nerves are derived from the vagus and the great sympathetic (cœliac plexus); some fibres come from the spinal nerves (right phrenic and the splanchnic nerves). The nerves accompany the branches of the hepatic artery and portal vein, forming plexiform ramifications provided with ganglia (Kölliker).

Topographical Anatomy.

The liver is found in the epigastric zone, occupying especially the right hypochondrium, extending more or less to the left, and forming part of the subdiaphragmatic viscera. It is convex on its upper surface, while its inferior surface is curved upon itself, the lips of the longitudinal fissures being approximated, so that in the normal position this surface is not visible on opening the abdomen. This concavity is most marked in the greater lobe, the lesser lobe being more nearly horizontal. The convexity of the liver does not look directly upwards but a little forwards. The organ is not quite horizontal, but somewhat inclined from left to right, so that the upper surface looks a little towards the right, while the inferior surface looks towards the left.

The posterior margin is united to the posterior wall of the abdomen, here formed by the diaphragm, by means of the coronary ligament, and corresponds to the cartilage between the eighth and ninth dorsal vertebræ. The anterior margin, which is a little lower on the right side, commences at the bony portion of the eleventh rib and following the costal arch, crosses the epigastric region at the level of the middle portion of the cartilage of the eighth rib on the right side, terminating at the middle of the seventh costal cartilage on the left side. The right extremity occupies the hypochondriac region of the right side, extending from the angle of the last rib down as far as the bony portion of the eleventh rib. The left extremity extends, becoming gradually thinner, as far as the fifth costal cartilage, but it does not occupy a fixed position and may pass more or less into the left hypochondrium, so far in some subjects as even to cover the spleen.

The convex surface of the liver is in contact with the vault of the diaphragm throughout. It therefore undergoes displacement during respiration, its highest point during extreme expiration corresponding nearly to the fourth rib.

The liver has immediate relations with the abdominal organs and mediate ones with the thoracic. Its upper and right surface is in relation through the diaphragm with the pleura and the base of the right lung; on the left side with the pericardium and the heart, which

rests upon the left lobe. The inferior surface of the right lobe is in relation anteriorly with the colon, medianly with the kidney, and posteriorly with the suprarenal capsule. The lobus quadratus corresponds to the first portion of the duodenum and its left extremity covers one portion of the anterior surface of the stomach. The lobe of Spigelius corresponds to the right pillar of the diaphragm and covers the lesser curvature of the stomach. The lesser lobe covers one part of the anterior surface of the stomach. The posterior margin is in relation with the inferior vena cava, the spinal column, the œsophagus, and the aorta.

Minute Anatomy.

The liver is completely invested by a serous capsule derived from the peritoneum, which is deficient only in the fissures and over the suprarenal facet of the inferior surface. But in addition to the peritoneal covering the hepatic parenchyma is enveloped in a very fine, transparent fibrous capsule, called Glisson's capsule, which is closely adherent to the peritoneum. This fibrous capsule not only covers the external surface, but gives off numerous offshoots which penetrate a short distance into the substance of the gland separating the more superficial acini. On the posterior margin the capsule gives off deeper prolongations, more especially around the inferior vena cava, accompanying the suprahepatic ramifications and becoming continuous with the interstitial connective tissue. A greater amount of connective tissue penetrates the liver from its inferior surface, accompanying the structures which enter through the transverse fissure, and follows the ramifications of the portal vein and of the hepatic artery, forming a rather loose connective-tissue sheath round each of them which permits the walls of those vessels to collapse if they are cut, while the branches of the suprahepatic vessels remain gaping when cut, because of the absence of such a connective-tissue sheath. Around the final ramifications of the portal vein and hepatic artery the connective tissue of Glisson's capsule forms a very intricate network, like a sponge, the spaces of which are filled with epithelial elements of the liver, so that by macerating a liver it is possible to obtain the connective tissue alone completely separated from the glandular substance.

In the midst of the connective-tissue network, so formed, is found the parenchyma of the liver, formed of cells of endermic origin, aggregated in groups called acini, or better lobules, since they cannot be compared to the acini of racemose glands, being deprived of a proper enveloping membrane. The hepatic lobules, which number,

according to a calculation of Sappey, about 200,000, are of polyhedral form, having a diameter of about 1 mm. and are separated one from the other by connective tissue and a special arrangement of vessels.

If a small branch of the suprahepatic vessels is cut longitudinally its wall appears to be riddled with numerous fine openings, around each of which the transparency of the vessel enables us to see a polygonal figure. These little openings represent the venules (intralobular veins) which enter the axis of the lobule at a right angle from the suprahepatic vein, the polygonal figures representing the basis of the hepatic lobules which are disposed along the branches of the suprahepatic (sublobular) vein like kernels of grain round the stalk.

The minute ramifications of the portal vein, entering the interlobular spaces (interlobular veins), surround the lobule, ramifying in such a way that a terminal venous branch is distributed to several lobules, and so that each lobule receives branches from several interlobular veins which, however, do not anastomose with each other. From those veins surrounding the lobule arises a rich anastomosing capillary network with fine meshes which penetrates into the interior of the lobule, the capillaries coming together again in the centre of the lobule to form the intralobular vein. The connective tissue penetrates the lobules, but not in appreciable amount in health, its quantity being, however, sensibly increased in certain pathological states.

The blood current in passing through these vessels takes the following course: coming from the vena porta by the interlobular veins, it enters the capillaries of the lobules through which it passes into the central veins, from these into the sublobular, and thence, with the blood from all the other lobules, into the vena cava. Rattoni and Mondino' have, as a result of their recent investigations, modified somewhat the above-mentioned scheme of the lobular circulation. They have endeavored to show that not only the portal branches, but also the ramifications of the nutritive branches of the hepatic artery and the venous radicles derived from this source, terminate in the capillary network of the lobule. It is interesting to note here that the blood pressure in the portal vein is very high because of the resistance in this intrahepatic capillary network which the blood must traverse before entering the systemic venous circulation. This is an anatomical condition which greatly predisposes to congestion of the liver and to the phenomena of diapedesis (Cohnheim). Furthermore, the blood stream in the portal vein does not feel the respiratory influence, as does that in the jugular, for example (Barry and Berard). Rosepelly has shown by means of tracings that the pressure varies in the

portal vein inversely as it does in the cava, so that, while, during expiration, a weak pressure exists in the suprahepatic veins there is a strong pressure in the divisions of the portal vein.

Returning now to the minute structure of the hepatic lobule, it remains to be said that in the meshes of the capillary reticulum hepatic cells are found which constitute the specific elements of the liver. Disposed in a radiating direction from the centre to the periphery of the lobule, they are of polygonal form without enveloping membrane, having a nucleus, and formed of granular protoplasm containing fatty molecules and small almost fluid masses consisting of glycogen.

Besides the muscular network in the lobule there is a very fine reticulum of true biliary capillaries, each with its own membranous wall formed of very minute flattened epithelial cells like those in the blood capillaries. These capillaries start from the intercellular spaces and anastomosing with each other give origin to the interlobular biliary canaliculi which encircle the lobules with the branches of the portal vein, and then uniting with neighboring vessels form conduits which themselves unite to constitute the hepatic duct.

There is also a lymphatic reticulum in the lobule of the liver which gives origin to perilobular lymphatic vessels accompanying the portal ramifications.

The hepatic artery, the nutritive vessel of the liver, furnishes nutriment to the lobules and to the walls of the veins and of the bile ducts.

As a result of important anatomical and pathological investigations, Sabourin has come to the conclusion that the liver should be regarded as a tubular gland. He regards the hepatic gland as a biliary gland, the centre of the lobule of which is occupied by an excretory canal. The space occupied by the interlobular veins would be, therefore, the centre of the lobule, while its periphery would be surrounded by efferent vessels, that is, by the subhepatic veins. These novel views cannot, however, be accepted without further confirmation.

Structure of the Bile Ducts.—The interlobular biliary canaliculi formed by the union of biliary capillaries are composed of a membrane, amorphous externally, and provided with a polygonal epithelium on its internal surface. As the vessel increases in volume it acquires an adventitia, and the pavement epithelium becomes cylindrical, the adventitia next becomes fibrous, and the amorphous membrane acquires fibrocellular muscles and elastic fibres. When the canals have attained a diameter of half a millimetre the internal wall begins to show minute openings, the mouths of little glands,

only by our rather limited knowledge of the general pathology of the liver, but also by many clinical symptoms which could not be otherwise interpreted except by this mechanism. Biliary lithiasis, some forms of icterus, cirrhosis, to mention only the most striking, are dependent upon the chemical alteration of the chyle, as has been quite clearly demonstrated by the latest researches and by a correct inductive interpretation of their pathogenesis.

Bile-Forming Function.—This, the oldest known function, was for a long time held to be the only mission of the hepatic gland, but is now, in consequence of many and accurate studies, regarded as a secondary one. The bile, which was once thought to be a product of secretion of the biliary glands distributed along the course of the bile ducts, is to-day, despite the researches of Robin, Legros, and others, regarded as a direct product of the hepatic cells which elaborate it from the materials brought to the liver in the blood of the portal vein. This is not a simple separation from the blood of matters contained in it, but is a true chemical elaboration produced by the hepatic cells which undergo histological changes during digestion (Heidenhain, Kayser). It is only by the hepatic cells that this transformation of raw material contained in the blood into biliary products takes place; and the recent researches of Löwit, who endeavored to show that the pigment of the blood may be transformed into bile pigment in other tissues as well, in the case of fishes, have been completely refuted by the experiments of Leyden, Stern, and Valentini.

The quantity of bile secreted in the twenty-four hours was estimated by Wittich at 553 c.c. (18½ oz.), by Westphalen at from 453 to 566 c.c. (15 to 18¾ oz.), by Ranke at 652 c.c. (21¾ oz.), and others have given even higher figures than these (1,500 c.c. or about 3 pints). Its secretion takes place without interruption and it is accumulated in the gall-bladder to be poured out more abundantly into the duodenum during digestion. The bile is a transparent, yellowish-brown liquid, of bitter taste, of neutral reaction, with a specific gravity of 1.026. It is composed of mucus, of two acids (taurocholic and glycocholic), coloring matters (bilirubin and biliverdin), cholesterin, and inorganic matters (salts of potassium, calcium, magnesium, and iron). It is very important to remember that this liquid never contains albumin in the normal condition, which is further confirmatory of the fact that the function of albumin is a purely intraorganic one, that is to say, that it is never destined to be excreted from the organism since its chemicomolecular constitution permits of its complete assimilation. And as a further proof of this physiologico-pathological law of the albuminoids we may re-

mark that albuminocholia exists in Bright's disease, and in all cases in which albuminoid dystrophy causes the elimination through various emunctories of that part of the albuminoids which has become diffusible and unassimilable. It has strong toxic porperties, nine times greater than those of urine, this toxicity being due to the presence of bile acids and bile pigments. Rabbits poisoned by it show symptoms of marked nervous excitement (Bouchard).

The action of bile in digestion is very complex: 1. It emulsifies fats and renders them capable of chemical decomposition and of absorption through the mucous membrane of the small intestine; 2. It stimulates the muscles of the intestine, and in this way promotes absorption; 3. It renders the intestinal epithelium better adapted to diffusion, for an animal membrane moistened with bile is made more fit for osmosis between two liquids of different densities; 4. By stimulating the muscular fibre it increases peristalsis and thus accelerates the flow of fæcal matter; 5. It moderates the decomposition of the intestinal contents, especially when much fatty food has been taken; 6. It suspends all peptonization of the chyme coming from the stomach, precipitating the pepsin, which would destroy the pancreatic ferment, which is such an important factor in digestion; 7. Fresh bile contains a little diastasic ferment which changes starch and glycogen into sugar. All these factors are important to good digestion, for chylifaction as well as absorption are gravely disordered when the secretion of bile or its passage into the duodenum is prevented by any cause.

The Formation of Urea.—The formation of urea, formerly regarded as a property of all the tissues, has recently, as the result of a great number of interesting investigations, come to be recognized as residing principally in the liver. The discovery of this important fact is due to Meissner, who found a greater quantity of urea in the parenchyma of the liver than in the other organs, and Cyon showed that the blood of the suprahepatic veins was more charged with urea than that of the portal vein. The experiments of Schroeder and Salomon are celebrated; these maintained an artificial circulation through the liver and obtained a transformation of ammonia into urea during the passage of blood through the liver. Another well-known experiment was that of Stolnikow, who applied electricity to the liver in man and dogs and obtained thereby an increased quantity of urea in the urine. These interesting physiological discoveries are borne out by accurate clinical observations. Meissner himself, studying the composition of the urine in acute yellow atrophy, found an enormous diminution of urea, and Bouchardat found that certain lesions of the liver were accompanied by a diminution of urea, while

others were accompanied by an increased excretion of this substance. These facts have been confirmed by many others, and Murchison, Frerichs, Brouardel, Regnard, Genevoix, Charcot, Semmola, and others have shown that the liver is one of the chief laboratories for the production of urea, that this is one of the most important functions of the hepatic cells, and that it is possible to draw conclusions as to the functional activity of the liver from the quantity of urea excreted. Thus in conditions of simple irritation of the liver there is an increased daily elimination of urea, while destructive lesions of the gland cause a marked diminution.

But another factor enters into the production of urea, one which is an important condition in all secretions, namely, blood pressure and the velocity of the blood current. The more active the hepatic circulation and the greater the intravascular pressure, so much the greater is the amount of urea eliminated during the twenty-four hours, provided always that this last factor is not associated with a pathological condition which may cause a disturbance of function. It must not be thought, however, that the liver is the only seat of formation of urea, as was believed for a certain time through a not uncommon exaggeration of clinical and experimental data. Indeed, urea, which represents the complete product of organic combustion, may be formed wherever nitrogenous matter is consumed, a process which takes place in all the organs, especially in the parenchymatous organs. Thus Lecorché and Talamon have found a large quantity of urea in the spleen, Picard and Wurtz have found it in large proportion in the lymph, etc.

The fact remains, however, that the liver occupies the first place in the production of urea. To us this appears to be due to the fact that the circulation within the liver is slower than elsewhere and the blood which flows through the organ is charged with a greater quantity of oxidizable nitrogenous substances, the products of digestion, which there undergo complete combustion. Furthermore, it may also be assumed that the liver is destined to transform into urea the products of incomplete oxidation of tissues (creatin, xanthin, etc.), products which, although formed in other parts of the organism, are finally transformed into urea under the influence of the hepatic cellules. When understood in this sense, that urea may result from metabolic changes in any of the cells, but that its formation takes place to greater amount in the liver, this theory avoids the exclusiveness of that of hepatic urogenesis, held by many physiologists, and also the denial of any participation in the formation of urea by this organ, a theory which is sustained by others to the no small injury of the interpretation of many pathological symptoms.

We must not omit all mention of the latest researches of Minkowski and Schroeder, which have shown that the liver, especially in birds, is the principal seat of the formation of uric acid which, according to Minkowski, is a product of synthesis of ammonia with lactic acid. It does not appear, however, that these facts hold good in the case of mammals, but rather that they are limited to birds, in which, as is well known, the principal derivative of the oxidation of nitrogenous substances is not urea but uric acid.

Glycogenesis.—We owe to the immortal Claude Bernard the discovery of this most important function of the liver. The presence of glycogen in the cells of the parenchyma is a fact that is now accepted by all, and some of the laws which regulate the production and the transformation of glycogen into sugar are already recognized. There are various bodies from which this substance may be formed; the carbohydrates of the food (Pavy), the fats (Salomon), glycerin (Weiss), taurin and glycin (Heynsius, Kuthe), the albuminous substances (C. Bernard), and gelatin (Salomon), will produce in larger or smaller amounts this special carbohydrate called glycogen, or animal starch. It diappears from the liver during prolonged abstinence from food, it diminishes during severe muscular exercise or under a diet composed exclusively of albumin and fat, but increases, on the other hand, under a diet composed chiefly of carbohydrates. Glycogen appears to represent in metabolism a reserve food, which, stored up in the hepatic cells, is transformed by them when needed into glucose and then poured out into the circulation. The suprahepatic veins contain blood with a higher percentage of sugar than does the portal vein. This sugar circulating in the blood is in a condition fit for organic combustion, thus contributing to general nutrition and to thermogenesis.

This transformation of glucose into glycogen in the liver and the conversion again of glycogen into glucose is continuous during life, and there is always stored up in the hepatic tissue a certain quantity of reserve material adapted to the nutritive needs of the organism which can be used to supplement ordinary nutrition during periods of special need, as in fasting or during prolonged labor. But after death the formation of glycogen from sugar, which is a vital process, is arrested, while the transformation of glycogen into sugar by means of the special ferment, which is a purely chemical process, continues. This is the explanation of the fact which has been observed when an artificial circulation was maintained in the liver removed from the body, that the fluid coming from the suprahepatic veins always contained for quite a long period traces of glucose.

Many writers, such as Schmidt and Rouget (who found glyco-

gen in small quantities even in the muscles), have endeavored to prove that glycogenesis is a common property of all the tissues and not of the liver alone, but it nevertheless remains a fact that the liver forms and stores up glycogen in large quantities, which it then returns to the circulation little by little, transformed again into sugar. The small quantity of glycogen contained in the other tissues may be regarded as a secondary fact, depending most probably on special biochemical processes of nutrition, and not a constant fact, as is hepatic glycogenesis.

Depurative Functions.—The facts observed by Lussana, who demonstrated that curare injected hypodermically is much more toxic than when ingested, and by Heger, who found that the liver retained from twenty-five to fifty per cent. of the alkaloids passing through it, may have first led to the thought of the liver as a protective organ, but we really owe to the interesting studies of Schiff the discovery of this important function of the liver. He showed that poisons are less active if injected into the portal vein and that the lethal dose of a poison is lower in frogs in which the portal vein had been ligated, and that the trituration of liver substance with certain alkaloids diminishes their toxic properties. These results were very soon confirmed by Lauterbach, who demonstrated, furthermore, that a single drop of nicotine was sufficient to kill a large dog when it was injected into the general circulation, but that it was possible to introduce two drops into the mesenteric circulation without destroying the animal.

In 1877 René wrote a thesis opposing the theory of the depurative action of the liver. But new and more extensive investigations made by Lussana, Jacques, Heger, and Roger demonstrated most completely that the liver arrests and destroys almost the half of the poisons circulating through it, thus protecting the organism from their effects. Among these are the vegetable alkaloids, salts of iron, of copper, and of ammonia, which meet with a barrier in the liver, in surpassing which they lose much of their toxic properties.

Very recently Gioffredi,[2] studying the protective power of the liver in acute and chronic alcoholism, has demonstrated very conclusively (1) that alcoholic poisoning produced experimentally in normal frogs and in those from whom the liver had been extirpated, was always more intense, more rapid, and more fatal in the latter; (2) that an emulsion made from the liver of a frog poisoned with alcohol would reproduce in healthy frogs and still more in those from whom the liver had been removed typical symptoms of alcoholism; (3) that this process of arrest is much more marked when the alcohol is given by the stomach than when it is injected hypodermically; (4) that the toxic property of a distillate of the liver of an animal poisoned by

alcohol is very marked, and may cause all the symptoms of alcoholic poisoning in those frogs; (5) that the protective action of the liver is more marked if the alcohol be given in daily small doses, so as to cause subacute poisoning; (6) that in cases of chronic poisoning, if the administration of alcohol is suspended for a certain time, the liver gradually empties itself of it either in substance or partly oxidized.

What is the mechanism of this protective function of the liver? Is there an elimination of the poison through the bile, a transformation of it into other innocuous products, or a simple arrest? Although the experiments of Schiff would seem to point to a true transformation of the toxic product, yet the researches of Jacques and of Gioffredi have demonstrated very clearly that the protective function of the liver is due to an arrest of the poions introduced and which are later eliminated through the bile in fractional quantities, which are not sufficient to cause appreciable modifications in the various organic functions. But the liver is not only capable of arresting the poisons introduced into the animal economy, it also arrests the toxalbumins produced by pathogenic bacilli in the organism. Roger, in the case of toxins of putrefying muscles and of the typhoid poison, Charrin, in that of the products of the blue pus bacillus, Camara-Pestana, in that of tetanus, have shown that the liver is capable of destroying the toxic properties. From all this is evident the importance which the liver possesses in the defence of the organism from infections and autointoxications. Placed between the intestine and the general circulation, commanding the entrance and the passage of the products of digestion absorbed from the intestine, it is in position to exercise its protective functions, against both the toxic products coming from without and those formed within the organism.

Hæmatopoietic Function.—It was long held that the liver was the chief blood-making organ; Lehmann was the most strenuous advocate of this theory, which is, however, now regarded as very problematical. It would seem, on the contrary, as though the liver were adapted to the destruction of the old blood corpuscles, which are no longer capable of taking part in the nutritive changes in the tissues. The analogy between hæmatoidin and bilirubin would tend to add weight to this opinion, and all the classical experiments of Schulz, of Kandl, and of Flügge, also support it and oppose the theory of the hæmatopoietic function of the liver. The researches of Foà and Salvioli into the origin of the red blood cells would deny to the liver the hæmatopoietic function, although it would appear that it does possess such a function in intrauterine life, gradually losing it until it finally disappears about the sixth month after birth.

Action of the Liver upon the Albuminoids and Peptones.—It would appear as the result of some researches that the albuminoids absorbed through the intestine undergo a certain modification in passing through the liver, which renders them more assimilable, and these researches have also been confirmed experimentally. Claude Bernard injected egg albumin into the jugular vein of a rabbit and found it excreted in the urine, while that injected into the portal vein was not so excreted. Bouchard injected casein into the jugular vein and found both casein and albumin in the urine, but only albumin when the casein was injected into the portal vein, as this substance is transformed into albumin in the liver.

Bouchard and Roger have made some most important researches into the action of the liver upon peptones. They found that when peptone was injected into a branch of the portal vein, neither peptone nor albumin was found in the urine, whereas if it was injected into a peripheral vein, both peptonuria and albuminuria occurred; thus proving that a portion of the peptone may be transformed outside of the liver into an albumin which is, however, not assimilable. Plosz and Gyergyai have shown in artificial-circulation experiments that peptone passing through the liver is transformed into albumin, while Seegen believes that it is transformed into sugar, and Arthaud and Butt that it is transformed into fibrinogen.

More extensive and closer investigations are called for here, for while it is true that cases of albuminuria and peptonuria are encountered in certain morbid states of the liver, we do not yet know either the circumstances or the mechanism of their production. We may here recall the fact that all these researches have only confirmed an opinion published some time ago by Semmola[3] in regard to the influence that the liver exerts upon the albuminoids of digestion. Resting chiefly upon the studies of Bernard and also upon a number of clinical facts, he concluded that the action of the liver was indispensable in rendering assimilable the albuminoids of digestion, that is to say, the transformation of diffusible albuminopeptone into non-diffusible albumin (serumalbumin) which is the form that serves the needs of physiological cellular activity. And it is the diffusibility of the serin resulting from effective hepatic function which is, according to Semmola, the fundamental condition of that dyscrasia of the albuminoids which necessarily results in the elimination of albumin by the urine, which is the principal symptom in Bright's disease.

We have since endeavored to demonstrate experimentally the theory of Semmola just mentioned. Blood was taken from the portal and suprahepatic veins of a large dog during digestion, and

placed separately in dialyzers under all the precautions necessary in researches of this kind. At the end of twenty-four hours dialyzable albumin in tolerably large amount had passed out of the portal blood, while from the blood of the suprahepatic veins no albumin at all, or at most mere traces, had passed through the dialyzer. This demonstrates conclusively that the principal effect exerted upon the albuminoids by the liver is in regard to their diffusibility, which is reduced in such a manner as to limit them to a strictly intraorganic function.

Great importance has been laid by some upon the formation of fat in the liver. But this is not an exclusively hepatic function, nor does it throw much light upon the study of functional changes in this organ.

From this brief review of the physiology of the hepatic cell it may be seen that all the various functions to the performance of which the liver is adapted may be grouped together under two principal heads: 1. The prevention of waste, and 2. depuration.

The formation of glycogen and of fat should, indeed, be regarded as economical functions; the liver is in this respect a storehouse for the deposits of surplus nutritive materials which are not immediately necessary to vital activity, and is thus always ready to make up any deficit which may occur in the animal economy. Thus there is in the liver the entire surplus of the products of intestinal absorption remaining after nutritive changes in the cells and organic combustion; and when, as a result of increased muscular action or after prolonged fasting, there arises a deficiency of fuel for oxidation in the tissues, this surplus deposited in the liver, now transformed into easily oxidizable products, enters into the circulation in order to make good the sudden loss. If, as has been aptly done by others, we compare life to a steam engine, the liver would represent the tender, always filled with materials for combustion which it has obtained from intestinal absorption.

On the other hand, the formation of urea, the protection against poisons, and the destruction of red blood cells constitute an emunctory function very similar to that of the kidney. The formation of urea from incompletely oxidized nitrogenous compounds which are formed by biochemical processes in the cells is the final step in the elimination of these products, which are not only useless but even injurious to life.

Urogenesis is, therefore, very nearly related to the action of the liver in arresting and eliminating poisons coming from without or those which are formed in the intestine, and as a result of chemical changes within the cells. In either case there is a freeing of the organism from products which are useless or harmful to it, and which,

if they should remain and accumulate, would act very injuriously upon the tissues, even producing grave disturbances in their chemical constitution and in their functional powers, disturbances which might readily lead to the death of the individual.

The destruction of the old red blood corpuscles, which is accomplished in the hepatic cells, ought also to be regarded as an eliminative function. These corpuscles, indeed, no longer capable of accumulating oxygen in the pulmonary cavities or of readily yielding it to the cells and to the tissues, have become incapable of fulfilling their biological destiny and thus represent a sort of ashes composed of morphological elements which are no longer adapted to their functional ends.

The bile represents the product of this complex chemical laboratory in the hepatic cell, the purpose of which is to assist in the digestion of food.

Complex and multiform as is the function of the liver, it consists especially in a chemical process which is, moreover, always at the bottom of cellular activity. But we need not suppose that there is any dissociation in the hepatic functions, as might at first be supposed. There is not within the liver cells a series of independent chemical operations, but all the steps of this single but complex process are intimately united to each other, that is to say, combustion, hydration, reduction, and duplication. Of this chemicomolecular laboratory we know only the ultimate products, the bile, urea, glycogen, destruction of red blood cells, arrest of poisons; but we do not know the intermediate steps, that is to say, the derivation and all the stages of transformation of these various substances. All these biochemical functions of the hepatic cells are closely bound to each other, like the links of a chain, as is continually shown by clinical and pathological facts.

GENERAL PATHOLOGY.

Just as the physiology of the liver is obscure so is its physiopathology still in an embryonal state and can become fully developed only when our knowledge of the fundamental laws on which its functions depend shall have become widened. In the present section we shall treat only of what is certain and has been demonstrated pathologically, clinically, and experimentally, as far as concerns the general pathology of this organ, and which therefore can be advantageously employed in an interpretation of the various morbid phenomena of the liver.

Peculiarities of the Hepatic Circulation and Their Importance in the Pathology of the Liver.

The liver is the richest in blood of all the organs of the animal economy, about a fifth of the entire mass of blood being in it, as Sappey has shown. The vascular network which contains this great quantity of blood is a double one, viz., a portal network, which may also be called functional, since it is concerned in all the different functions of the liver, and an arterial or nutritive network, which is adapted to the nutrition of the parenchymatous and interstitial elements of the organ. By reason of this complex anatomical constitution of the vascular network, and by reason of the peculiar position of the organ, the hepatic circulation differs notably from that in other parenchymatous organs, from which also result important peculiarities in the pathology of the liver. The blood pressure in the hepatic artery undergoes modifications which are exactly the reverse of those taking place in the aorta; it is increased during inspiration because abdominal pressure is increased by that act, and on the other hand it is diminished during expiration in consequence of the raising up of the diaphragm and the resultant lessened abdominal pressure. In the portal venous system the pressure is very low, varying from 7 to 24 mm. of the column of mercury; and it is even lower in the hepatic veins, varying from 4 to 5 mm. The result of this is that the blood can pass very readily from the portal vein to the vena cava, when there is no obstruction to its flow within the liver.

The respiratory movements assist greatly in accelerating the movement of venous blood through the liver. Thus, during inspiration the pressure in the ascending vena cava diminishes and consequently also that in the hepatic veins, while the portal pressure increases because of the compression of the abdominal contents through the sinking of the diaphragm. During expiration, on the other hand, pressure in the hepatic veins increases, while that in the portal vein diminishes; the vascular network in the liver, however, will be filled with blood under greater pressure.

Other factors also contribute to facilitate the hepatic circulation; among these is the continued patency of the hepatic veins even when the intrahepatic pressure is increased, the rhythmical contraction of the trunk of the portal vein, intestinal peristalsis, and the aspirating action of the diastole of the right auricle which produces a negative pressure in the vena cava.

We must not omit mention of the influence of the nervous system which affects the circulation in all the organs, the liver among the

rest. Claude Bernard, in his well-known experiment, found marked hyperæmia of the liver after a puncture of the fourth ventricle, a hyperæmia which did not seem to be due to paralysis of the vasomotor nerves, but to a stimulation of the vasodilators, for it did not last longer than twenty-four hours. The vasodilator fibres which have their origin in the bulb emerge with the first dorsal roots to form with the sympathetic the splanchnic nerves.

Corresponding with this complex circulatory condition in the organ and with the many causes which influence the hepatic circulation there are many and complex etiological factors which contribute to its disturbance. The immediate and direct dependence of the hepatic circulation on the cardiac and thoracic movements explains the frequency of hyperæmia of this organ when there is any interference with these movements. Impeded expansion of the thorax in consequence of various diseases of the lungs or pleura causes a certain degree of blood stasis in the liver, since the differences in pressure of the blood in the vena cava and the portal vein, which are so essential to the circulatory equilibrium, no longer exist, or do so at most in lessened degree.

Again, functional weakness of the cardiac muscle is a most important etiological factor in circulatory disturbances of the liver, this dependence being so absolute that the liver is regarded as compensating in some degree for the impeded general circulation. It is in fact very evident that an increased venous pressure being the final result of all cardiac weakness, a greater quantity of blood will be accumulated where the venous network is most extensive and where the pressure is less. This is precisely the reason for the hyperæmia of the liver which is so constantly observed in, and which sometimes leads to the detection of a latent valvular disease of the heart. The liver is the first organ to feel a disturbed compensation in a valvular lesion, and it shows it in an increase of volume caused by the passive hyperæmia, the pressure in its veins being increased at a time when circulatory disturbances have not yet become evident in the other abdominal organs or in more remote parts of the general circulation. Nor do these circulatory modifications always remain as such, for, constantly increasing, they may cause special pathological changes.

It is also important to consider the circulatory relations which the liver, by means of the vena porta, has with the vessels of the intestine and spleen. The portal radicles derived from the superior and inferior mesenteric and splenic veins convey to the liver all the venous blood from the intestine and spleen, thus uniting these organs by close vascular bonds. Every obstacle, therefore, to the portal

circulation will be reflected directly back into the abdominal circulation, producing more or less marked stasis. Obstacles of whatever sort, extrinsic as well as intrinsic to the portal circulation, or narrowing by morbid processes of the most varied sort which the portal ramifications may undergo in the parenchyma of the liver, being a cause of increased pressure in the portal radicles, will cause passive hyperæmia of the spleen and an exudation of serum in the peritoneal cavity, resulting in ascites.

To these facts of general pathology there are two important clinical corollaries, and the practical physician should never forget that when the portal flow is obstructed two results are produced, the one passive hyperæmia of the spleen, and the other ascites. The presence of these two symptoms and their complex relation to impeded circulation in the portal vein and in its hepatic ramifications will be a most important aid to the diagnosis of certain morbid processes in the liver which might be confounded with peritoneal troubles.

Finally, we must not omit, in studying the circulatory changes in the liver, to note the part which intestinal peristalsis takes in facilitating the passage of the portal blood, or the disturbances of cerebro-spinal innervation which may cause active arterial congestion of the liver. Indeed, we often observe clinically passive hyperæmia of the liver caused by intestinal torpor, or, on the other hand, active hyperæmia due to disturbances of nerve function.

Relations of the Liver and the Intestine.

So intimate are the embryological, so close the anatomical, and so important the functional relations between the intestine and the liver that this organ was for a long time looked upon as an intestinal gland, like the pancreas, salivary glands, etc. Arising as a simple duodenal diverticulum during the first days of development of the embryo, it remains up to its period of complete development intimately united to the intestine in two ways, both circulatory and secretory.

Of the first of these we have already spoken in a preceding paragraph as far as regards the general pathology of the circulatory disturbances of the liver, but it is not less interesting to consider what takes place in the intestine when the portal circulation is impeded by any cause whatever. The passive congestion of the radicles of the portal veins which results from this obstruction, produces in the intestinal mucous membrane a condition but little adapted to the performance of its functions. The accumulation of venous blood results in a nutritive disturbance of the mucosa, shown by a slight hyperplasia of the epithelium, which is much more perishable than nor-

mally, and by a functional alteration in the glands of the stomach and the crypts of Lieberkühn in the small intestine from which there is a hypersecretion, and there is also an exaggerated production of mucus. Of no less importance is the venous stasis occurring in the pancreas, the veins of which empty into the superior mesenteric and the splenic. The result of venous congestion of this gland is a diminution of its secretion, so important in the digestion of proteids, fats, and carbohydrates. Parallel with these important factors which interfere with gastric and intestinal digestion, the increased pressure in the venous radicles of the intestine offers a direct mechanical impediment to absorption by the veins as well as to that by the lymphatics, since the latter, running in general alongside the venous trunks, are compressed when the latter become swollen with blood. But the results of venous stasis are not limited to these changes, since the delayed chemical processes in the digestive tract may cause abnormal gastroenteric fermentations with the development of various products, often gases, which irritate the mucous membrane and may thus produce secondary disturbances.

We must not omit mention of another fact which explains how sometimes symptoms of gastrointestinal catarrh may precede and dominate those of a grave hepatic disorder. In addition to the changes in the intestinal mucous membrane resulting from the fermentation of the food, there may be opportunity given to the bacteria present to exercise their pathogenic action; these micro-organisms, already existing in great numbers in the gastrointestinal tract or introduced from without in the food, may possibly acquire pathogenic qualities in consequence of their changed surroundings which they would not otherwise have, or, secondly, they may become developed in consequence of the absence of normal gastrointestinal secretions. In this way is explained the fact that a true gastrointestinal catarrh may result from circulatory disturbances in the liver. But in this case there happens what is frequently observed in pathological physiology, namely, that a pathogenic cause of a process induces effects which are themselves also causes which aid in the production and increase of this process. It is precisely for this reason that in the diagnosis of functional gastroenteric disturbances of obstinate and rebellious character the physician ought never to forget to look for diagnostic points which may reveal the insidious and as yet concealed disturbances of hepatic function as the true cause of the digestive disturbances.

But the stasis does not always remain within these limits, for when the impediment to the portal circulation has become very great the final consequences of interference with the venous circulation

may be diapedesis or a true hemorrhage. Thus in advanced cirrhosis there may be a passage of red blood corpuscles through the walls of the capillaries which are altered in consequence of disturbed nutrition, or the rupture of these walls may give place to hemorrhage which sometimes becomes uncontrollable and results finally in death. But it is not always extreme disturbances of the portal circulation which determine the occurrence of gastric or intestinal hemorrhage, for sometimes this may occur in an early stage of circulatory disturbances in the liver, produced by some cause which has hitherto evaded the closest study. In such cases it is possible that the cause of the hemorrhage in the digestive tract may be a local affection which itself produces secondarily the lesions found in the liver.

The liver and intestine are also intimately related by means of the bile channels. The ductus choledochus, the cystic and hepatic ducts, the gall-bladder, and all the ramifications of the biliary ducts, even the intralobular capillaries may be considered, and embryology authorizes such a view, as a diverticulum of the intestine itself, as, that is to say, a continuation of its mucosa. There is thus a close anatomical and physiological connection, and, in consequence, the pathological relations are also very close.

We shall not speak here of the results of obstructed flow of bile into the intestine, the so-called intestinal acholia, for this will be more properly considered in the section on jaundice. It will be interesting, however, to note here the morbid processes which may arise from extension to the biliary passages of those inflammatory or neoplastic processes which often take their origin in the small intestine. It has long been observed clinically that in intestinal catarrhs, especially those localized in the first portion of the tube, that is to say, in the duodenum and jejunum, the morbid process may extend by continuity to the mucosa of the biliary canals and cause there a catarrh of the bile ducts with its secondary morbid phenomena. We ought also not to forget the possibility that malignant neoplasms of the bile ducts may originate from the propagation of a primary neoplasm of the duodenum, involving Vater's ampulla.

Very many and important etiological considerations arise in the study of the relations between intestinal infections and morbid processes involving the liver. The small intestine, and especially the duodenum, always contains a very great variety of micro-organisms; the staphylococcus pyogenes aureus, the streptococcus pyogenes, and the bacterium coli are found here almost always under normal conditions, and many other both innocuous and pathogenic bacteria may come from without. If these bacteria do not find favorable conditions for their development no infective process will result. Two main facts

demonstrate that the origin of an infectious morbid process is due to the favorable conditions surrounding the micro-organisms; thus many bacteria of great virulence are destroyed in the intestine when the intestinal juices are normal, without the animal economy suffering in the slightest degree; this is seen in the case of the typhoid bacillus, the cholera spirillum, etc. Furthermore, some bacteria which are entirely innocuous in a condition of health, such as the bacterium coli, may acquire pathogenic qualities and thus cause true infections as a result of modified surroundings. It is a fact of great importance, upon which we have always insisted, and one which the practical physician ought never to forget if he would have a secure and rational guide in the treatment of infections, that in these the soil represented by the biochemical conditions of the tissues and of the intraorganic surroundings is of much more importance even than the pathogenic micro-organism itself, for the latter, when the organic defence is in physiologically normal condition, may remain innocuous, while if the intraorganic conditions are favorable to its implantation and growth there may easily arise an infection, the etiological element of which is found diffused throughout the external world.

In case of an intestinal affection the channels of its propagation to the liver are, as has been said, the blood-vessels and the bile ducts. The conditions for the origin and development of an infection coming through the blood channels are always most favorable. Indeed the circulation of blood within the liver is much slower than that in any other organ or tissue, not excluding the spinal cord. If finely powdered cinabar is injected in the blood of a rabbit it will still be found in the liver at a time when it has already disappeared completely from the general circulation. On the other hand, the hepatic capillaries are exceedingly minute, more so than those of the brain or lungs, so that there may be true microbic emboli in the portal ramifications which will then give origin to an infectious process. But along with these conditions, which are so favorable to infection, there exists a true defensive means, which consists in an increased phagocytic power of the endothelial cells of the hepatic capillaries. Guarnieri[1] has studied this in malarial infections and has found that the phagocytism is exerted not only by the endothelial cells but also by the stellate cells of Kupfer. The occurrence of an hepatic affection, however, favored as it is by the circulatory conditions and impeded by the phagocytic powers of the cells, becomes possible especially when the latter are found either anatomically or functionally altered, as occurs in hepatic insufficiency which we shall study presently.

The bile channels also represent a means of infection from the intestine. For a long time the bile was thought to have a certain anti-

septic power, although cases were reported of transmission of an infectious disease by means of the bile (Morgagni, Balocchi, Deider), but gradually a disbelief in the antiseptic properties of bile has gained ground, until at the present day there is no one who believes it to be endowed with parasiticide properties. Thus the studies of Bufalini,[5] Linderberg, Charrin, and Roger[6] have shown that the bile modifies in no respect the virulence of cultures, and that even if it does exert a slight antiseptic effect in acid media, it exerts none whatever in alkaline media, such as the intestinal contents. More recently Bernabèi[7] has succeeded in growing, in culture media containing normal bile, the bacillus of Eberth-Gafki, the pneumobacillus of Friedländer, and more vigorously than usual the micro-organism of glanders, while Létienne[8] has obtained a very vigorous growth of the bacterium coli, especially in the bile of certain patients with pneumonia.

But although there is no bactericidal property in bile, the fluid is nevertheless completely aseptic in the normal condition, although the intestinal contents contain vast numbers of bacteria. The experiments of Netter,[9] of Duclaux, of Escherich, of Vignal,[10] and of Dupré[11] have shown that although the intestinal tube contains a great variety of micro-organisms, such as the staphylococcus pyogenes aureus and albus, the streptococcus pyogenes, the bacterium coli, etc., the ductus choledochus and the bile contained in it are perfectly aseptic, except for a very short distance above the duodenal extremity. On the other hand, Létienne has studied the condition of the bile in many individuals dead of various diseases and in twenty-four out of forty-two cases has observed the presence of micro-organisms, either the same which had caused the death of the patient (pneumococcus, typhoid bacillus, or streptococcus) or other bacilli, such as the staphylococcus citreus, the streptococcus, other varieties of cocci, the bacterium coli, etc. These facts show that the infection of the bile occurs very readily and that only a few unfavorable conditions need exist in order that it lose its aseptic state.

A question now arises which is of great importance in its physiological bearings: How comes it that the bile, not being an antiseptic medium, and the liquid column being in direct relations with the interior of the intestinal canal so charged with micro-organisms, is always in physiological conditions sterile? It is impossible to answer this important question satisfactorily, for as yet only one of the reasons for this fact is known. This is the fact that the continual flow of a sterile liquid into an infected region serves in a measure to keep the column sterile, but this cause alone cannot be invoked in the case of the bile, since physiology teaches us that the discharge of bile into the intestine occurs with many irregularities and intermissions. It

is least during fasting, the fluid being then accumulated in the gall-bladder, and is increased during digestion, especially from the third to the fifth and from the thirteenth to the fifteenth hour after taking food. The asepsis of the bile is therefore due to other conditions which thus far have eluded physiological investigation, although it must be admitted that the first factor is one of importance, since the conditions which alter the flow of bile are also a cause in another sense of its infection. Biliary stasis is the most frequent cause of infection of the bile passages as has been shown by the researches of Gombault and Charcot,[11] who found vibrios in the bile ducts after they had ligated the ductus choledochus, and by the classic experiments of Netter[12] and of Duclaux, who demonstrated that when the ductus choledochus was ligated two or three centimetres above the ampulla of Vater the bile remained sterile, while it became infected whenever the duct was ligated immediately above Vater's ampulla, that is to say, when a small portion already infected was cut off above the ligature. These experimental results are readily explained when the ductus choledochus is ligated above the microbic tract; its contents and the walls of the duct are thus shut off from any parasitic invasion, and although the best condition for biliary infection, such as is stasis, now exists, yet no infection can take place, since the pathogenic agent cannot pass the artificial barrier. When, however, the duct is ligated near the ampulla of Vater, a portion containing microbes is left above the ligature, and now the stasis which results favors septic changes in the bile induced by the micro-organisms which are found in the lower portion of the column. These experimental conditions may be reproduced pathologically in the biliary passages. They may become obstructed, indeed, in various points, and when this is in the neighborhood of the ampulla of Vater biliary infection occurs very frequently. We must not, however, believe that no infection of the bile can take place when stasis occurs through obstruction of the channels at a distance from the intestine, for infection may also arise through the blood channels. Of this we shall speak more at length when we come to discuss the relations between the liver and the various forms of infection.

Relation of the Liver to Autointoxication.

The protective action of the liver is shown most markedly in intestinal intoxication. In normal digestion the final absorbable products which are formed are the soluble albuminoids, emulsified fat, glucose, and maltose, while in indigestion a great number of aromatic and nitrogenous compounds are formed from the albuminoids and also many acid substances, such as lactic and butyric acids, etc. It

has been shown by Albertoni and others that if the albuminoids and carbohydrates reach the large intestine without having been transformed into soluble products they undergo putrefaction. We must not, however, suppose that normal digestion is not accompanied by the production of compounds which are injurious to the organism. It is difficult, if not impossible, to believe that in the complicated intestinal laboratory, especially with such a quantity of nutritive material of complex chemical composition and containing a large quantity of inassimilable bodies, many aromatic and toxic volatile acid compounds will not be produced; these are formed by the chemical action of the gastrointestinal juices as well as by the decomposing action of the many bacteria present in the intestine. There are various gradations between normal digestion and pathological indigestion which escape our means of estimation and which prevent us from setting any limits between them. The protective action of the liver is therefore a function of the organ even under conditions of health, and is greatly increased whenever, under morbid conditions, the production of toxic substances in intestinal digestion is increased. The decomposition of alimentary substances may take place even in the stomach, when there is an absence or diminution of free hydrochloric acid. In the small intestine the albuminoids, even in cases of altered digestion, do not undergo decomposition, the action of microbes being exerted especially on the carbohydrates. Thus from the sugar may be formed lactic, acetic, succinic, and carbonic acids, ethylic alcohol, and hydrogen. In the large intestine, on the other hand, occur the decomposition and putrefaction of the albuminoid compounds which have not been rendered soluble by the pancreatic juice. From this decomposition of the albumin are formed indol, scatol, phenol, the aromatic acids, lactic acid, the volatile fatty acids, organic bases, ammonia, methane, sulphuretted hydrogen, methyl, mercaptan, etc. Among the products of intestinal decomposition certain bodies endowed with great toxic power are deserving of special mention. One of these is peptin or peptinotoxin (Albertoni and Brieger), a body of ill-defined chemical composition which renders the blood incoagulable, lowers the blood pressure, and paralyzes the nervous centres. From the decomposition of albumin there are formed also in the intestine leucin and tyrosin, which exercise no toxic effect upon the organism but which are of great importance because of their ready convertibility into urea, and the xanthin bodies which have a stimulating effect upon the nervous system analogous to that of caffeine.

Certain alkaloids are formed by the decomposition of proteid substances in the intestine; these Gautier called leucomains; among

the best known of them are pentamethylendiamine (cadaverine) and tetramethylendiamine (putrescine). The fatty acids which may be formed in the intestine act injuriously either by the subtraction of the alkalies or in consequence of a slightly narcotic influence which they possess. Finally, more or less injurious gases may be generated in the intestine, such as sulphuretted hydrogen, which Senator has shown may give rise to a morbid condition called by him hydrothionæmia, and methylmercaptan, which is always found in the intestinal contents (Nencki). It is the liver which acts as a barrier to guard the organism against the invasion of these various substances. Under normal conditions, that is to say, when the various bodies above mentioned are not produced or are produced only in small amounts, the liver may transform the toxic products into innocuous substances or eliminate them again by the bile into the intestine in non-absorbable chemical combinations, in this way purifying the portal blood. The first of these methods is seen in connection with leucin and tyrosin, which are transformed into urea in the hepatic cells; the second method is one which we may believe takes place, although we have no certain facts to demonstrate it. In a recent very important work Lugli" has studied the toxicity of the bile before and after ligature of the portal vein. The principal results obtained, which also support the theory of the elimination through the bile of toxic products which the hepatic cells separate from the blood, are the following: 1. The average toxicity of the bile of a dog, collected immediately after death and injected into a rabbit, is about 6.4 c.c. per kilogram of the animal's weight; 2. The toxicity of the bile collected from a fistula is 21.5 for the rabbit and 22.2 for the guinea-pig; 3. After ligature of the portal vein the bile is reduced in quantity, density, and odor; its solid constituents are reduced from 7.89 per cent. to 4.63 per cent., the ash from 0.97 to 0.79 per cent., the fats from 0.23 to 0.20 per cent., and the bile acids from 5.96 to 3.13 per cent.; on the other hand, the water increases from 92.11 per cent. to 95.37 per cent., but the mucin is reduced from 0.70 to 0.48 per cent.; (4) The composition of the solid constituents before and after the ligature differs considerably; after ligature in 100 parts of solid residue we find more ash, more fats, more mucin, and less bile acids and pigments than before the ligature; (5) The toxicity of the bile after ligature of the portal vein is reduced so that to kill a rabbit it requires on an average 51.4 c.c. per kilogram of the animal's weight; but this toxicity tends to increase after a certain number of days and to approach the normal average; (6) The toxicity of the bile in general is in relation with its density and the quantity of its solid residue, but may depend more especially upon the bile acids and pigments; (7)

Decolorized bile before and after ligature of the portal vein has a toxicity at least four times less than that of normal bile. Recently this protective function of the liver has been denied. Quierolo,[15] starting from the principle that the toxic products resulting from the chemical changes in the intestine and absorbed by the portal system are in solution in the blood serum, has sought to study the toxic properties of various exudations, comparing peritoneal effusions following portal stasis with pleuritic effusions, and has found that, in general, the peritoneal fluid was possessed of no greater toxic properties than the pleuritic exudation; and he concludes from this that the portal blood, which has undergone no change by passage through the liver, does not contain toxic principles in quantity greater than the blood of the general circulation which has already felt the influence of the hepatic cells. The same author has also experimented on a few dogs, by means of a modified Eck's fistula, that is to say, the transplantation of the portal vein into the vena cava, and was unable to obtain any symptoms which might suggest poisoning, which ought to have been caused if it were true that the function of the liver was to protect the organism from intestinal intoxications. He concludes that these experiments prove that the theory of depurative function of the liver is incorrect as regards toxic materials produced in the intestine, and give some support to the theory suggested some years ago by Stick, which attributed a protective function to the intestinal epithelium.

But if we examine the experiments of this kind more closely, comparing their results with a great number of clinical and experimental facts, we come gradually to the conclusion that neither the first nor the second sort of experiments is absolutely free from criticism. We know, indeed, that the exudations and transudations have a varying toxic property, according to the quantity and the nature of albumin contained in them, as Rummo has shown; but Quierolo took no account in his experiments of the percentage of albumin contained in the transudations whose effects he studied. It is not proven either that all the poisons are taken up by the blood serum as they would have to be in order to appear later in the transudations. On the other hand, the second experiment, which Quierolo considers of great importance, is not above criticism. The vicarious function of the kidneys, increased through the diminished activity of the liver, may explain why no autointoxication occurs; and it is no argument against this to say that the toxicity of the urine of the dog operated upon is not found to be greater than that of another dog, for there is room for fallacy here when we consider how great individual differences there are in the urotoxic coefficient; we ought to determine the

toxic equivalents of the urine in the same dog before and after operation, in order to determine with certainty whether the kidneys are eliminating a greater amount of toxic products. We may conclude, therefore, from a long series of clinical facts and a vast amount of experimental data acquired by many and skilful pathologists, that, notwithstanding isolated objections, the protective function of the liver in intestinal autointoxication is well proven.

A fact which speaks in favor of this protective function is that when the hepatic cells cannot exercise in normal degree their protective function, either because of some trophic disturbance or because of functional weakness, or perhaps because the products of intestinal chemical processes are brought in too great quantity, toxic substances enter the general circulation and give rise to clinical pictures which have only recently begun to be studied and to which the name of autointoxication or autotoxæmia has been given. We may remark, however, that the presence of these was surmised and their true nature was foretold by one of the present writers (Semmola[16]) as early as 1883, who at that time, speaking of the toxic products which are formed in the organism, wrote as follows: "The rôle of which (toxic products) in the production of a great number of nervous symptoms accompanying these acute diseases is of the greatest importance, and I believe that sooner or later it will give us the key of the morbid succession which up to the present time pathological histology has in vain assumed to explain; for really these nervous phenomena are above all of a chemical and not of a morphological nature; that is to say, they are true poisonings produced by substances which the organism manufactures within itself under the influence of acute infectious processes."

Important considerations also arise from a study of the changes which the toxic products coming from altered chemical processes in the intestine, or from the absorption of alimentary substances, excite in the hepatic parenchyma. We are told indeed by general pathology that the exaggeration of a function may reach such a point as to constitute a cause of disease. Thus excessive mental labor may produce an excitable weakness of the nervous system, that is to say, cerebral neurasthenia; great muscular exertion may cause an exaggeration of the heart's action with consecutive hypertrophy; forced expiratory efforts may result in emphysema, etc. The same thing occurs in the case of the liver; the exaggerated exercise of its organic protective function may be the cause of changes at first functional and later trophic and anatomical. Furthermore, we must give the importance it deserves to another factor which may lead to changes in the hepatic cells, viz., the specific action of the products of absorp-

tion brought by the portal blood. It is in the formation of toxic substances that we must look for the cause of many morbid processes and grave pathological changes in the liver. Alcoholism and plumbism are, indeed, most important factors in the etiology of cirrhosis, poisoning by arsenic or phosphorus gives origin to serious hepatic lesions, such as fatty degeneration, acute yellow atrophy, etc. The cause of certain forms of cirrhosis which cannot otherwise be explained, resides probably in altered chemical processes in the intestine, as we shall see when we come to treat of this disease.

But the protective action of the liver is not exerted only in regard to morbid products of intestinal absorption, since it is seen also in regard to substances absorbed by the general circulation, as one of us (Gioffredi) has shown in the case of alcoholic poisoning caused by the subcutaneous injection of solutions of various kinds of alcohol (ethylic, amylic, propylic). Inducing in this way repeated alcoholic poisoning in kittens, he found a great accumulation of alcohol in the liver, while the other tissues were entirely free from it. This is evidently due to a special elective affinity of the liver for alcohol. It is this elective affinity which, although the true explanation of it is unknown, dominates physiological and pathological processes. This has been established by the bacterio-chemical studies of modern medicine, which have shown that, in the intoxications (through ptomains) which the various clinical forms of infection produce, the various symptoms, whatever be the toxins derived from the specific bacteria, are referable to the various tissues influenced by the toxins. There are very many drugs, also, which, when introduced into the general circulation, are eliminated by the liver. Iron, copper, zinc, silver, lead, mercury, etc., are almost completely removed from the general circulation by the liver; and often when the eliminative function is imperfect there arise grave anatomical changes, a condition which was first noted by Mialhe, and called by him organic stagnation.

If now the liver exerts a protective action in regard to poisons, absorbed from the intestine or introduced into the general circulation, as well as to autotoxins of intestinal origin, it may be assumed that it also acts as an emunctory in the case of autotoxins resulting from abnormal fermentative processes in natural and pathological cavities or in the glands and tissues, and of those which have their origin in an imperfect functional action of organs designed to destroy, by their biological activity, the toxic products of metabolism. Great obscurity reigns here, however, and it is only very recently that experiments have been undertaken with a view to the determination of this function of the liver. Very important researches, which begin to

throw a little light on this interesting question, have been made in the Pathological Institute of Naples by Gioffredi, d'Amore, and Falcone, and repeated more recently and confirmed by Boccardi. In dogs from which the thyroid gland had been extirpated, foci of true fatty degeneration were found scattered through the liver in those animals which survived for longer periods, while in those which died a short time after the operation the organ was markedly hyperæmic. It would appear, therefore, that the accumulation in the blood of the special toxic principle no longer destroyed by the thyroid gland caused an increased functional activity in the liver, manifested by hyperæmia; while subsequently the elimination of this toxic substance produced trophic and anatomical alterations in the organ.

But conclusions of far greater importance can be drawn from the researches of Semmola upon the pathogenesis of Bright's disease. Having produced a condition of heteroalbuminuria in dogs by the injection of egg albumin, he found true lesions of the hepatic tissue. In this case the liver exercised its protective function by eliminating albumin incapable of absorption and therefore injurious to the economy, and to this exaggerated and perverted function were due the pathological alterations found in this organ in advanced stages of the experimental albuminuria.

We see from what has preceded how closely the subject of autointoxication is bound up with that of the hepatic functions. A diminished functional activity of the liver gives rise to autointoxication, while, on the other hand, autointoxication is often the cause of functional and pathological changes in the liver.

Relation of the Liver to Infections.

We have already noted the conditions which favor the development of an infection of the liver when an infectious process already exists in the intestine; the sluggishness of the circulation and the fineness of the vascular network are the factors which favor the invasion of the parenchyma of the liver by the infectious agent.

Also in general infections these conditions favor the implication of the liver. Hepatic abscesses are not solely of portal origin, but may be produced also through the agency of the general circulation, when suppurative processes exist in other organs or tissues. It is not necessary for this that there should be a true septic condition of the blood, or that there must be capillary embolism, for the pyogenic microbes having once entered the general venous system may pass through the capillaries of the lungs without exciting any morbid process, and being thence transported by the hepatic artery to the liver, they are arrested there where the circulation is retarded. Their

presence there and the toxins produced by them excite lesions of the vascular endothelium, obliteration of the capillaries, emigration of leucocytes, suppuration, and miliary abscesses. We may thus find hepatic abscess as a complication of peripheral suppuration, especially in the bones. Miliary tuberculosis of the liver is produced by the same mechanism; the tubercle bacilli entering the general circulation are arrested in the fine network of the liver and there give rise to tuberculosis.

Metastasis in the liver occurs frequently in the case of malignant tumors. The latter may give rise to emboli which through the specific character of their elements will be the cause of a reproduction of the tumor. Among the tumors of this nature which are reproduced in the liver the most common are sarcomata, which as is well known are propagated along the blood-vessels.

Of greater importance are the lesions of the liver occurring in general infections of the blood, not as metastases, but as a specific reaction of the hepatic parenchyma to the infective agent. It is especially in recent times and by Italian pathologists that the relations between infectious processes and hepatic changes have been studied from the clinical and experimental point of view. We shall refer again to these studies when we come to treat of the special infections of the liver. Here we shall speak only of the general relations.

The interesting studies of Laveran, followed by those of Marchiafava, Celli, Golgi, Baccelli, Guarnieri, and others, have directed the attention of pathologists especially to the anatomical and functional changes which the pathogenic agent of malaria excites in the liver, and the name of malarial liver has been given to the complex of morbid conditions, the origin of which is the hæmatozoon of Laveran. No less intimate is the dependence of hepatic disease upon syphilis and typhoid infection. We need not discuss here the question whether the lesions found in the liver in these diseases are due to the specific agent and its products. It is certain, however, that lesions of the liver occur so frequently in typhoid processes that we have come to recognize a form of cirrhosis called post-typhoid. The liver is also attacked in other infectious diseases, especially smallpox, influenza, puerperal septicæmia, and yellow fever. Recently Siderey has made a very exhaustive study of the anatomical changes of the liver occurring in smallpox. There is first, he says, an intense congestion of the liver with migration of leucocytes and swelling of the endothelium of the capillaries, and later the hepatic cells participate in the infectious process, presenting lesions varying from simple swelling to complete fatty degeneration. The same author observed similar alterations in the liver in cases of cholera, in which,

however, he found also an extensive invasion of embryonal elements. Maragliano and others have noted complications on the part of the liver in influenza, consisting in an increase in size of the organ, accompanied sometimes by icterus. Rilliet found in puerperal septicæmia and in infectious enteritis nodules in the liver formed of embryonal elements and degenerated hepatic cells which accumulated within the lobules, and at the same time the portal spaces were invaded by a great number of embryonal cells. The changes of the liver in yellow fever are of great importance, for the specific virus is located here in great part and produces anatomical changes similar to those of acute yellow atrophy. Laure, who has studied the changes in the liver in various infections, found always that the hepatic cells became cloudy and infiltrated with fat granules, the capillaries of the lobule were dilated, and the perilobular connective tissue was hyperplastic, and encroached upon the lobule so as to interfere with the circulation and consequently the nutrition of the hepatic cells.

Relations of the Liver and the Blood.

When it was believed that the function of the liver was preëminently a hæmatopoietic one, it was thought that anatomical changes in this organ would have a most important effect upon the blood. There was believed to be an anæmia from insufficient blood formation in destructive lesions of the hepatic parenchyma. But it was never explained how, when in the course of various morbid affections a great part of the liver substance was degenerated or atrophied, there never was found a pernicious anæmia, the clinical picture of which would dominate that of the hepatic disorder; and, on the other hand, in grave forms of anæmia the cause was not found to lie in functional disturbances or pathological changes in the hepatic parenchyma, the affection being referred to other causes and being produced in other ways.

But gradually, as a result of the investigations of Pflugge, and of Foà and Salvioli, the theory of a hæmotopoietic function of the liver has been rejected, and now we do not regard the anæmic condition observed in the course of hepatic disorders as due to a disturbed blood-making function of the liver, but rather as the result of other factors. Digestive disturbances, insufficient glycogenesis and urogenesis, a lessened power of protection against autointoxication, all conditions necessarily produced in hepatic disorders, are factors which certainly ought to have their influence upon the nutrition of the tissues in general and therefore of the blood tissue, the anæmia of liver troubles being therefore an indirect result of the morbid process. This is shown also by many clinical facts.

We have already seen that to the liver is now attributed the function of destroying the old blood corpuscles, and we have classed that among the protective functions, since it frees the organism from elements which are no longer able to fulfil their biological function and have therefore become superfluous, if not harmful. This is the theory which is generally accepted, although it has not been absolutely demonstrated experimentally. Lehmann found a proportionally greater number of red blood corpuscles in the hepatic veins than in the portal vein and upon this based his theory of the hæmatopoietic function of the liver. But Pflugge has explained this by the fact that the blood passing through the hepatic capillaries loses a great quantity of water in the formation of bile, and that, therefore, the blood of the hepatic veins will naturally contain a higher percentage of red corpuscles. And this is the reason why we cannot demonstrate with certainty that a destruction of the used-up red cells takes place in the liver, for we are unable to say how much water is abstracted from the blood and therefore cannot determine with any approach to accuracy whether, and how many, red blood corpuscles are destroyed. This theory is based solely upon physiological deductions, strengthened by clinical observations. The analogy in chemical composition between hæmoglobin and the bile pigment is one of the chief arguments in favor of this theory, since it is easy to suppose that the blood pigment undergoes some slight modification under the influence of the hepatic cells and is thus transformed into bile pigment. And that the bile pigment may be derived from the blood pigment is all the more admissible since it has been maintained that the change may occur within the blood itself without intervention of the hepatic cells, the so-called hæmatogenous icterus arising in this way. But there is another important fact in the strict dependence of the quantity of bile pigment produced upon the number of blood cells destroyed. We know, indeed, that the red blood cells are destroyed by certain poisons (arsenic, phosphorus, pyrogallic acid, naphthol, etc.) and in such cases a great quantity of hæmoglobin in solution is carried to the liver; the secretion of bile is increased, and there is an augmented production of bilirubin.

Admitting therefore the destructive power of the liver upon the old red blood cells we may study from this point of view the changes which the blood undergoes in hepatic disorders. But these researches are neither easy nor certain, since our methods of study of the chemical and morphological constitution of the blood are far from exact, notwithstanding the multiplication of processes and apparatus. The blood changes in diseases of the liver cannot, for the reasons above given, be in regard to the quantity of hæmoglobin or to the

number of red or white cells, since their production is not interfered with in these diseases, but it is necessary to study other changes for the explanation of which, however, all experimental means have been wanting. We can thus understand how the most careful study of the blood in various destructive alterations of the liver has hitherto failed to demonstrate the presence of any morphological or chemical changes. Recently, however, the study of the blood from a new point of view has enabled us to observe some special alterations in the red corpuscles.

Catellino has studied the action of light upon the red globules and has noted that they become much less resistant, easily losing their hæmoglobin, after passing through the liver, in cases in which a large number of hepatic cells have been destroyed. This he believes to be due not to any change in their constitution, but to a deleterious influence exerted upon them by the blood plasma charged with products which the hepatic cells have not been able to transform into innocuous substances. There is no doubt that this hypothesis is in accord with the physiological conception now held of the hepatic function, but we do not think that we ought to ignore altogether the new conditions created in the blood as a result of the failure of the chief eliminating organ to destroy the used-up blood cells. We believe that in this case these cells accumulate in the blood, but are endowed with a diminished power of resistance to external influences, in accordance with their physiochemical constitution.

Having thus noted the changes in the blood resulting from disease of the liver it remains to study the effects which are produced in the liver by changes in the blood. We shall not speak here of the consequences resulting from an exaggerated hæmatolysis, whether of infectious or toxic cause, for that subject will occupy us when we come to treat of the pathogenesis of icterus; we shall direct attention only to the effects produced upon the liver and its functions when the blood undergoes changes in its normal constitution. In chlorosis, especially when it is somewhat marked, we often find fatty degeneration of the hepatic cells. In a case of chloranæmia, in which the red globules were reduced to 407,000 per cubic millimetre, Teissier found extensive fatty degeneration in the liver, involving nearly all the cells; the bile in the gall-bladder was colorless, of neutral reaction, and did not give the reaction of Gmelin or that of Pettenkofer, and the spectroscope showed only a faint line of urobilin. In leucocythæmia fatty degeneration and scattered lymphoid foci are frequently found in the liver. Worm-Müller and Winge found in one case a collection of adenoid tissue with an accumulation of embryonal cells. Anatomico-pathological alterations of the same nature are found in

leucocythæmia. All these changes are to be regarded as an effect of diminished metabolism and lowered cellular oxidation; the blood which is poor in red corpuscles (many of these being also deformed) and in hæmoglobin which is so necessary in the combustion of the tissues, is no longer capable of effecting the gaseous changes.

Hepatic lesions secondary to greater destruction of the red corpuscles are very common. Thus in progressive pernicious anæmia which is characterized by a great destruction of the red corpuscles Grohé found a grayish-black discoloration of the liver, caused by a deposit of sulphide of iron, and other more recent writers also speak of the large amount of iron in the liver; thus Quincke found 0.21 per cent. and Rosenstein 0.52 per cent., while in the normal liver only 0.12 per cent. of iron is found (Remak). These facts, now accepted by all, are strongly confirmatory of the theory that the liver is an organ essentially destructive to the red corpuscles. In this case, however, the numerical diminution of the red corpuscles in the blood is not to be attributed to the exaggeration in function of the liver, but rather to the abnormal morphological and chemical constitution of the globules themselves, they being thereby rendered unfit for their biological purposes.

In melanæmia, in which a great destruction of corpuscles occurs in consequence of malarial infection, a large quantity of the blood pigment is set at liberty; there is found in the liver, and especially in the intralobular vessels and in the central veins, a large amount of black pigment which sometimes also invades the cells of the parenchyma (Virchow). It appears from this that much material is carried to the liver for the formation of the bile, and when this is too great in amount for the functional activity of the liver it remains deposited there, it being impossible to transform it into bile pigment. Finally, we may recall the fact that in hæmoglobinuria, in proportion to the destruction of the red blood corpuscles, there occurs a greater afflux of material to the liver, which transforms the blood pigment in great part into urobilin; this, if the corpuscular destruction is slight, may be revealed solely by a true urobilinuria, while, if the process is very marked, untransformed hæmoglobin is eliminated by the kidneys.

This functional exaggeration is not, however, the cause, but rather the effect, of the grave alterations in the constitution of the blood, a fact which shows still more clearly how intimate are the functional relations between the liver and the blood, so that every dyscrasic alteration in the latter will be echoed in the function or nutrition of the hepatic gland. These relations are not thus limited, however, but exist also in regard to the chemical constitution of the two tissues, the blood and the liver. Salkowski made a chemical examination of

the liver in leucocythæmia and found in 2,500 gm. of liver substanc bodies resembling peptone, 0.0032; tyrosin, 1.718; leucin, 0.864; hy poxanthin, 0.2426; other xanthinic compounds, 0.538; succinic acid 0.0852; no uric acid. Bockedal and Landwehr found in 1,400 gm of liver substance much peptone, leucin, and tyrosin; 0.717 gm. o xanthin, 0.080 gm. of succinic acid, and 0.086 gm. of lactic acid; hy poxanthin and uric acid were absent. In the liver of an individua dead of progressive anæmia Lebert found much leucin and tyrosin.

But in addition to the morphological alterations in the constitu tion of the blood which produce histological, functional, and chemi cal alterations in the liver, the disturbances of metabolism whicl react upon the chemical constitution of the blood also induce no less important morbid changes in this organ. Beginning with the mos simple disease of metabolism, namely, polysarcia, characterized by the transformation into fat, and the deposit of the same in the tissues, of a large part of the products of intestinal absorption, there is a marked infiltration of fat in the liver which may even reach fatty de generation. As regards these anatomical changes in the liver in polysarcia we must not forget that some authors, especially Murchi son, have regarded the latter as a result of the former. As, indeed, we have already remarked in speaking of the physiology of the liver, this gland has a lipogenous function; and it is possible that an exag geration of this function might result in an accumulation in the bloo of fat which, not being wholly destroyed by organic combustion would be deposited in the tissues. Functional alterations in the live might thus be the cause of either obesity or emaciation, according t whether there is an increase or a decrease in its vital activity. W believe, however, even if the liver has some part in the pathogenesi of this disturbance of metabolism, that its part is a very small one for there would be no accumulation of fat in the organism, even wer it formed in excess in the liver, if the organism were able to consum it, that is to say, if the equilibrium were maintained between th formation and the destruction of fat.

No less important relations, which we think call for some discus sion, exist between uricæmia and functional changes in the liver. From the fact that uric acid is found in rather large proportions i the liver, Murchison first and, after him, Charcot attributed grea importance to the uricogenic function of the liver in the production o gout, which consists in an abnormal accumulation of uric acid in th blood (Garrod). Charcot was the most ardent advocate of this theory, which he supported by many clinical facts and pathogenetic induc tions but without bringing forward any experimental data which could throw clear light upon the question. He notes that many authors

have referred to a swelling of the liver as a prodromic symptom of a gouty paroxysm, and this swelling coincides with the increase of uric acid in the blood. Charcot believes the increased formation of uric acid and its accumulation to be the result of the functional disturbance in the liver, and the uric-acid saturation of the blood so caused provokes the paroxysm. In support of this view he recalls the fact that gout is very common in lead poisoning, and he regards it as a result of the hepatic lesions caused by the lead. The same author also reports some cases of hepatic disease in which there was uricæmia together with tophaceous deposits in the fingers and pinna of the ear.

Facts of this kind are certainly of the greatest importance, since they demonstrate the intimate functional relations between uricæmia and the liver, but they do not, in our opinion, prove that uricæmia is of hepatic origin, a theory which is supported by no clinical or experimental fact of certain interpretation. Indeed, upon analyzing the arguments of Charcot, they seem to point to the very opposite conclusions. The presence of uric acid in the liver in mammals is not an indication of the aim of its functional activity. The liver, which is the greatest laboratory in the body for the disassimilation of proteid substances, reduces them to their last degree of consumption, which is represented in man by urea, in birds by uric acid. But because the latter may be found in very small amount in the liver of man we cannot infer that therefore the formation of this substance is the aim of the hepatic function; we might as well say that one of the hepatic functions is the elaboration of hypoxanthin, leucin, etc., since these substances are found, even in the normal state, in the substance of this organ.

The symptoms on the part of the liver, prodromes of the gouty paroxysm, coincident with the accumulation of uric acid in the blood are due, we believe, to hyperæmia caused by overwork of the gland in its effort to transform the excess of uric acid into urea, rendering it soluble and capable of elimination. The question is not one of a pathogenic cause, but of a physiological compensation which is sometimes sufficient to free the blood from its load of uric acid. Neither the frequency of gout in lead poisoning nor the presence of uricæmia as symptomatic of various anatomical changes in the liver is a proof of the hepatic origin of uricæmia, except in the sense that anatomical changes in the liver may prevent it from converting into urea the oxidized products of the proteid substances.

The close relation existing between the glycogenic function of the liver and diabetes mellitus has directed the attention of clinicians and pathologists to this important factor in the etiology of diabetes. The various anatomical and functional disturbances in the liver may

give rise to glycosuria in two very different ways. Thus, when there is hyperæmia of the liver the increased production of glycogen induces a hyperglycæmia, and consequently glycosuria. On the other hand, in the case of lessened activity of the hepatic functions, or even when its glycogenic function is wholly abolished, the sugar which the portal vein contains in excess cannot be arrested and transformed into glycogen in the liver, and is therefore passed on into the general circulation and is excreted in the urine. It is interesting to note that the glycosuria caused by over-activity of the liver is continuous, since it is the result of a continous function of the gland, but that due to a failure in the sugar-destroying function of the liver is intermittent, since it appears only when there is an absorption of sugar from the intestine; the latter is an alimentary glycosuria, the former is not.

The glycosuria from increased functional activity of the liver may also arise in one of two ways: as a result of increased circulatory activity or by direct irritation of the hepatic cell. The first mode of origin may be illustrated experimentally by stimulating the vasodilators of the liver or by paralyzing the vasoconstrictors. A glycosuria from irritation of the hepatic cells may be excited experimentally by injecting sulphuric ether into a branch of the portal vein, or even by introducing this substance into the stomach. The appearance of sugar in the urine during asphyxia is to be attributed to the stimulation of the liver cells by the carbonic acid with which the blood is saturated; when the asphyxia is gradual, however, glycosuria is not produced, since the reserve supply of sugar in the portal blood is lessened.

The study of this glycosuria of toxic origin is important, although very difficult, for many factors enter into the mechanism of its production. Thus in the glycosuria resulting from the inhalation of chloroform, ether, or carbonic oxide, besides the direct irritation of the hepatic cells caused by these substances, we may recognize other not less important factors. It is known, indeed, that stimulation of the pulmonary branches of the pneumogastric induces glycosuria reflexly (C. Bernard); and certainly the poisons above mentioned, when inhaled, must irritate the terminations of these pulmonary branches of the vagus, and the glycosuria might be a result of this. These substances, furthermore, have a paralyzing action upon all the tissues of the body, a result of which would be that less glucose would be burned up in the organism, and the blood, surcharged with sugar, would dispose of some of it by way of the kidneys. Which of these factors—hepatic, pulmonary, or tissue—is most important or which of them acts chiefly, we are as yet unable to determine. On the other hand, it has been shown clearly that the glycosuria occurring in strychnine poisoning is the result of increased functional activity of

iver. The glycosuria following poisoning by curare is again obscure, some explaining it as a result of the asphyxia, while s have stated that it may occur even after extirpation of the liver. e case of phloridzin poisoning, von Mering's experiments have n that the glycosuria is produced as a result first of increased formation of glycogen into glucose, and later of a decomposi- of the albuminoids.

henever the blood charged with the products of intestinal absorp- s unable to yield up its contained sugar to the liver, either be- of nutritive changes in this organ (poisoning by mercury, ar- or phosphorus) or by reason of obstruction in the portal system, results glycosuria from hepatic insufficiency, or better the so- alimentary glycosuria. The existence of this form has been ately demonstrated both experimentally and clinically. C. rd produced it by gradual ligature of the portal vein, Pavy by ntation of the portal into the renal vein, and Colrat, Lépine, thers have observed it in the course of cirrhosis of the liver. We refer to this more at length in the section on the general symp- ology of diseases of the liver.

Relations of the Liver and the Kidneys.

the section on the physiology of the liver, we directed attention lly to the protective power of this organ, shown in its function emunctory or as a modifier of toxic substances coming from ut or elaborated within the organism. This is one of the most tant functions of the liver and one in which it is closely related kidney. No less important is the urogenic function of the liver ich it assists the renal function and at the same time, transform- to ureathe in completely oxidized nitrogenous compounds cir- ng in the blood, increases the urinary secretion, since urea is physiological diuretic, as Bouchard has shown. The func- relations between these two organs, which have very similar ological aims, are reciprocal and may even find expression in anatomical constitution, that is to say, functional alterations e of them may induce nutritive and anatomical changes in her. Thus the renal changes occurring in diabetes (which may hepatic origin) are well known, Ebstein having found a true ne- of the epithelium of the convoluted tubules, Ehrlich a glyco- infiltration of the epithelium of Henle's loops, and others a e degeneration of the epithelium of the straight tubules.

t that which more concerns us here, and which the practical cian ought never to forget at the bedside, is the mutual com- tion of the renal and the hepatic functions. Thus when in con-

sequence of disturbed innervation or, more frequently, of anatomical changes there is a diminished functional activity of the liver, the so-called hepatic insufficiency, the activity of the kidneys is increased the attempt being thereby made to eliminate from the organism whatever may have accumulated in the blood. The most striking and convincing evidence of this is shown in jaundice. Whatever may be the origin of this condition, it is in the kidney only that we find physiological compensation whereby injurious consequences are averted. The bile acids and pigments accumulating in the blood are eliminated by the kidneys, thus life being rendered possible, while a simple jaundice may become very grave if renal insufficiency be present. But this intimate relation is observed in many other conditions, and we may even say that the functional activity of the kidneys is increased in all cases of hepatic lesion. The reverse of this is also true, the liver compensating in a certain measure when the kidneys are insufficient to eliminate toxic products accumulating in the blood. A striking instance of this is the elimination of albumin (albuminocholia) in the bile in Bright's disease, a fact first demonstrated by Semmola. The albumin circulating in the blood, which has become incapable of assimilation and transformation into living tissue, is a toxic substance, injurious rather than simply useless to the animal economy, and must be eliminated by the kidneys and in small part also by the liver. The same thing is seen in the case of poisoning, and the bile constitutes, after the urine, the principal means of the elimination of toxic substances, especially certain mineral poisons.

But this functional dependence leads also to a pathological dependence, since, when the altered liver is no longer able to eliminate, arrest, or transform into innocuous substances all the toxic products arising from intestinal absorption, the latter are eliminated by the kidneys and in passing through them may induce irritative conditions in the epithelium of the glomeruli of the convoluted tubes, giving rise to nutritive disturbances and subsequent degeneration. The patient's condition is then greatly aggravated, for to the hepatic insufficiency is added renal insufficiency, and the autointoxication, increasing constantly in degree, finally puts an end to life. On the other hand, as long as the functional integrity of the kidneys is preserved, very grave lesions of the liver may not be incompatible with life, since the toxic products accumulating in the blood may be eliminated in the urine. This offers an explanation of how, when in cases of grave infection the liver and kidneys are affected at the same time, a very serious condition may arise and lead rapidly to the death of the patient. In acute yellow atrophy in which together with the grave lesions of the liver there exist equally grave changes in the kidneys,

re is produced a threatening and even frightful morbid picture, in great part to autointoxication; and it is probable that the rse of this affection would be much less serious were it not for the stence of these grave renal lesions.

From these considerations we may conclude that it is always essary to look to the renal functions in cases of disease of the liver, e it is on them that we have to rely for a physiological compen- on which may temporarily make up for the suppressed functional vity of the liver, and give time for a cure of the morbid process his organ.

Relations of the Liver and the Heart.

The relation between the heart and the liver is not very well un- stood, although the coexistence of affections of the two organs noted by Rega, Baglivi, Sauvage, Baillé, Abercrombie, and rs, who reported cases in which hepatic disease followed func- al disorders of the heart. Stokes[17] was, however, the first who e a serious study of cardiac complications arising in the course ertain hepatic disorders. He analyzed a number of instances of coexistence of the two conditions, and came to the conclusion that as only through the nervous system that the complication could rought about. Garrod, studying the disturbances of cardiovas- r function occurring in the course of gout, regarded them as de- lent upon the dyspepsia and the disorders of the liver which are ommon in this condition. But the most accurate and detailed y of the functional and anatomical changes occurring in the heart ecutive to diseases of the liver was made by Murchison, although Giovanni had before that time noted the occurrence of cardiac nomena of gastric origin, and had offered a rational explanation of ame. Murchison studied, in his immortal work on hepatic dis- s, the physio-pathological relations between the heart and the , and mentioned all the functional changes in the heart which may bserved when the biological activity of the liver is more or less al- l. He noted the occasional occurrence in the course of hepatic ses of palpitation, arrhythmia, and of more or less marked and ng intermittence induced by slight causes or even coming on taneously; and he also described a true angina pectoris the ori- f which could be explained only by a preëxisting hepatic lesion. same author attributed to the liver various functional and ana- al alterations of the respiratory organs, manifested by asthmatic ks or by bronchitis. He explained these cardiopulmonary ges by assuming that the hepatic lesions had led to an accumu- n of toxic products in the blood, which, according to their nature,

might irritate or induce a paretic condition in the pneumogastric nerves.

But in place of this interpretation, which is a purely hypothetical one sustained only by general considerations regarding the protective function of the liver, another has been put forward. This latter may indeed serve to explain certain clinical facts, but it cannot be invoked as a sufficient and the only reason for all the complex and varied disturbances of function of the cardiovascular system which may arise in the course of hepatic diseases. Rendu,[18] Strauss,[19] Pitres,[20] Mossé,[21] Teissier,[22] and Barié[23] have published a vast number of cases in which the heart lesions were confined almost exclusively to the right side, and in which the etiological factors were found not only in hepatic but also in gastric, intestinal, and uterine diseases. According to the French school, the pathogenesis of these cardiac changes consists in reflex irritation passing from the bile passages to the vasomotor centre, and thence to the pulmonary vessels, inducing contraction in the latter. In this way an increase of pressure occurs in the pulmonary artery, in consequence of which the right ventricle becomes dilated and hypertrophied, and if the dilatation is very marked the final result may be dilatation of the right auriculoventricular opening with consequent insufficiency of the tricuspid valve. When, therefore, we find cases of dilatation and hypertrophy of the right ventricle, with or without insufficiency of the tricuspid, we may think of this explanation of the condition. But the cardiac disturbances consecutive to hepatic lesions are not limited wholly to the right heart, as has been maintained by the French school. Thus Gangolphi[24] has reported nine cases of simple jaundice in which there was a murmur with the first sound over the mitral area, accompanied by all the signs of dilatation of the left ventricle, which the author explains by a functional paresis of the myocardium caused by the presence of bile in the circulation; this paresis leads to a dilatation of the ventricle with weakness of the papillary muscles and thus causes an insufficiency of the mitral valve. Fabre[25] has also described five exactly similar cases in which there was no ground for attributing the functional disturbances to changes in the right heart.

But in addition to these cases showing involvement of the left heart, there are many other instances reported in which there could be no question of an anatomical lesion of either side, but purely of functional disturbances, such as palpitation, intermittency, arrhythmia, etc., leading finally to true cardiac insufficiency. It is evident that such cases must have a different explanation from that given above, as Patella[26] also has insisted upon in his criticism of the theories of the French writers. Furthermore, that this explanation cannot hold

ll cases of hepatic disease accompanied by jaundice is rendered
probable by the researches of Leyden, who has shown the injuri-
effect upon the heart muscle of the bile acids and pigments when
oduced into the circulation; under their influence the myocardium
become degenerated and thus easily dilatable and incapable of
racting with normal force.

Then, again, in cases of hepatic disease in which there is no icterus,
cardiac disturbances may be explained in another way from that
ne French school. In these cases we may meet with various dis-
ances of cardiovascular function unaccompanied by any demon-
ble anatomical lesions of the cavities or valves, and therefore not
e explained by an assumed dilatation of the right ventricle or by
fficiency of the tricuspid valve. There is here a simple functional
rbance due to altered innervation, or perhaps a functional lesion
ne cardiac muscular fibre, of which the exciting cause is the pres-
in the blood of poisons coming from the intestine or resulting
metabolic processes in the tissues, and which the liver in its
ed state is incapable of arresting and destroying.

Finally, some cases can be sufficiently explained in a purely
hanical way. In some cases of hypertrophic cirrhosis, for exam-
the liver is greatly increased in size so as to press upon the
t and displace it so that the apex beat may even be found in the
l intercostal space in the anterior axillary line. The organ then
almost horizontally upon the diaphragm, and its weakened action,
mittent and irregular pulsation, and the malleolar œdema are all
y explained by this fact.

Thus we believe that there is no one etiological factor active in
ases, but that autointoxication from hepatic insufficiency, reflex
m of the pulmonary capillaries leading to dilatation of the right
t, and displacement of the organ by an enlarged liver may each
all in individual cases cause the cardiovascular disturbances seen
ne course of various diseases of the liver. The explanation
ed by Potain is admissible only when we are able to make out
presence of actual lesions of the right heart, and cannot be in-
d indiscriminately in all cases in which affections of these two
ns coexist.

We have already, in speaking of the hepatic circulation, referred
e changes in the liver consecutive to cardiac disease, and we shall
n to the subject when we come to the section devoted to passive
estion of the liver and the lesions dependent upon it which have
ved the name cardiac liver.

Relations of the Liver and the Nervous System.

We often see clinically patients suffering from hepatic diseases of various kinds who present nervous symptoms of varying kind and degree, from simple neuralgia to true convulsions, from a slight vertigo to more or less pronounced paresis. We cannot regard the occurrence of these two forms of disease as purely coincidental, for an accurate study of these patients will reveal a well-marked pathogenic dependence, or, better, an actual relation of cause and effect. We shall not describe here all the nervous symptoms which may be present in cases of hepatic disorder, as we shall have to return to the subject in the section on general symptomatology; but we may say only that these nervous symptoms, which are of the most varied sort, may be observed in connection with a great number of hepatic affections, always those which are associated with marked disturbance of function of the organ. They are most important in their gravity and deserving of the closest attention on the part of the physician when they occur in hepatic affections accompanied with jaundice, and in those in which there is a more or less marked destruction of the parenchyma resulting in hepatic insufficiency.

The entrance of bile into the blood. constituting jaundice, is a very frequent cause of these nervous symptoms, which are, as may readily be supposed, produced by the toxic action of the bile acids and pigments upon the nervous system. This is proved by experiments as well as by clinical induction. But leaving aside cholæmia as a cause of nervous disturbances, there is another much more important factor in hepatic insufficiency resulting from the more or less extensive destruction of the liver substance. Murchison laid very great stress upon the presence of uric acid in the blood as a cause of the nervous symptoms. We have already seen what value there is in this theory of the relation of uricæmia to the functions of the liver, and we need not enter upon the subject again; we would remark only that Murchison, in his recital of the nervous symptoms dependent upon hepatic disease, has given a very complete catalogue of the nervous symptoms of gout, since in his belief the latter disease finds its most important pathogenic factor in disturbance of function of the liver. But uricæmia, in fact, cannot explain the pathogenesis of the nervous symptoms observed in the course of hepatic diseases, for recent researches have shown that uric acid in the blood has but little toxic property, since individuals are encountered who excrete large amounts of uric acid and urates in the urine and who yet present no nervous symptoms.

The real cause of the nervous disturbances accompanying diseases

he liver is hepatic insufficiency and the autointoxication consecu- to it. The anatomical changes in the liver render this organ in- ble of transforming and eliminating the toxic products of metab- n and of intestinal digestion, and these accumulating in the d act injuriously upon the nervous system and give rise to the ptoms now under consideration. We must not forget, however, the occurrence and the intensity of these nervous symptoms de- l in considerable measure upon the condition of the kidneys. If natomical and functional integrity of these organs is preserved, may be able to eliminate the toxic substances which the liver allowed to enter the general circulation, and thus avert the ex- ion of nervous symptoms; but if the kidneys are affected at the e time as the liver, the nervous symptoms will be aggravated. s in acute yellow atrophy of the liver these symptoms are very nounced, for the lesions of the kidneys in this disease are at least s great importance as those of the liver itself, and autointoxica- occurs early, since no efficient means of physiological compensa- remains.

There is, however, a great lacuna in the study of the pathogenesis erve intoxication in cases of hepatic disease, for we are as yet rant of the nature, chemical composition, and biological action of oxic agent.

We as yet know little also of the effect upon the hepatic func- s of lesions of the nervous system. Many clinical facts prove out question that there may be a marked reduction, or even in e cases an actual abolition, of hepatic function as a result of pro- d mental exhaustion. The sudden occurrence of jaundice, more ss marked in degree, as a result of a strong mental shock is a which has long been recognized clinically, and which may be ied almost daily. An explanation of the way in which this oc- has recently been offered by Oddi,[77] who has demonstrated the ence in the ductus choledochus of a sphincter formed of smooth s having a special innervation and a special spinal reflex exist- t the level of origin of the first pair of lumbar roots. We have a number of cases of this kind, some of them most striking; in recent instance there was a profound icterus following a mental k, and when this yielded to the therapeutic measures adopted was a very marked emaciation accompanied by pronounced ilinuria.

The influence of the nervous system upon the liver has been nstrated also by certain experiments. Claude Bernard noted ctive hyperæmia of the liver following puncture of the fourth icle. Kaufmann, in his study of glycosuria of nervous origin

due to lesions of the fourth ventricle, has shown that it arises through the intermediary of the liver in which there occurs a greater production of glucose. In experiments made by us in 1861 [*] we demonstrated the influence of electrization of the pneumogastric in glycosuria of hepatic origin, obtaining by this means a diminution in the quantity of sugar excreted in the urine.

Finally, although this has little to do with the question we are now considering, we may recall the fact observed long since that abscesses of the liver frequently follow abscesses of the brain. This was formerly explained by the assumption of a sympathy between the brain and the liver, but now it is recognized as due to a retrograde embolism occurring through the inferior vena cava and the hepatic veins.

Acholia and Cholæmia.

It has long been a disputed point, and is not yet wholly decided, whether in the causation of the symptoms of intoxication occurring in hepatic insufficiency the most important factor is the entrance into the blood of bile, or whether it is the failure of the liver to separate from the blood the substances which go to make up the bile. The first of these is the opinion which has for a long time prevailed. Starting with the belief that the bile is an excrementitious product, many pathologists have assumed that its entrance into the circulation and into the tissues of the body must necessarily result in an intoxication to which they have given the name of cholæmia. It is in this way that they have explained the symptom complex of pernicious jaundice of whatever pathogenic mechanism. In order to determine this fact experiments were made upon dogs, and death was seen to follow injection of bile into the subcutaneous connective tissue. But these experiments were shown by Frerichs and others to be faulty, for even large amounts of bile, freed from mucus, might be injected into an animal's veins without the production of any severe toxic symptoms, provided always that there were no coexisting lesions of the kidneys. But what proves more than any experiment that cholæmia cannot be invoked as the only cause of autointoxication in cases of hepatic lesions, is the daily observation of patients with pronounced jaundice, in whom the blood and tissues are saturated with bile, who present no marked signs of poisoning. Furthermore, we see that the degree of autointoxication bears no relation to the intensity of the cholæmia. And finally, we may see the occurrence of toxic symptoms when there has been no entrance of bile into the blood, as, for example, in advanced stages of cirrhosis of the liver in which there has been very extensive destruction of the liver sub-

ıce. However, it would not be correct to argue from these con-
rations that the interpretation of certain nervous phenomena by
læmia is altogether erroneous. One of us (Gioffredi) has studied
biological action of the bile acids and has shown that when they
injected in rather large quantity into animals, especially rabbits,
may cause the appearance of rather grave nervous phenomena
er of excitement or of depression. Austin Flint, Jr., has asserted
the cerebral symptoms consecutive to jaundice in organic affec-
s of the liver are due to the retention of cholesterin in the blood
ıolesterinæmia. This author regards cholesterin as the product
ıutritive changes in nerve tissue, and believes that it is a func-
of the liver to eliminate it from the organism, throwing it out
the intestine, where it is converted into stercorin, under which
ı it is found in the fæces. If, however, it is retained in the blood
tissues it will act as a poison, and its origin will explain its spe-
effect upon the nervous tissues. These views of Flint have been
ırmed by Picot and by the researches made by Koloman Müller
erning the toxic properties of cholesterin. It may be permitted
emark, however, that, if the absorption of all the elements to-
er of the bile cannot excite the grave symptoms of autointoxica-
encountered in hepatic disorders, it is difficult to understand
these symptoms can be caused by cholesterin alone. In cases of
complete obstruction of the ductus choledochus, in which it is
ssible for the cholesterin to reach the intestine, we do not invari-
find phenomena of poisoning.

'rom the considerations just mentioned, therefore, it follows that
emia cannot be taken as a sufficient explanation of all cases of
ntoxication in diseases of the liver. Undoubtedly the presence
le in the blood is injurious, but we cannot attribute to it all the
symptoms observed in the course of hepatic disorders. Traube
ed the theory of cerebral anæmia as a substitute for that of cho-
a. In grave disorders of the liver, whether accompanied by
lice or not, there is always a profound disturbance of general
tion, that is to say, there is a condition of more or less marked
exia and anæmia; in this disturbance the cerebral circulation
part and suffers either from the diminished tension in the
ies or from the altered constitution of the blood; and hence
symptoms of nervous irritation or depression which are really
xpression of cerebral anæmia. Cohnheim has also accepted this
y, although in a more limited sense. It is clear, however, that
heory cannot be accepted in every case as explaining the occur-
of nervous phenomena in the course of diseases of the liver.
ıight be justified in accepting it in those cases in which there is

very marked cachexia, but it is altogether insufficient to explain the occurrence of cerebral symptoms in those cases in which there are no profound nutritional changes.

The theory which was first established by Frerichs and which has since that time gained ever wider acceptance, in proportion as the physiology of the liver has come to be better understood, is that of acholia or the suppression of the eliminating function of the liver. According to this view, the nervous phenomena are to be regarded as symptoms of poisoning by the products of metabolism or of digestion which are no longer eliminated by the anatomically altered liver. Neither Frerichs nor others who accept this theory of acholia regard as solely of importance the retention in the blood of substances which ought to have gone to make up the bile, for they believe, and in this we are wholly in accord with them, that we should take into consideration also the fact that there occurs from the same cause (hepatic insufficiency) an incomplete disintegration of the proteid matters, an incomplete transformation of them into soluble products (urea) which can be eliminated by the kidneys. The hepatic cells, more or less altered, are unable to carry on the process of disintegration of the albuminoids to its final stage, the production of urea, but give origin instead to more complex and less oxidizable products of decomposition, such as leucin ($C_6H_{13}NO_2$) and tyrosin ($C_9H_{11}NO_3$). These substances are found in acute yellow atrophy, an affection in which, as is well known, there is a rapid destruction of the secretory elements of the liver; they are met with in the parenchyma of the liver as well as in the urine under the form of very characteristic crystals, while at the same time the urea becomes greatly diminished in amount or even entirely disappears. It is quite true that these bodies may also be found in the urine in other diseases in which there is not such a profound destruction of the parenchyma of the liver, such as cirrhosis and certain cases of obstruction of the ductus choledochus, and also in certain infectious general diseases, such as typhoid fever. But even in the latter instance the presence of leucin and tyrosin is to be attributed to functional insufficiency of the liver, which is no longer able, whether in consequence of the increased labor thrown upon it in the elimination of the toxins of the disease or of the toxic products more abundantly formed in the intestine, or because of actual lesions of its parenchyma caused by these toxins or by the typhoid bacillus, to transform the proteid bodies into urea.

The theory of acholia has been demonstrated also experimentally. Thus Gioffredi has observed that frogs sometimes die, after extirpation of the liver, with the classical nervous symptoms, such as paresis

e voluntary muscles, increased reflexes, exaggerated spinal irri-
ty, and even true tetanic convulsions, observed in advanced
s of cirrhosis of the liver and other grave destructive affections
is organ, and which are the forerunners of death. Sterne and
owski found that birds, after extirpation of the liver, died with
mptoms of vomiting, irregular respiration, and collapse, some-
preceded by convulsions. Recently Hahn, Massen, Nencki,
Pavlov experimented by making an Eck fistula on dogs. The
ls, sixty in number, were seized with nervous phenomena,
and tonic convulsions, somnolence, ataxia, and sensory disturb-
; these symptoms, which were followed by death, were aggra-
when meat was given to the animals to eat. The authors believe
hese symptoms were due to poisoning by carbonic acid, which
rmal liver is able to transform into some innocuous body—a
r proof of the protective action of the liver.

e may now conclude that, although the presence of bile in the
or cerebral anæmia may in certain cases explain the occurrence
mptoms of poisoning, especially of poisoning of the nervous
n, nevertheless the most important factor is the presence in the
and tissues of certain substances which normally are used in
boration of the bile, and of others which the liver has become
ble of transforming into urea. We have much yet to learn in
udy of the nervous symptoms associated with destructive le-
of the liver, and especially are we ignorant of the nature of
toxic principles, what is their physiological action, and whether
re the same in all individuals or whether they are of inconstant
sition.

GENERAL SYMPTOMATOLOGY.

e study of the general symptomatology of the liver is of great
tance, since the comparison of all the objective and functional
s in the organ offers a secure guide in diagnosis which is so
lt and obscure in many hepatic disorders. Furthermore, an
te description of all the symptoms of whatever nature which
es of the liver may present will save much repetition when we
to describe the special diseases, since many of them have a
r of symptoms in common.

e general symptomatology of hepatic disorders is based abso-
upon the general pathology of this organ. Thus when we
what are the disorders to which the liver may be subject and
chanism by which they are produced, it will be comparatively
o determine and to describe the symptoms which hepatic

diseases may present. But if we recall what has been said above concerning the physiology and pathology of the liver, it will be evident what a variety and complexity of symptoms we may encounter in hepatic disorders. These symptoms may concern almost all the organs in the body in addition to the liver itself. Indeed, the semeiology of the liver is one of the most varied, since it is not limited to the organic and functional changes in the liver itself but involves those of almost the entire animal organism, and may present such a varied and complex picture as to render most difficult a diagnosis of hepatic disease.

On the other hand, it is well to remember that in rare cases we may have an hepatic disease which will begin and even reach quite an advanced stage without giving any symptom whatever, or at most some ill-defined symptoms which do not attract the attention of either physician or patient. This is due to the fact that an affection of the liver, however fatal it may be in its final evolution, may exist with quite extensive lesions, which will give no sign of their presence until they have invaded vital parts of the organ and have thus caused disturbances in the circulation or in the glycogenic, protective, or other functions of the gland. This is of great importance clinically, for we not infrequently see patients who themselves are unaware of any liver trouble and in whom we can discover no signs of such by the most minute examination, who later, for some generally undiscoverable reason, suddenly betray the symptoms of an hepatic disorder which has perhaps already reached such an advanced stage as to be beyond the power of our therapeutic measures to relieve. In such patients the liver, already the seat of more or less extensive lesions, is yet able to perform its functions satisfactorily through extra labor thrown upon the healthy portions; there are then no important symptoms which point to the existing trouble, but when the sound portion is so reduced in extent as to be no longer able to perform the work of the whole organ, symptoms of well-established disease suddenly appear. In some cases also the kidneys may be able to discharge the most necessary functions of the liver, and then no signs of disease of the latter organ may be evident until the kidneys themselves are affected and renal insufficiency becomes established.

The various symptoms of hepatic disease may be divided into the two main classes of objective and functional, which themselves present a great variety of subdivisions. The objective symptoms may be divided, according to the manner in which they may be detected, into those discoverable on inspection, on palpation, on percussion, and on auscultation. The functional symptoms are divisible into

latory, digestive, urinary, toxic, hæmatic, and nutritive. We speak in this section also of icterus, which furnishes us with a important and complex clinical picture.

Objective Symptoms.

hese signs are of great importance, since they furnish us with t information concerning many of the anatomico-pathological ations in the organ under examination, while the subjective signs us only indirect information of the existence of an hepatic n. In the following we shall precede the description of the signs nable in disease by a brief account of what is to be noted in al states of the liver.

Inspection.

n the normal condition inspection of the right hypochondrium ls nothing worthy of note; we see complete symmetry of the hypochondriac regions, a depression in the epigastric region the umbilicus, and an even and uniform rising and widening of parts in inspiration. In young children, however, the liver is ortionately large, and we may see in them a prominence in the hypochondrium due to its presence there.

spection offers valuable evidence, however, in many diseases of ver. First, we have to note the condition of the superficial ab- nal veins, which in health are barely visible, but in cases of he- disease may become dilated and prominent, in some instances ring as serpiginous purplish cords, sometimes varicose and in- ed in such a way as to form a wide-meshed network to which ame caput medusæ has been given. These veins may reach a eter of a centimetre, at other times they are seen as little pyram- tumors round the umbilicus, or, when varicose, have the ap- nce of strings of beads. On palpation of these veins, especially they are of large size, we may obtain a quite distinct fremitus, on auscultation we can hear a soft, almost continuous venous

he significance of this venous network in the abdominal wall is he blood can no longer pass through its normal channels and is ted into collateral branches. We have already referred to the omoses of the portal vein with the other venous systems, the sory portal veins. Whenever any obstacle to the flow of the l blood occurs relief is obtained through these accessory veins, he so-called internal collateral circulation is established. But is aided by another, which we may call the external collateral ation, formed by the fifth group of veins of Sappey—the para-

umbilical veins—composed of several small veins, which often surround the obliterated umbilical vein in the shape of a plexus, forming a communication between the portal and the superficial abdominal and superior epigastric veins, through which in mammals the blood is carried into the innominate and thence into the superior vena cava. Several cases of persistence of the umbilical vein have been described; this would present an easier way for the establishment of the collateral circulation. Among the most common of the causes of obstruction of the portal circulation which would be indicated by the caput medusæ is cirrhosis.

This dilatation of the superficial abdominal veins may, however, be due to the presence of an obstacle in the inferior vena cava. In order to determine to what cause the dilatation is due, Cantani and Roncati have shown that we must note the manner of distribution of the venous stasis on the abdominal wall. If there is simultaneous dilatation of the branches of both the superior and the inferior epigastric veins we must assume that the obstacle exists in the portal circulation; while if the dilatation begins first in the field of the inferior epigastric and invades later that of the superior, we may infer that the circulation in the ascending vena cava is impeded.

But returning to inspection of the right hypochondriac region, we note in the first place that quite often a localized œdema in this region is an indication of suppurative hepatitis. But a sign that exists in many diseases of the liver in which hypertrophy is a feature is a diffuse elevation of the hepatic region; whether the patient be lying down or erect, the right side of the base of the thorax is seen to be more prominent than the left, the costal arch is increased, and in some extreme cases the lower ribs may be so thrown out that they become twisted, the lower margins pointing anteriorly and the posterior face looking downwards. There may, however, be at the same time a bulging of the left hypochondriac region due to enlargement of the spleen which frequently exists with hepatic disorder, especially in malaria. The deformity of the ribs may be less marked when the bones have become less elastic, as in old age, and more marked in children in whom the bones are especially elastic. In the former case, especially when the liver is somewhat depressed, the swelling is principally below the line of the ribs. An important point in differential diagnosis between hepatic disease and pleurisy is that in the elevation of the ribs due to an enlarged liver there is no bulging of the intercostal spaces, whereas if the swelling be due to pleuritic effusion the intercostal spaces will be more prominent than the ribs. The latter condition has, however, been observed in certain cases of abscess of the liver.

In addition to the diffuse tumefaction of the right hypochondriac ion there may be circumscribed swellings of the liver in various nts. These are due to lesions which do not involve the entire enchyma of the organ, the most common of which are echinococc- cysts and abscesses. An isolated swelling of hemispherical form upying the middle of the epigastric region may be due to dilata- of the gall-bladder from any of several causes which we need not to enumerate here.

Sometimes the lower margin of the liver may be made out by pection, when it will be possible to note the displacement of the an during inspiration. This may be due to unusual thinness of abdominal walls, to an enlargement and downward displacement he liver, and finally it may be seen in the absence of every ab- nal factor which might produce a distention of the abdominal etes. We have seen a very marked instance in a case of cirrhosis. Finally, inspection of the hepatic region enables us occasionally to the presence of pulsations in the liver. This hepatic pulse may autochthonous or transmitted. In aneurysmal dilatations of the a accompanied by passive hyperæmia of the liver the latter organ show very evident pulsations in a direction from behind for- ls, but which do not possess the character of expansion. A simi- orm of pulsation may be observed when an anatomical or func- al insufficiency of the tricuspid valve leads to an ectasis of the cava and produces a marked regurgitation into this vessel during ole. We have observed a case of this kind in which at the au- y there was a secondary insufficiency of the tricuspid valve follow- mitral stenosis and insufficiency; there was an advanced stage rdiac cirrhosis in the liver, and the ascending vena cava and right ricle of the heart were enormously dilated. The autochthonous e of the liver may be arterial or venous. In connection with narked arterial symptoms which aortic insufficiency may induce ugh the exaggerated tension caused by hypertrophy of the left ricle, there may, in rare caes, be a systolic pulsation of the liver; is to be attributed to an extraordinary fulness and strong pulsa- of the hepatic artery and of its ramifications in the parenchyma e liver.

venous pulse is found in tricuspid insufficiency and is produced, the jugular pulse, by the backward wave of blood sent out by the raction of the right ventricle; this, upon arriving at the liver, es an increase in its volume synchronous with the cardiac systole, iver acting in a certain measure like a vascular tumor. The tic pulse follows that of the jugular, but Friedreich has shown it may also precede it, and then its tracing will show an ana-

crotic curve. Dagnini[29] has studied the presystolic hepatic pulse in certain subjects of heart disease; this is produced by a rapid regurgitation of blood from the right auricle during the auricular systole and occurs when the auricle is dilated, when its walls are hypertrophied, and when its communication with the vena cava is very wide and incapable of closing, while at the same time there is a permanent obstacle to the passage of blood from the right auricle to the corresponding ventricle; or there may be disorders of compensation preceded by a lesion of the auriculoventricular orifice.

Palpation.

Among all the means of investigation at our disposal in diseases of the liver palpation deserves perhaps the first place, for it not only gives us exact notions of the enlargements of the organ, but it also enables one with the *tactus eruditus* to judge with precision of the form, consistence, condition of the surface, etc.

The liver being almost completely covered by the ribs on the right side, it is hardly in normal conditions accessible to palpation except in the epigastrium, where the arching up of the ribs leaves part of the right lobe and the left lobe of the liver covered only by soft parts.

In order to practise palpation of the liver, the patient should be lying in the supine position, and all tension of the abdominal muscles, whether voluntary or reflex, should be overcome. This may sometimes be accomplished by distracting the attention of the patient, conversing with him concerning the beginning of his disease, the degree of his sufferings, his general health, etc. He should be made to take a deep inspiration when the most favorable moment for making the examination will arrive, for then the liver is pushed down by the descent of the diaphragm and at the same time the abdominal muscles are relaxed. In order to prevent reflex muscular contraction the palpation should be made with a warm hand, and we should begin to palpate by making light pressure with the hand turned so that the radial border is directed upwards, the ulnar border downwards in correspondence with the costal arch, the pressure being gradually increased so as to get the margin of the liver in the hollow of the hand. If the abdomen is distended with liquid or gas the patient must be turned on his left side so as to let the fluid flow away from the liver. The palpation should be made from right to left; beginning ordinarily at the lower edge of the tenth rib, we pass over to the left hypochondrium and then go from the costal arch downwards and backwards. When we are unable to palpate the margin of the liver in this way, as may happen, especially when there is much meteorism

scites or when the muscular tension is marked, we may begin the ation below, in the para-umbilical line, passing slowly upwards rds the costal arch.

ut in cases in which the abdomen is greatly distended by gas or , or in which there is marked rigidity of the abdominal muscles, r because of pain or in consequence of exaggerated reflex irrita-, we are sometimes unable to palpate the liver, even when it is ged. In these cases we may often succeed by means of the so-l tapping palpation, which consists in palpating rapidly with ips of the fingers, seeking to distract the patient's attention and ach the liver at a moment when the abdominal muscles hap-to have become relaxed. The patient should lie on his back, tly inclined to the left side. This method may, however, be ex-ly painful in certain cases, and in others it may be dangerous, at it is not admissible in all cases indiscriminately. Thus it be very painful in cases of perihepatitis and of great distention isson's capsule, and dangerous in cases of abscess or of hydatid lying superficially and having thin walls. In these cases we resort to what is known as combined palpation. With the left open and applied against the right lumbar region pressure is upwards in the attempt to raise the liver and bring it closer to bdominal wall, while the right hand makes gentle but continuous ure with its ulnar border and gradually works down so as to the border of the enlarged liver.

énard, in studying the alterations of the liver symptomatic of n cases of diabetes, devised a special method of examination ich he gave the name of the "thumb method." It consists in ng pressure in the right lumbar region with the four fingers left hand, while the right hand makes pressure from below up-in the right groin, the endeavor being made to feel the border liver by the thumbs of the two hands; to facilitate this the t is instructed to take deep inspirations in order to depress the as far as possible. We have tried this method in many cases ave found that, while it is often useful, it is not superior to the ned method above described.

we have said above, in the normal state the liver is inacces-o palpation, since it is hidden behind the ribs, but there are ceptions to this rule. These exist in the case of children in the liver is very large in respect to the capacity of the abdo-and in that of women who are impelled by vanity to reduce the the waist by tight lacing.

lpation is one of the most important means of diagnosis of the roubles that we have at our disposal. In the first place it en-

ables us to judge of the degree of painfulness of the organ; and the existence of this pain often furnishes valuable evidence of enlargement of the organ, for it may sometimes be elicited by pressure with the index finger along the lower margin of the liver in cases in which this point could not be made out by percussion because of extreme tympanites, or by palpation because of tension of the abdominal walls. Pain, either spontaneous or provoked, of the liver is always due to more or less marked distention of the peritoneal investment of the organ, or rather to an inflammatory process going on in this capsule. We must always remember, however, that distention of the capsule causes pain when it occurs rapidly, but when the liver has increased in size gradually the capsule is stretched slowly and no pain is provoked. Thus we find that pain is a marked symptom in active or passive hyperæmia of the liver, in cases of hydatid cysts of rapid growth, of abscesses of the liver, etc. But, on the other hand, in cases of amyloid or fatty liver, in hypertrophic cirrhosis, and even in hydatid cysts, when their growth is slow, we find that pain is usually absent. The pain is, however, often very great when the capsule of the liver is involved in the morbid process, as in perihepatitis, syphilis, cancer of the liver, or the like. In these cases the pain in the hepatic region is often one of the most trying of the patient's symptoms. Finally, pain may be due to irritation in the gall-bladder or bile ducts, as in cases of gall-stone in the cystic duct or ductus choledochus.

A very important point which is determined by means of palpation is as regards the size of the liver. The organ increases in size as a result of various causes, such as circulatory disturbances (active or passive hyperæmia), an obstruction to the flow of bile (gall-stones, biliary cirrhosis) inflammatory processes (interstitial hepatitis, hypertrophic cirrhosis, syphilitic disease, suppurative hepatitis and consecutive abscess), neoplasms (carcinoma, gumma, echinococcus, adenosarcoma), or degenerative processes (fatty or amyloid). The enlargement of the organ varies in different cases, so that the lower margin may be but a few fingers' breadth below the border of the ribs or may extend into the hypogastric region, and on the left side it may invade the hypochondrium or pass over so as to fill completely the left flank. The greatest increase in size occurs when the organ is the seat of circulatory disturbances, of degenerative changes, or of neoplasms. Thus in hyperæmia from stasis, in amyloid degeneration, in cancer, in hydatid cysts, the organ may reach such a volume as to occupy the epigastric, mesogastric, and part of the hypogastric regions.

Of exceeding interest is the study of the surface of the liver, its

and consistence, and the modifications which the surface of the n may undergo are also very important in a diagnostic sense. s it appears granular in cases of ordinary cirrhosis, studded with protuberances having an umbilicated centre in cases of multiple noma, lobular in gumma of the liver, and largely projecting in of abscess or hydatid cyst.

he shape of the organ may be preserved in cases of enlargement, ially when this is due to circulatory disturbances of either the l or bile vessels, and in inflammatory or degenerative processes, is to say, in diseases which involve the entire organ in the morbid ss; but, on the other hand, the form of the liver may undergo le changes as a result of inflammation (ordinary cirrhosis), and ially in case of tumors, such as hydatid cysts, cancer, or abscess. he consistence may remain unaltered or it may vary from an c hardness to that of stone, or from a soft state to actual fluctu- . In passive hyperæmia and in biliary stasis the consistence of rgan is somewhat increased; it is hard in carcinoma, very hard pertrophic cirrhosis, and like a stone in amyloid degeneration. ses of echinococcus cysts or of abscess there is an elastic con- ce or the organ is soft or even fluctuating, always so when the collection is near the surface of the organ and accessible to pal- n. But this same consistence may be found in cases of cancer, s been cited several times by A. Biondi. Of special importance peculiar feeling of tension which is almost always observed in id cysts of the liver and which is frequently a valuable differen- gn from other hepatic diseases.

mors of the liver, like all subdiaphragmatic tumors, are dis- l during respiration, following the movements of the dia- m. These movements may be absent, however, in certain cases, when as a complication of the hepatic trouble the movements diaphragm are slight or even absent; (2) when there are ad- s to the abdominal wall or to the spinal column; (3) when is a marked displacement of the liver as a result of relaxation ture of the suspensory ligament; (4) when the tumor of the nas attained a very great volume so as to press up into the ic cavity.

e shape of the margin of the liver is sometimes a fact of some stic value which may be obtained by palpation; thus in hyper- nd in hepatitis the edge remains sharp, in amyloid degenera- becomes obtuse, and it is nodular like the rest of the organ in f carcinoma.

der the head of palpation we have to mention the perihepatic n and the hydatid thrill. The perihepatic rub is a peculiar

sensation like the creaking of leather, which may also be heard on auscultation. It is produced by the rubbing together of the roughened parietal and hepatic layers of peritoneum. It may be appreciated by placing the hand on the hepatic region near the border of the ribs during the respiratory movements.

The hydatid thrill, which was until recently regarded as a sign of great value, is gradually losing its reputation in this respect. There are several methods for eliciting this phenomenon. The most common consists in holding the left hand on the tumor in order to feel the fremitus which is produced by percussing with the right hand in the neighborhood. Davaine advises that three fingers of the left hand be placed over the most prominent portion of the tumor and that percussion be then made on the middle one of these. Eichhorst says that the sign can be most easily elicited by placing the index and middle fingers of the left hand, widely separated, on the tumor and then percussing lightly with a finger of the right hand between them. The hydatid thrill may be compared to the sensation felt on percussing with the pulp of the finger on a mass of rather firm jelly. Luzzato has compared it to the sensation obtained by percussing a bladder filled with water. This phenomenon has been regarded by many as pathognomonic of echinococcus cysts, whence is derived its name of hydatid thrill. The interpretation which has been given to this symptom is very varied. Cruveilhier thought it was caused by the rubbing of the daughter cysts against each other. Concato looked upon the membrane as the most important factor in the production of the thrill, for he says that he has observed it in the case of sterile cysts, and also that it could not be obtained when the walls of the cyst had undergone calcareous degeneration. Others explain it as due to little waves of fluid accompanied by vibrations in the tense walls of the cyst. The latter appears to be the more rational explanation and the one which corresponds more exactly with the clinical facts. The hydatid thrill is not present in all cases of echinococcus cysts, indeed it is quite rare in them, and it is also met with in other affections, such as ovarian cysts or simple ascites. Caradelli reports a case in which this sign was very distinct and in which at the autopsy there was found a right hydronephrosis. Thus we see that the hydatid thrill has lost all its value as a means of diagnosis and has become simply a semeiological curiosity. Its only significance is that it indicates the presence of a fluid contained in a tense sac, a condition which can be recognized by many other physical signs.

Palpation gives us equally important results as regards alterations in the gall-bladder. This viscus may be found enlarged, soft, and

uating in dropsy of the gall-bladder, and sometimes, when there
insuperable obstruction in the cystic or common duct, we may
ble to empty the bladder by moderate pressure, hearing at the
time a peculiar gurgling sound (Nothnagel). We may also, in
in cases in which the abdominal walls are thin and the gall-
ler is not tense, be able to feel the contained gall-stones and
a peculiar rubbing sensation, such as that felt on squeezing a
of marbles or pebbles. This sensation will be the more evident
nore numerous and rough are the stones and the less abundant
e fluid in the gall-bladder.

efore leaving the subject of palpation of the liver we must refer
method proposed by a recent writer for exploring the gall-
ler. The patient standing erect, the examiner places the left
against the right flank of the patient, making pressure so as to
the liver as far forward as possible, while the right hand, some-
hollowed, is placed against the anterior surface so that the tips
e four fingers grasp, as it were, the inferior margin of the ninth
the thumb is then pressed in below the costal border. With the
iner's hands in this position and making gentle but firm pres-
the patient is instructed to expire strongly, when it will be pos-
for the right hand to reach the lower border of the liver and the
ladder. The author of this method claims that by means of it
s been able to palpate the gall-bladder even when the conditions
perfectly normal.

Percussion.

hen it is desired to determine by percussion the limits of the
along the anterior surface of the chest, the patient must lie on
ack, but must be standing, sitting, or lying on his left side if it
ired to mark out the posterior limits of the gland. The liver
a solid organ gives a flat sound on percussion, but this dulness
absolute over the whole extent of its surface, since the sound is
ied by the layer of pulmonary tissue which overlaps the upper
nterior surface of the liver; the dulness of the liver, therefore,
hat of the heart, is to be distinguished as relative and absolute,
e two the most important as regards the clinical data which it
hes being the absolute dulness. The upper limit of this abso-
ulness corresponds on the parasternal line to the upper border
sixth rib, on the mammary line to the lower border of the sixth
n the anterior axillary line to the upper border of the seventh
n the posterior axillary line to about the same point but a trifle
on a line with the angle of the scapula to the ninth rib, and on
ravertebral line to the eleventh rib. The upper line of liver

dulness passes transversely forwards, but presents a curve with convexity looking upwards at the level of the anterior axillary line. These are the limits as given by Thierfelder, but Weill says that the dulness begins on the axillary line at the sixth intercostal space and on a level with the angle of the scapula at the tenth rib.

The lower limits of the liver dulness are to be determined by means of rather light percussion, since strong percussion may give rise to a tympanitic sound caused by the transmission of the vibrations in the liver to the adjacent stomach and colon. But the lower limits of the liver can be more certainly determined by palpation than by percussion. When, however, the latter method is employed we should start and percuss the abdomen from below upwards, and in this way we can more readily appreciate the change in the percussion note as it passes from the tympanitic note of the abdomen to a duller sound as we reach the liver. The inferior line of dulness is found in the median line from 2 to 4 cm. ($\frac{4}{5}$ to $1\frac{3}{5}$ in.) below the costal arch; on the mammary line it corresponds with the costal arch in the adult while in children and women it is somewhat lower; in the axillary lines it reaches to the tenth intercostal space; on the line passing through the angle of the scapula it is found at the upper border of the eleventh rib, while at the paravertebral line it is lost in the dulness of the right kidney.

Laterally the liver dulness varies considerably; it may end at the left border of the sternum or may even reach the left mammary line. Frerichs says that it may vary between 5 and 7 cm. (2 and $2\frac{4}{5}$ in.) from the median line of the sternum. Posteriorly the liver dulness extends to the vertebral column.

Weill has sought to determine the area of absolute dulness of the liver and finds that in the median line its length is 7 to 9 cm. ($2\frac{4}{5}$ to $3\frac{3}{5}$ in.), on the parasternal line from 9 to 12 cm. ($3\frac{3}{5}$ to $4\frac{4}{5}$ in.), and on the axillary from 7 to 11 cm. ($2\frac{4}{5}$ to $4\frac{3}{5}$ in.); on a line with the angle of the scapula it is only from 4 to 6 cm. ($1\frac{3}{5}$ to $2\frac{3}{5}$ in.).

Of course the position of the liver varies greatly according to the shape of the thorax, and it also changes during respiration, the area of hepatic dulness varying according to the changed position of the liver and to the greater or lesser degree to which the organ is covered by the lungs. It also varies according to the position of the patient.

The relative dulness of the liver is that obtained by percussion over the part covered by the lungs. As we approach the limits of the liver, percussing over the lungs, the full, clear pulmonary sound acquires a higher and shorter timbre, becoming gradually more and more dull and less clear till finally it merges into the flatness of the uncovered liver. The upper limit of this relative dulness runs

llel with that of absolute dulness and is separated from it by a nce of 3 or 4 cm., thus not corresponding exactly with the upper er of the liver, which latter is about 5 cm. (2 in.) above the line of lute dulness. We see, therefore, that a part of the liver is inacces- to examination by percussion, since it is covered by a portion of so thick as to be incapable of transmitting the dull note of the . It is, nevertheless, important to determine the limits of rela- dulness of the liver, since the knowledge so acquired may serve n the diagnosis of pathological changes in the upper surface of iver which would otherwise escape all objective research.

he area of hepatic dulness is reduced in cases of atrophy of the n, such as simple atrophy, acute yellow atrophy, shrinking of the after the opening of an abscess or an hydatid cyst, and especially nary atrophic cirrhosis, in which we often find that the dulness ating the presence of the left lobe of the liver has entirely dis- ared, for that lobe is often and early attacked by this morbid ess. But the area of dulness of the liver in cirrhosis is often ap- ntly less than it is in fact, because of the presence of ascites or orism. But the pathological conditions in which the liver is apparently reduced in size are more numerous than those in h there is an actually diminished area of dulness; this is seen in meteorism, abdominal pneumatosis, ascites, wandering liver, ionary emphysema, and pneumothorax.

When the intestines are distended with gas there is an enlargement ie entire abdomen, the walls of which are tense, and the base ie thorax is widened, in consequence of which the liver is ed farther to the right than in normal conditions and undergoes t of rotation backwards, so that a smaller portion of its surface is ntact with the thoracic walls. The anterior surface of the liver so frequently covered by the distended intestine, or when this is so the latter presses so firmly against the liver that percussion over this gland is transmitted to the intestine and gives a tym- ic resonance.

n abdominal pneumatosis the area of liver dulness is decreased igh the same causes. The distended intestine, indeed, does not against the liver, but instead of this there is gas in the peritoneal y which covers the liver and completely masks its dulness.

scites, independent of the lesions of the liver which may cause it, also occasion a change in the area of hepatic dulness. It is the important that we should remember this apparent reduction in of the liver in cases of ascites, since there may be a close con- on between ascites and an actual reduction in size of the liver.

he area of hepatic dulness may be absent in cases of movable

liver, almost always a congenital condition, or of transposition of the viscera, which is always a congenital condition. The diagnosis may be made in the first case by finding the liver in some other part of the abdomen, and we may occasionally, by a dexterous manœuvre, return the organ to its normal position. The diagnosis in the second case may be made by determining the mutually altered positions of the liver and spleen.

The most important of the thoracic conditions which may cause an apparent reduction in size of the area of hepatic dulness is pulmonary emphysema. This causes an increase in volume of the lungs, especially of the margins at the base, while the thorax is dilated, being always in position of inspiration. We have, therefore, two conditions which narrow the area of hepatic dulness, namely, the intrusion of the lungs into the costophrenic space, and also a slight rotation backwards of the liver, resulting from the widening of the base of the thorax. Owing to these causes the area of dulness is reduced chiefly at its upper portion, and in cases of marked emphysema the liver dulness in the mammary line may commence at the seventh rib or even lower.

Finally it is easily understood how the upper border of dulness is lowered in right pneumothorax. The air which has penetrated into the pleural cavity, especially when it exists there under strong pressure as often in pneumothorax, distends the entire cavity, thus filling completely the costodiaphragmatic space and covering directly the antero-superior surface of the liver. In cases of marked pneumothorax the liver may also be displaced downwards.

The area of hepatic dulness may also be enlarged, either actually or apparently, that is to say, either because of actual increase in size of the liver or by reason of lesions affecting the neighboring organs or cavities. The most common causes of enlargement of the liver are active or passive congestion, inflammatory processes (hypertrophic cirrhosis, parenchymatous hepatitis), fatty and amyloid degeneration, and neoplasms. A. Biondi has noted a law which he found to be constant, namely, that when there is an increase in volume in these cases there is also an increase in weight, hence the liver sinks down in the abdomen and the increased area of dulness is abdominal rather than thoracic. An exception to this law, however, is found in inflammatory processes, especially abscess of the liver or hydatid cyst, in which the increased dulness may be chiefly thoracic; the reason for this is that the inflammations of the liver being ordinarily accompanied by perihepatitis, adhesions form beween the liver and the diaphragm and thus prevent the descent of the former.

An apparent increase of the area of hepatic dulness is more com-

than an actual increase. Among the causes of this we have first otice rickets, in which the organ is compressed through the de- ity of the chest and increased as regards its longitudinal diameter. refore in patients in whom we find a lateral curvature of the e and chicken-breast, we need not conclude too readily that the r is enlarged because we find its area of dulness increased. The e effect which occurs as it were naturally in rickets may be pro- d artificially in women by tight lacing.

Many changes in the thoracic organs may also cause apparent ease in the liver dulness. Thus morbid conditions which pro- dulness of the base of the right lung, especially croupous pneu- ia and pleurisy with adhesion, may be a source of error, suggest- an enlarged liver. Error may, however, be avoided by taking of the physical signs of the pulmonary lesion. In order to dif- tiate between the dulness of an enlarged liver and that due to risy with effusion, it is important to note the course of the upper of dulness. Von Jaksch has shown that the upper edge of the dulness presents a line gradually descending from before back- ls in a curve with concavity looking downwards, while in pleu- effusions the exact opposite occurs, the line being one with avity looking upwards. This difference is readily explained by natomical conditions present. Of course, in order to obtain this rential sign the patient must be examined in the erect position. her point of differentiation is that the dulness due to pleuritic ion varies more or less according to the position of the patient, when extensive adhesions are present; but the line of liver ess is constant or nearly so, whatever the position of the patient. lthough this is the rule, we must note, however, that there are exceptions in which disease of the liver was found at autopsy, ugh the direction of the line of dulness as well as its variability ested the existence of pleuritic effusion. Cantani reports a case is kind in which there was a large abscess of the liver which lated the dulness of a pleuritic effusion.

here are other conditions, much more rare, however, than those mentioned, which may cause an apparent increase in the area of dulness. Thus a tumor or a collection of fluid between the r surface of the liver and the diaphragm, subphrenic abscess, cause a downward displacement of the organ and thus simulate rtrophy. The upper margin of the dulness may present itself as a curve with concavity downwards, passing from before back- s and from above downwards, and in this case it would be impos- to distinguish the condition from one of real enlargement of the

Bright was the first to notice a clinical fact of this kind, and

Murchison reports a case in which there was an encysted collection of peritoneal fluid between the liver and the diaphragm which simulated an enlarged liver, while in fact the organ was actually atrophied. These cases are, however, very rare, and the other symptoms will usually suffice to indicate the nature of the trouble.

An apparent increase in the size of the liver may be due to tumors of the kidney or of the mesentery, the dulness caused by their presence being continuous with that of the liver. A differentiation can usually be made by observing the irregular contour of this dull area and also by a consideration of the other clinical symptoms. Tumors of the stomach or colon may also suggest an enlarged liver, but the percussion note in these cases is not absolutely flat, but has a tympanitic quality due to the air contained in these hollow viscera.

Auscultation.

Under normal conditions the heart sounds may be heard quite clearly upon auscultation over the liver, and it has even been claimed that the limits of this gland may be more accurately defined by auscultation than by percussion. It has been contended, however, that this may lead to error, since the heart sounds may be propagated along the ribs, and auscultation can therefore be relied upon only for defining the lower edge of the liver below the border of the ribs.

Certain arterial murmurs may be heard on auscultation of the liver, namely, those caused by compression of the aorta, by aneurysm of this vessel or of the hepatic artery, or, finally, by a stenosis of these vessels. Thus a loud arterial murmur may sometimes be heard in cases of compression of the hepatic artery by diffuse carcinoma of the liver or an impacted gall-stone. In cirrhosis also, when there is well-marked compensatory circulation, a venous murmur may be heard originating in the accessory portal system. Finally, and this perhaps is one of the most valuable signs furnished by auscultation of the liver, we may sometimes hear a friction sound, the perihepatic rub, which, as we saw above, may also be felt upon palpation.

Functional Symptoms.

After the analytical study already made of the pathological and clinical reations between the liver and other organs as regards their anatomical and functional changes, little remains to be said of the symptoms of the altered function of the liver as manifested in functional changes of other organs.

Cardiovascular Phenomena.

'e may have here either purely functional disturbances or actual ›logical changes, constituting a true cardiac complication of the ic disease and sometimes even by its gravity obscuring the pri- affection, which may then appear to be the consequence rather the cause of the heart trouble. We shall not undertake to in- ɛt the pathogenesis of these secondary cardiac disturbances, we have already treated of them at length above, but shall ibe simply the clinical facts as they are observed in practice. ›f the most common of these cardiac symptoms is palpitation. patients complain of a passing irregularity of the cardiac pul- ıs rather than of long-continued palpitation, and when examin- ıch patients we find in addition to an increased frequency of the a slight irregularity in the force of the apex beat, a rather strong ›eing followed by several weaker ones. Together with these car- listurbances we may have some symptoms pointing to disordered ion, such as pyrosis and epigastric oppression, and these may some influence in the production of the cardiac irregularity. ıison has noted in some cases of hepatic disease an exaggerated ion of the larger arteries, especially of the abdominal aorta, marked in the epigastric region, which he attributes to imper- igestion in persons of a nervous constitution. These exagger- ulsations, not only of the aorta, but also of other arteries, inde- nt of renal cirrhosis or of aortic regurgitation, is, according to uthor, due to a pathological condition of the blood following f hepatic origin.

slow pulse, apart from that occurring in icterus, of which we ıpeak more at length in a subsequent section, may often be seen ɛs of hepatic disorder accompanied by irregularity and inter- ce. In such cases the disturbed heart action may be due to psia consequent upon the hepatic disorder. The irregularity termittence of the pulse accompanying diseases of the liver are y more marked during repose than when the patient is exercis- ıd they may also be provoked by the use of certain articles of

ırchison has described two cases of angina pectoris which he ed as due to uricæmia, caused, according to his theory, by c lesions. We have already expressed our views concerning ation between gout and diseases of the liver, and we cannot ad- at the stenocardia is dependent upon disorders of the hepatic ›n, but believe that it is the direct consequence of uricæmia,

that is, of a special disturbance of metabolism having no pathogenic relation with diseases of the liver.

The anatomical lesions of the heart and of its valvular system dependent upon diseases of the liver regard chiefly the right side; but the mitral valve may also suffer in like cases, and there may be symptoms of cardiac insufficiency without any true valvular lesion, especially when large tumors of the liver interfere mechanically with the heart's action.

Digestive Disturbances.

The intimate relations existing between the liver and intestine explain the frequency, we may even say constancy, of digestive disturbances in cases of hepatic disorder. But we must remember that disturbances of gastrointestinal digestion are often not the effect but the cause of diseases of the liver. Thus dyspeptic disturbances caused by the ingestion of irritating substances or by over-eating determine marked hyperæmia of the intestine and of the stomach, and thus secondarily cause hyperæmia of the liver. Certain cases of cirrhosis which cannot be explained by alcoholism or infection have recently been shown to be the result of autointoxication. We must therefore distinguish carefully between the digestive disturbances consequent upon hepatic disease and disorders of the liver resulting from indigestion.

In certain cases of liver trouble the tongue may present nothing abnormal, but in many cases, especially when the disease has existed for a long time, there is a whitish viscous coating of the tongue with many prominent and congested papillæ, especially around the edges and at the tip. The appetite may be preserved, but usually there is complete anorexia, with loathing for fatty substances, and this especially when there is a defective flow of bile into the intestine. The patient, especially when there is jaundice, may complain of a bitter taste in the mouth, which is due to the irritation of the gustatory nerve terminations by the biliary salts circulating in the blood.

The most constant trouble and one which furnishes a valuable guide in treatment, is dyspepsia due especially to intestinal acholia and to venous stasis. The patients complain of a weight in the epigastrium, eructations, marked pyrosis, and sometimes even vomiting. These gastric disturbances may occasionally end in chronic catarrh of the stomach, and the physician may sometimes be led into error and regard the gastric disorder as the only trouble, overlooking the causal affection of the liver. As one of us (Semmola) has pointed out, a careful analysis of the urine will show whether we have to do with a primary intestinal affection, or with one secondary to a disease of the liver.

Constipation is sometimes present in hepatic disorders and may ue to acholia or to venous stasis. At other times, instead of con- ation there may be diarrhœa, or the two states may alternate. usually stated that diarrhœa is due to increased secretion of bile, h stimulates the muscular coat of the intestine. But this may be caused by a catarrhal condition of the mucous membrane re- ng from venous stasis.

Another order of symptoms is seen in hemorrhages from the ach or intestine the pathogenesis of which we have already ex- ed in the previous section. In cases of hepatic disease accom- ed by circulatory disturbances in the portal system we frequently œsophageal varices or hemorrhoids. The latter are seen chiefly rrhosis, but may also accompany functional disturbances of the .

The symptoms of indigestion due to acholia will be treated of specially in the section on jaundice.

Ascites.

One of the most interesting conditions as regards the symptoma- gy of diseases of the liver is ascites. This is recognized by the metrical enlargement of the entire abdomen, noticeable especially ally as the patient lies on his back. The umbilical cicatrix is ened or sometimes even protuberant. In man this ascites may be accompanied by hydrocele. Upon percussion, flatness is ob- d, which varies according to the position of the patient. Another rtant sign which distinguishes ascites from solid tumors is fluc- on. In women information of some value may be obtained by al exploration. When even a comparatively small amount of (200–300 c.c.) is present the uterus will be found apparently ss weight and the cervix will be characteristically movable. In tion to these objective symptoms there are also functional ones h may be of great importance not only by way of diagnosis, but as furnishing an indication for speedy therapeutic measures. most important of these is dyspnœa, which may in certain cases o marked as to threaten the life of the patient. This is due to t pressure upon the thorax, not only by the fluid in the abdomen, also by the tympanites which almost invariably accompanies as- . In addition to dyspnœa, there are also a number of other tional disorders, among which we need only mention an aggra- n of the already existing gastric and intestinal disorders, disturb- s in micturition due to pressure on the bladder, œdema of the minal walls or of the lower extremities, albuminuria from com- sion of the renal veins, etc.

Ascites must be differentiated from other fluid collections within the abdomen, especially from ovarian cysts. Information of much value may be obtained from the shape of the area of dulness and the changes occurring in it during the varying positions of the patient. Thus when the patient is on her back the upper limit of the dull area is a curved line with concavity looking upwards, while in ovarian cyst the concavity looks in the opposite direction. In the case of an ovarian cyst there is always tympanitic resonance in the lumbar regions, even when the patient is lying on her back, but in ascites there is dulness in these regions. The change in the upper border of the area of flatness in cases of ascites without marked distention will also serve to distinguish this condition from an ovarian cyst. The fluid in the two conditions varies, that of ascites being a limpid, pale-yellow serum of a specific gravity of 1.015, containing a large amount of albumin, while the ovarian fluid is viscid and often of a brownish or chocolate color due to the presence of blood.

We may also occasionally have to distinguish between ascites and a distended bladder, or cystic tumors of other abdominal organs, such as hydatid cysts of the liver and hydronephrosis, but error may be avoided usually by care in making the examination.

Urinary Symptoms.

The results of urinary analysis are always of the greatest value in assisting to a diagnosis of liver troubles. The urine being the final product of metabolism and containing the waste products of intraorganic combustion, constituting thus the final expression of all the biochemical processes taking place in the cells and in the tissues, affecting the alimentary substances circulating in the blood as well as the protoplasm of the cells, we should expect to find in it important modifications of composition when the functions of the liver are interfered with by disease. Returning to the comparison, which we made in a previous section, of the liver as the tender of a locomotive, the urine might well represent the ashes, which would naturally vary in quantity and quality according to the materials contained in the tender which are employed for combustion. Indeed, the physician should never neglect an examination of the urine, since this will furnish him with a guide perhaps even more sure than that of the objective symptoms in arriving at a diagnosis of hepatic disease. We shall, therefore, endeavor to describe briefly all the changes which the urine may undergo in cases of liver disease, reserving, however, a description of the urine in jaundice until we come to the section especially devoted to that condition.

Beginning with the physical characters of the urine, we note that

quantity excreted in twenty-four hours is almost always reduced, times even to 200 or 300 c.c. (about 7 to 10 ounces). This oli- may be the direct consequence of the hepatic disease, or it may e result of the same causes as those producing the liver disease; in heart disease we may find a nutmeg liver and a diminished tion of urine, both due to the same cause. In some cases, how- we may find polyuria, as, for example, in diabetes of hepatic n. In this case, however, the dependence of the polyuria upon epatic disorder is by no means certain, since very probably both ependent upon some nervous lesion.

he reaction of the urine in cases of hepatic disease is not of value. The acidity is usually increased by reason of the greater ntration of the urine, and to the same fact is due the increase ecific gravity. An increase in specific gravity should therefore ad us to infer that there is an increase in the solid constituents e urine, for there may be a reduced amount of solids with in- ed specific gravity when the amount of fluid is also reduced. ve must not forget that the increased specific gravity may in n cases indicate an actual increase in the solid constituents, as urea, albumin, or sugar.

ne urine is almost always changed in color in diseases of the Aside from the change of color due to the presence of bile nts in jaundice it is usually highly colored, turbid, and deposits undant reddish sediment of urates colored by uroerythrin.

ne quantitative examination of the urine is very important from point of view, as the information obtained will be a guide to us nly in diagnosis but also in prognosis and therapy. We may tate a law which the clinician ought never to forget when he finds lf in the presence of a patient suffering from hepatic disease. there is an increased elimination of urea, so that instead of the ge of 25 to 30 gm. (375 to 450 gr.) in the twenty-four hours we rom 35 to 40 gm. (525 to 600 gr.), we ought to suspect the ex- e of an irritative process in the liver with, however, preserva- of the anatomical and functional integrity of the hepatic cells, led there are no febrile symptoms or any other signs of mark- listurbed metabolism. When, on the other hand, examination urine reveals a notable diminution in its daily excretion of if this cannot be explained by a retarded metabolism from other cause, we must admit a destructive process in the liver, g as its necessary consequence an interference with the func- activity of the organ. We believe, therefore, that in the man- nt of a case of hepatic disease, the most important sign we by which we can determine whether our remedies are having

a beneficial action and are meeting the rational indications, is an increase in the quantity of urea excreted. We must, however, remember that a marked diminution in the amount of urea is not necessarily an indication of hepatic disease, but may often be a sign of the renal disorder complicating the affection of the liver. There may then be not only a diminution in the amount of urea formed in the organism, but also an impeded excretion of that already present. The amount passed during the twenty-four hours may be reduced to from 15 to 20 gm. (225 to 300 gr.) in some cases, but in others to very much less, even 50 cgm. (7½ gr.) a day in cases of serious destruction of the liver substance (acute yellow atrophy) complicated with renal insufficiency.

Some authors have thought that great importance should be attached to the variations in the amount of uric acid contained in the urine, an increase of the urates being regarded by Lecorché as a constant symptom of many hepatic lesions. We have stated above our opinion as to the uricogenic function of the liver, holding that this organ has no part in the formation of uric acid, but rather reduces the quantity in the organism by converting it into urea, a more soluble and therefore more easily eliminated body. The presence of large amounts of uric acid in the urine may be therefore a sign of diminished functional activity of the liver. And, indeed, in cases of liver disease accompanied by marked destruction of the parenchyma of the organ, there will be, together with a diminished excretion of urea, an increase in the amount of uric acid which has escaped the oxidizing process in the liver.

The determination of the amount of urobilin and of uroerythrin is important. Although urobilinuria is not an absolutely pathological fact, yet the constant presence of this substance in large quantity in the urine is an important indication of hepatic disease. We shall discuss this symptom and its significance more at length when we come to treat of the pathogenesis of jaundice, and we need only remark here that urobilin may unquestionably be of hepatic origin. When there is a secretory insufficiency of the liver this organ elaborates urobilin as well as bilirubin from the coloring matters of the blood. Uroerythrin, which corresponds to the rocacic acid of Proust and the purpurin of Golding Bird, is a urinary pigment, the importance of which in a diagnostic sense is constantly increasing. Like urobilin this substance is an evidence of anatomical or functional disorders of the liver, and Reale attributes to erythrinuria the significance of an intense urobilinuria. When there occurs an exaggerated destruction of red blood corpuscles uroerythrin appears in great quantity in the urine in which is found concurrently a large

at of urobilin. Indeed, in paroxysmal hæmoglobinuria, after sappearance of the hæmoglobin from the urine, we find uroery- ria together with urobilinuria (de Renzi, Reale, Riva). We eadily understand how uroerythrinuria and urobilinuria may symptom of hepatic insufficiency, the liver being unable to orm the blood pigment, set free as a result of some morbid s from the red blood cells, into bile pigment, but succeeds only sforming it into an intermediate pigment, urobilin. Semmola own that in many hepatic disorders, especially cirrhosis, the ty of uroerythrin is inversely to that of urea excreted in the -four hours.

ere are other pathological elements also which may be found in ne in cases of hepatic disease and which are an expression of the l disease of the liver or of the consecutive renal disorder. We nd albumin and peptone in the urine in cases of hepatic affec- The former is more often an expression of the renal disorder, either occurs secondarily to the liver disease or is caused by ne factor as the latter. We have already referred, however, to ion of the liver upon the albumin coming to it in the blood as t of intestinal absorption, and we can readily understand, there- ow a diminution of this action of the liver, as a result of ana- or functional disturbances, may prevent the conversion of albumin, the unchanged portion being then eliminated by the s. The importance which we believe is possessed by pepton- a symptom of hepatic disease has not yet been granted to ll authorities, although many have noted the presence of pep- the urine in such cases. One of us (Semmola) has shown that uria may be a prodromic symptom of cirrhosis of the liver, regards it as an expression of hepatic insufficiency, the organ nable to effect all the necessary biochemical changes in the opeptone brought to it in the blood coming from the intestine. microscopical examination of the urine may also give very e information in revealing the presence of leucin and tyrosin. substances are the indication of an advanced destruction of atic parenchyma, and are found especially in affections such yellow atrophy. But although we recognize the evil signifi- the presence of these bodies, we are unable to say whether dependent upon an interruption in the functional activity of r or are in relation with the destruction of its elements. facts would seem to point to the latter supposition as being rect one. Thus we find these substances in greatest abun- the urine when there is a very great destruction of the liver nd furthermore, it has been seen in experiments upon geese

that the amido-acids are split up even after total extirpation of the liver, whence it has been concluded that the leucin and tyrosin come directly from the destruction of the liver substance. We do not believe, however, that the facts in our possession warrant us as yet in regarding the question as settled. Leaving out of consideration the results of experiments upon geese, since they are by no means positive and since also the chemistry of the hepatic functions may be different in those animals and in man, the fact that tyrosin and leucin are found more abundantly in the urine in cases of extensive destruction of the liver substance may be interpreted in various ways.

The pathological changes occurring in a large number of the liver cells render this organ unable to transform the extractive matters coming to it as a result of intraorganic combustion, and these, accumulating in the organism, are eliminated in part in the urine. The discovery of these substances in large amounts in the liver is no sign that they are formed there; these bodies are but slightly soluble, and when brought to the liver by the blood they stagnate there, being no longer transformed into the more highly oxidized and more soluble end products. There is also a general law which no pathologist or clinician should ever forget, namely, that every morbific agent, infectious or toxic, has its organ or tissue of election upon which it exerts its specific action. We might cite examples of this without end, but we need only mention the action of the typhoid bacillus upon Peyer's patches and the spleen, and that of strychnine upon the spinal cord. This law may possibly explain the presence of leucin and tyrosin in large quantities in the liver, accumulating there not as a direct result of destruction of the liver cells, but indirectly in that the organ is no longer able to exert its transforming action upon these substances. Chemistry teaches us that these bodies, together with creatin, creatinin, xanthin, hypoxanthin, uric acid, etc., are the products of incomplete oxidation of albumin, the final degree of oxidation of which is represented by urea; and furthermore, all recognize the fact that the liver is the chief organ concerned in the formation of urea. In cases of serious destruction of the hepatic substance in the urine we find a diminution of urea together with the presence of leucin, tyrosin, and other products of the incomplete oxidation of proteid matters. We may, therefore, conclude that the explanation of the decrease of urea and of the presence of the other bodies is a diminished oxidizing function of the liver. We must not forget, however, that these nitrogenous compounds resulting from the incomplete combustion of albumin may be found in the urine also in the course of infectious processes, especially in typhoid fever. But even in these

their presence may be due to the existence of hepatic lesions
ı occur so often in the infectious diseases.
ome deny that the presence of these substances is of great diag-
e importance, and von Jaksch says that we often find under the
scope what is called tyrosin but which on chemical examination
nd not to be this body at all. But even though it may be pos-
to make a mistake of this kind occasionally, nevertheless that
l not take from the value of its presence as a diagnostic sign of
lesions of the liver.
e may finally mention certain other substances which are oc-
ıally found in the urine, the presence of which, however, is of
loubtful significance. Murchison, for example, has asserted that
mia and urinary calculi may be of hepatic origin. Among the
he includes calculi of cystin, xanthin, and oxalate of calcium, as
s those of uric acid. Cystin and xanthin are substances which
osely related to uric acid, being apparently the result of an in-
ete oxidation of the albuminous matters arising from imperfect
onal action of the liver; and the relations between oxalic acid
ric acid make it reasonable to suppose that oxalic calculi may
med in cases of hepatic disease. All these facts are, however,
m demonstrating that urinary calculi are an evidence of the
ıce of disease of the liver. Lépine has remarked upon an
se of phosphoglyceric acid in cases of hepatic steatosis. But
er valuable this might be as a sign of hepatic steatosis, the
tion of the amount of phosphoglyceric acid is too difficult a
s to make it of general application.
ere remain two other urinary symptoms of hepatic disease
must be studied here, namely, alimentary glycosuria and the
ic coefficient.
mentary Glycosuria.—In the remarks upon physiology which
ed our study of the general pathology of the liver, the import-
as made manifest of the glycogenic function of the hepatic
ıat is, its power of transforming into glycogen the glucose
l from intestinal absorption, this glycogen being a true reserve
t which being later gradually re-converted into glucose is
off by the blood in the suprahepatic vessels to nourish the
. This physiological interpretation of facts explains the pa-
cal processes which may occur. Should the hepatic cell be in
ormal condition it would be unable to transform completely the
absorbed from the portal system into glycogen, and this,
g unchanged into the general circulation, would be eliminated
kidneys, thus constituting alimentary glycosuria. To prove
sence of this symptom, from 100 to 150 gm. (three to five ounces)

of simple syrup is given to the patient, fasting, and the urine passed in the next seven to eight hours is carefully collected. It is then subjected to a chemical examination, and should glucose be found, it is a proof of the existence of alimentary glycosuria.

This symptom has been attributed to a functional or an anatomico-pathological lesion of the hepatic cell, rendering it incapable of transforming all the sugar ingested into glycogen. Colrat, Couturier, Lépine, Roger, Hanot, Dujardin-Beaumetz, Surmont, etc., hold it to be an important indication of functional insufficiency of the liver. But as Colrat, Couturier, and Lépine have shown clinically, and C. Bernard and Pavy have demonstrated experimentally by occlusion of the portal vein and its radicles, this glycosuria may be dependent upon the development of a collateral circulation, the blood charged with glucose derived from intestinal absorption not passing through the liver, but entering directly into the general circulation. In addition, it is interesting to observe that the sugar may be seized upon by the other organs; we know, for instance, that muscle glycogen exists, it having been proved by many experiments that the muscles are capable of transforming glucose into glycogen. Now it might be possible that even where there is a grave lesion of the liver, with anatomical and physiological alterations of its parenchyma, the sugar ingested would be in part retained by the liver and in part by the whole muscular system, in which case the condition of hepatic insufficiency would not cause the symptom of alimentary glycosuria. Moreover, gastrointestinal lesions frequently coexist with hepatic disease, and by their interference with the digestion and absorption of nutritive matters prevent the appearance of alimentary glycosuria; this would be due to the non-absorption of the sugar and not to the fact that the liver is in such a condition as not to be able to convert it into glycogen.

Taking into consideration the various circumstances which may interfere with the production of alimentary glycosuria, Weill states that there are four conditions necessary to its production: 1. Normal absorption by the intestines; 2. Unimpeded circulation in the portal vein, or the development of a collateral circulation; 3. A diffuse lesion of the hepatic cell; and 4. A diminished absorption of sugar by the tissues.

Considering the cases in which alimentary glycosuria does not occur in spite of the existence of grave hepatic lesions, it is evident that this symptom can give us but little information in regard to the degree of severity of the hepatic process. For this reason the conclusions reached in regard to its value by some practitioners who do not bear in mind the many researches made since 1876 (Valmont,

ke, Landouzy, Herard, Déjerine, etc.) are most inexact. These
ches have demonstrated that the problem is a complex one,
limentary glycosuria is by no means always due to hepatic in-
ency, but that it may also occur in cases of grave cachexia, and
whenever the nutritive interchange is seriously interfered with.
om our own clinical experience we are able to affirm that ali-
ry glycosuria is frequently absent in cases in which the extent
hepatic lesion would have led one to expect to find it, while
n appears in cases in which the affection of the liver is in its
stage, or in cases of disease of other organs and systems.
special diagnostic importance can be attached to the symptom
ficial alimentary glycosuria.

otoxic Coefficient.—We have already devoted ample space to a
eration of the protective power of the liver, and to this power
the chief importance of the toxicity of the urine in hepatic
e. This new and interesting method of studying the func-
condition of the various organs in different diseases we owe to
ard, who in 1877, in his famous lectures upon autointoxica-
rst called attention to the toxic condition of the urine. Many
rious were the researches which followed; a great step in ad-
and one which opened out a broader horizon to the physio-pa-
y and rational therapy of the subject was taken by Semmola,[30]
monstrated the complete relation which exists between those
oms of toxæmia which are manifested in many diseases, and
ic phenomena produced by the intravenous injection of urine
mals; this demonstration served to explain the clinical symp-
nd to determine in doubtful cases whether they were due to a
orbid process or to an elective toxic action, thus giving us a
orthy guide to the appropriate and rational treatment to be
d.

origin of the toxicity of the urine may be inferred from the
at toxic substances are constantly being formed in the intimate
mical work of living tissues, in the nutritive and respiratory
ges of the cells, in the successive metamorphoses of intestinal
m and absorption, which consist of the most complicated proc-
of oxidation, dehydration, breaking down, and formation of
ts, and in the action of intestinal saprophytes, and that the
sm is constantly endeavoring to rid itself of these toxins
h the emunctories. The liver, as we have already noted, is
the most important of the emunctory organs. When, by
of destructive morbid processes its functional powers are
d, a large number of toxic products accumulate in the blood
e eliminated by the chief emunctories of the body, the kid-

neys. It is in this way that in severe parenchymatous affections of the liver there is an increase of toxins in the urine, and the urotoxic coefficient is increased just as in the event of retardation of the exchanges in the organism, this toxic coefficient is considerably decreased.*

This fact is dependent upon the close functional relation between the liver and the kidneys; there is, in other words, a true physiological compensation, which provides a sort of natural remedy, freeing the system from autointoxication. Many researches have been made with a view of determining whether in hepatic diseases there is any increase in the toxicity of the urine, and whether any prognostic sign of value could be drawn from this fact. Bouchard,[31] and then Feltz and Ehrmann[32] investigated the toxicity of the urine in jaundice due to organic disease of the liver, and found it increased. Roger[33] in his work on the action of the liver on poisons reports some cases of liver disorders in which the urine was hypertoxic. Lipari[34] also found that the toxicity of the urine was above the normal in hypertrophic cirrhosis, while in atrophic cirrhosis it was about half of the physiological amount. Surmont[35] made further investigations, and reached the following conclusions: In catarrhal icterus the urinary toxicity is not increased, but often appears to be diminished during the height of the disease; it increases during the period of recovery, especially at the time of the urinary crisis when there is increased elimination of urine and of biliary pigments. In chronic diseases of the liver there is a more or less marked increase of the urinary coefficient if there are anatomical lesions of the hepatic cell, as in atrophic cirrhosis, neoplasms, and serious degenerations; this does not occur unless a large number of the hepatic cells are profoundly affected, so that in hypertrophic alcoholic cirrhosis, in which

* Bouchard has called attention to several important points. He studied the toxic power of the urine, and established an exact method for determining its degree, and he also endeavored to discover what influence the various physiological and pathological conditions of the organism had upon it. He noted that the urine secreted during sleep has a convulsive action, while that passed during waking hours is narcotic in effect and more poisonous. He demonstrated the fact that these two varieties of poison are mutually antagonistic. This determined, he analyzed normal urine to find its toxins, and was able to isolate seven toxic bases, of different character and biological action. The average urotoxic coefficient of human urine in rabbits, according to Bouchard, is 0.465. To determine this coefficient we first determine the urotoxin, dividing the amount of urine used to kill the rabbit by the weight of the animal; we then seek the amount of urotoxin in the twenty-four hours, dividing the total volume of urine for that time by the amount of urine necessary to kill 1 kgm. of animal. Dividing the amount of urotoxin in twenty-four hours by the weight of the man whose urine we are testing we obtain the urotoxic coefficient.

ells are still able to exert a protective influence, the urotoxic co-
ent is not increased. The same writer noticed the rather close
ion existing between the increase of the urinary toxins and ali-
ary glycosuria; both of these symptoms, being the result of he-
insufficiency, are apt to appear together, and demonstrate the
acity of the hepatic cell to react to the various physiological
uli.

Iore recently L. Bellati,[36] under the direction of Professor Cola-
, has made many accurate experiments upon dogs to determine
rotoxic coefficient, and has come to the conclusions: 1. That in
tic diseases there is no relation between the factors of regressive
morphoses (azoturia, etc.) and the toxicity of the urine; 2. That
rinary toxicity, aided by the various hepatic lesions, without
constant rule either keeps pace with or is in inverse ratio to the
gen and the urea; 3. That the liver exerts a real protective ac-
for the organism against poisons of autoformation; 4. That a
tant relation exists between the extent of the anatomical and
ional lesions of the liver and the urinary toxicity.

Finally, Semmola, as the result of extensive clinical observations
h confirm the great diagnostic importance of this protective
ion of the liver during the course of various chronic diseases,
come to the conclusion that a biological examination of the urine
solutely indispensable when, during the course of an hepatic
se, some acute or paroxysmal symptom suddenly occurs, because
as noticed that these attacks, which have sometimes been sup-
d to be due to malarial infection, are in reality forms of toxæmia
to imperfect or totally suppressed protective action on the part
e liver.

t would appear to be clearly proved that in hepatic diseases of a
uctive nature the toxicity of the urine is nearly always increased,
for this reason the symptom is of diagnostic importance. It is,
ver, not enough to prove the presence of urotoxin, but the uro-
coefficient should also be accurately ascertained. To stop short
e urotoxin, that is to say, at the amount of urine necessary to
ne kilogram of rabbit, might lead us into grave error, and the
t obtained could not be accepted as of any value. In hepatic
ses, as for instance in cirrhosis, the daily quantity of urine is
ently diminished for various reasons, as, for example, abdominal
s, a complicating peritonitis, the anatomical condition of the
eys, etc., and it is quite natural that in such cases the urine
ld be hypertoxic, because the normal toxic principles are in an
ual state of concentration, and the increased amount of urotoxin
herefore not be regarded as a symptom capable of rendering any

assistance in either the diagnosis or the prognosis. To determine the urotoxic coefficient which is in relation both with the amount of urine eliminated and with the weight of the body, would be more to the purpose, but even this does not impress us as the valuable measure which it is considered by many to be. In fact, we believe that it may have no importance at all when jaundice is present. We have in our practice seen many cases of severe stasis icterus accompanied by the abundant elimination of biliary pigment in the urine which possessed a urotoxic coefficient of sufficient degree to kill a rabbit when only a few cubic centimetres were used, and yet in which no other symptoms pointed to the presence of grave anatomico-pathological lesions of the hepatic parenchyma, and a perfect recovery ensued. As we said before, the increase of the toxicity of the urine in such cases is due to the pigments and the biliary acids, and may therefore be a symptom of the intensity of the jaundice rather than of the anatomical or functional condition of the liver. Yet a determination of the urotoxic coefficient may throw much light upon hepatic diseases other than those accompanied by jaundice.

Grave hepatic alterations may exist without the toxicity of the urine being increased, as for instance when, as frequently occurs, an anatomical lesion of the kidneys coexists with an hepatic lesion; when, in other words, renal insufficiency is superadded to hepatic insufficiency, it is evident that the autointoxication determined in the organism being of high degree, and usually causing death, the kidneys are at the same time insufficient to the compensatory task of eliminating the toxic substances which have accumulated in the blood. We have had occasion to observe a case of ordinary cirrhosis at a very advanced stage, complicated by nephritis, accompanied by a serious condition of toxæmia with intense nervous symptoms, in which the urotoxic coefficient, although frequently tested for, was never found to be increased, and, indeed, seemed rather to be lower than normal.

In the case of grave hepatic lesions which do not involve the whole of the parenchyma of the liver, as for example in cancerous neoplasms, it is easy to understand that in the liver itself there may be physiological compensation of great value; the hyperactivity of the healthy portion of the gland may be adequate to the protective function required, and thus may be avoided the accumulation of toxic products in the blood with the consequent hypertoxicity of the urine. This may be the explanation of those cases of gumma and of cancer of the liver in which the urotoxic coefficient is not increased.

Furthermore, even in cases of grave and diffuse degeneration in which the ecbolic properties of the hepatic cell must certainly be

usly impaired, this symptom may be entirely wanting. As a these degenerative forms are accompanied by profound changes neral nutrition which cause all the chemico-molecular processes e body to become notably diminished, so that even the amount xic substances produced is lessened; if then there remains suffi- activity in the hepatic cells which have been unaffected by the se to neutralize or to destroy the action of these diminished ns, we can readily see that in spite of the existence of grave de- ion of the parenchyma of the liver there may be no increase in rotoxic coefficient.

ne absence of this symptom therefore should not lead us to form nclusion that the hepatic lesion is necessarily of small impor-

greater diagnostic value is the presence of increased toxicity es where jaundice is absent. But here also certain influences independently of the hepatic lesion, tend to raise the urotoxic ient. This increase may be dependent upon the cause of the ic lesion, especially when that is an infective disease. We for instance, that the urotoxic coefficient is higher than normal laria, which is the starting-point for so many cases of cirrhosis. rile movements connected with affections of the liver it is not ble to attach too much importance to increased toxicity of the for Feltz and Ehrmann, Lépine, and Aubert have shown that ine of fever patients is hypertoxic.

en aside from the reasons above given, it is very certain that ne cases of hepatic disease, although the urotoxic coefficient e increased, it cannot be considered a very reliable diagnostic tion, since causes unknown to us may act to bring about the se. Practice daily demonstrates the truth of this assertion. ve in many cases of hepatic disease investigated the condition urine, and have become convinced that even a notable increase urotoxic coefficient does not inevitably point to a marked lesion hepatic cell.

is toxicity of the urine may (as may the alimentary glycosuria) aid in the diagnosis if it be found in connection with a train r symptoms, but alone it has not the importance with which metimes credited.

Toxic Symptoms.

e protective power exerted by the liver against external poisons as against those produced within the tissues and during the ses of intestinal chemism, explains the ease with which toxic oms arise in diseases of the liver. These may involve all the

organs, and cause so great a variety of symptoms that to describe them all would be to enumerate all the phenomena possible in the various diseases of the liver. They include cardiac disturbances, renal lesions, the results of grave hæmatolysis, and the most severe nervous phenomena.

It is our intention to describe the toxic symptoms occurring during the course of diseases of the liver when treating of each special affection of that organ. With reference to what was said in the section upon the relations of the liver and nervous system (p. 438), we may here state that when functional nervous disturbances cannot be attributed to the pouring out of bile into the blood, then, instead of adopting Murchison's theory of uricæmia, we should rather believe them to be due to the action of other substances whose nature we shall not understand until biological chemistry shall have given us definite knowledge of the metabolic processes in the liver; then, and not until then, we shall be able to give a complete picture of the morbid symptoms of that toxæmia which occurs with such frequency in hepatic diseases.

Hæmatic Symptoms.

There is great uncertainty as to the relation of the composition of the blood to the hepatic functions, and complete obscurity as to the effect which disease of the liver may have upon the blood and upon hæmatogenesis. While we are still occupied with the question as to whether the liver is concerned in the formation of the blood corpuscles or in the destruction of those which have been rendered incapable of performing their destined work; and while, for the reasons given above, we do not yet know exactly what alterations the blood undergoes in passing through the liver, we can readily understand that the more minute investigations of the structural condition of the blood in patients suffering from hepatic disease have not resulted in the demonstration of constant alterations which might be of diagnostic and therapeutic value. The researches which have hitherto been made into the diminished resistance of the blood corpuscles to external agents in ordinary cirrhosis, and which if enlarged in scope to include other hepatic changes might lead to some result, have been too limited and too one-sided to be of special importance.

It is probable that the investigations of pathologists should be directed to the chemical changes undergone by the blood in the liver, rather than to its morphological variations. It is very certain that the blood is not excluded from the complex biochemical processes which are continually taking place in the liver; in fact, it is more than likely that in this tissue more than in any other occur the chemical

omena of oxidation, dehydration, decomposition, composition, which have so far escaped the power of chemistry to explain. It be taken as a proof of this assertion that the temperature of the l is increased by its passage through the liver, that in the supra- tic veins being warmer than the blood in the portal vein; and we that the production of heat in the organism is related to chemical omena. Researches have been begun with a view to determine pecific gravity, the degree of alkalinity of the blood, its power agulation, etc.; but while these investigations do honor to the intentions of the experimenters they are not sufficiently broad eir scope to be of great value.

Nutritive Symptoms.

the several disturbances of the biliary secretion which are met it very often occurs that the digestion of either the albuminoids fats is interfered with, and at the same time absorption is not nplished as readily as usual, the final result being a diminished al nutrition. If at the same time bile is emptied into the blood, jurious action upon the cells and tissues will further contribute ir malnutrition, and a true cachexia may be the final result.

oreover, the lesions of the liver cells may by the most com- processes induce so great a nutritive disturbance as in itself stitute the most serious symptom of the disease and occasion- he immediate cause of death. In fact, the structural changes gone by the hepatic cells so alter their functional powers that protective influence, as well as their capacity for storing up nent, is seriously impaired.

ycogenesis and lipogenesis are interfered with, the disintegra- f the proteids derived from the activity of the nutritive inter- e no longer occurs in normal fashion, and protection of the or- n from the effects of the toxic matters derived from intestinal sm is destroyed, and in this manner all the phenomena of ab- on, assimilation, and decomposition are interfered with in their important point, the final result being grave nutritive disturb- which almost always arise during the course of hepatic disease, hich may assume the form of a veritable cachexia, completely ating all the other symptoms.

om Murchison's researches in especial we know that there are mena of abnormal nutrition dependent upon functional disturb- of the liver which may cause an abnormal deposit of fat, in words, obesity, or the opposite extreme of emaciation. It is o understand that impairment of the powers of the liver should probable cause of some form of hereditary obesity, from the

knowledge that we possess of its lipogenetic functions. There may be an abnormal tendency upon the part of the glycogen, or of the glucose absorbed from the intestines, to become changed into fat, or still more probably, there may be incomplete oxidation of the fatty substances. Theories apart, we know that in animals fed largely with farinaceous, sweet, or fatty substances the proportion of fat in the hepatic cells is much greater than in animals which are kept on a limited diet and given a great amount of muscular exercise.

The emaciation may be dependent upon functional disturbances of the liver, and is, as we know, one of the most marked, and in some cases diagnostic symptoms of diabetes, a disease whose connection with changes in the liver we have already noted.

The functional symptomatology, varied, complex, and inconstant as it is, possesses, as we have seen, the greatest diagnostic and prognostic importance in hepatic diseases, and in many cases is of value in determining the treatment. Circulatory and nutritional disturbances are of especial importance in diagnosis, while urinary and toxic symptoms are valuable in prognosis, and lesions of the digestive tract should receive more attention than they now do in the line of therapeutic indications.

Having described the objective and functional symptomatology of liver affections, only icterus and its symptoms remain to be discussed.

Icterus.

We have reserved a special section for the consideration of the symptoms and pathogenesis of icterus, both because of its great importance in the study of hepatic diseases and for the sake of greater clearness.

In the most varied morbid changes of the liver we find this special phenomenon which attracted the attention of physicians in the earliest days of medicine, but which only of late has been elucidated as to its pathogenesis by means of a vast amount of study, experimental research, and clinical observations. The importance of this symptom is shown by the fact that when we meet with it in practice we are always justified in suspecting an anatomical or functional disorder of the liver. The intimate relation between jaundice and hepatic lesions having been clearly demonstrated, its occurrence is of the greatest diagnostic importance even in complicated cases. While all the other functional disturbances caused by hepatic disease may possibly be so interpreted as to throw doubt upon the diagnosis, icterus always leads to the belief that some disorder, whether primary or secondary, is to be found in the liver.

efinition.—Although modern researches have given us a very idea of icterus and of its pathogenesis, it is far from easy to an exact symptomatic and etiological definition of the phenome- because both the cause and its mode of action may be extremely l. We will merely state that by the term icterus we mean a id symptom characterized by a special yellowish pigmentation tissues and fluids of the body.

Symptoms.

he description of icterus is somewhat complicated, because it s to the functional disturbances of so many organs and tissues. ve clearness to an analytical study of its various aspects we will them into: 1. Cutaneous symptoms; 2. intestinal symptoms; retory symptoms, and 4. symptoms of autointoxication (cho-).

Cutaneous Symptoms.

anges in the color of the skin constitute the most evident symp- f icterus. The yellow tinge may vary from a coloration so pale be easily taken for anæmia, to the color of sulphur, of saffron, reenish-yellow, or, rarely, to a blackish-yellow (black jaundice), r to the bronze tint of Addison's disease. Not only the skin, he visible mucous membranes are affected, as, for example, the nctivæ, the labial and the genital mucosæ, etc.

here are certain points of greatest intensity of coloration which ell to bear in mind. The yellow tinge appears first upon the nctiva covering the sclerotic, where, owing to the transparency membrane and to the fact that the background is white, it is evident. It is also apparent upon the mucosa of the lips and ccal cavity, especially by the sides of the frenum linguæ, and e the more readily seen if by pressure we render the parts ic. If the patient force his mouth wide open, which causes arts to become anæmic, two yellow streaks may be seen upon ft and upon a portion of the hard palate. The coloration ppears at the labial commissure, the nostrils, forehead, neck, abdomen, and limbs, this order of appearance depending, of , upon the comparative thinness of the skin in these various ns of the body, and not upon a gradual distribution of the nt.

rked daily variations may be noticed in the coloration, espe- in the cases of retention icterus, as, for example, in biliary is, when the jaundice increases during the attacks of colic and ishes during the intervals, this being due to the greater or less

elimination. Daily variations are also caused by the diet, by the amount of bile secreted by the liver, by the rapidity with which this is transported by the blood, and by the activity of the intestines and the kidneys, as we have already pointed out.

Finally, the yellowish tint is more pronounced in the aged, especially if the skin is wrinkled, than in the young, in the fat, or in those whose natural coloring is very light.

The earlier physicians reported cases in which the jaundice was present in the form of spots, or upon one side only of the body. Thus Frank, under the name of *icterus dimidiatus*, describes a case in which the icterus occurred upon one side of the body only; this fact he explains by assuming that the cutaneous fixation of biliary pigment is under the influence of the nervous system, and that a unilateral anatomical or functional lesion of this system would result in the non-pigmentation of the affected side. Although this hypothesis is by no means proved, it is not impossible that the nervous system may play a part in the causation of these phenomena, and that it also may in some way influence the cells of the Malpighian layer when they become impregnated with the pigment which is abnormally circulating in the blood.

When the icterus is slight in degree, and in its early stages, the yellow color of the skin is derived from the blood plasma, which is icteric in color, but later, owing to the process of osmosis continually taking place between the blood and the tissues, the latter become thoroughly impregnated, and then the yellow coloration of the skin may be said to be autochthonous. The bile pigment becomes fixed in the Malphigian layer and is found in its cells in the form of fine granules, which almost completely fill the protoplasm. It is of interest to note (and will help us to understand why the yellow coloration of the skin persists even after all traces of biliary pigment have disappeared from the urine), that the Malphigian cells have so great an affinity for this pigment that, when after cholæmia of long duration they have become impregnated with it, they no longer give it up to the nutritive fluids, but retain it until they exfoliate. This accounts for the persistence of jaundice for a certain length of time, even when the morbid cause has entirely disappeared and the anatomical lesions and functional alterations have been fully compensated.

Not only the skin, but every tissue and apparatus of the body, as well as normal or pathological fluids, become impregnated with the biliary pigments, and turn a yellow color. This fact is clearly revealed at the autopsy, the intensity of the coloration depending upon the severity of the jaundice and the original color of the affected tissues.

liver, in whose disordered functions the jaundice originates, oes important alterations, some of which may be considered cause and some as the effect of the icterus. Of the first we peak when treating of the various hepatic diseases.

liver is found to be of a greenish-yellow color, or even of a reen. Examined histologically, the cells are seen to contain ish granulations, which are sometimes arranged in masses and protoplasmic portion surrounding the nucleus. The biliary es are distended with bile, and the intralobular bile capillaries en filled with greenish masses of bilirubin, which have the ap- ce of true pigment calculi. The pressure of bile in the small ries may finally become so great as to cause rupture, and the it exuding among the hepatic cells presses them together so crobiosis may result.

kidneys of patients suffering from icterus are enlarged and ddish-yellow color, the capsule being easily detached. Upon , their parenchyma is seen to be of a greenish-yellow, espe- n the cortical portions. Under the microscope we can see ere is an infiltration of pigment in the cells of the tubules in we may sometimes even find small crystals of bilirubin. These are those of the lighter forms of icterus, but they may become re as to constitute an intense inflammatory process, or, in other a nephritis secondary to the elimination of irritating products.

liver and kidneys are of course the organs most sensitive to sence of biliary pigment in the blood, the first because in its functional powers is found the pathogenesis of the jaundice, second because they constitute the most important organ of ation for the bile poured out into the blood; but other organs are affected. The pleura, peritoneum, and pericardium, the s, subcutaneous fat, bones, and tendons are also stained yel- d to a less degree the brain, nerves, pancreas, salivary glands, etc.

n the fluids of the body are stained by the bile circulating in od; the aqueous and vitreous humors, for instance, being so that some have attributed to this fact the visual disorders of e, which in reality are due to other causes. If pathological ion or transudation occurs in a patient suffering from jaundice, id will be stained yellow because it is entirely composed of erum in which the biliary pigments are dissolved.

ltration with pigment is, however, not the only alteration un- e by the skin. A tormenting pruritus is often observed. It after the jaundice has been present for some time, although reports a few cases in which it preceded the icterus. It varies

in intensity, sometimes becoming a source of the greatest suffering to the patient; there may be periods of exacerbation and again of intermission. It is apt to be worse at night. In a few cases the pruritus extends over the whole body, but it is usually most severe in certain spots, having a predilection for the plantar surfaces and interdigital spaces of the hands and feet. As a consequence of this itching, we find excoriations, desquamations, and true prurigo papules.

It is admitted by all that the cause of the pruritus is the biliary pigment, which, being deposited in the Malpighian layer, irritates the terminal fibres of the sensory cutaneous nerves. Eichhorst reports the case of a celebrated physicist who maintained that he had particularly noticed that before the appearance of the jaundice he had felt a sensation of coldness at certain parts of the skin, and this he had also noticed in the case of others. This fact would suggest vasomotor disturbances due to the physiological action of the biliary acids.

Cutaneous eruptions may accompany icterus, which may be erythematous or bullous, or there may be true erythema.

There is one special skin lesion in jaundice, especially if the disease be of long standing, an eruption which was first studied by Addison and Gull, who gave it the name of vitiligoidea, and which has been minutely described by Strauss under the more familiar name of xanthelasma. This eruption, which is usually limited to the eyelids, but which in some cases extends over the whole cutaneous surface, is composed of slightly elevated spots of a pale yellow color, or of nodules of varying sizes, elastic or cartilaginous in consistence, of a reddish color with small yellow opaque spots. It is always at the internal canthus of the eyes, whence it extends to the upper, and in a less degree to the lower eyelid (Hutchinson), exhibiting a marked tendency to asymmetry. The nodular form is painful, and is often accompanied by intense and incoercible headache. Xanthelasma may also occur upon the mucous and serous membranes, and in the parenchyma of the viscera. Its origin has been attributed to the irritating action of the constituents of the bile upon the cutaneous elements. From an anatomico-pathological point of view we may distinguish two stages of the disease: the first or irritative, in which there is nuclear proliferation; and the second characterized by the fatty degeneration of both the preëxisting and the newly formed elements of the connective tissue.

Other lesions of more or less severity may be found upon the skin, such as ecchymoses, vibices, etc., these phenomena being in relation with the frequently infectious cause of grave icterus.

Intestinal Symptoms.

important part taken by the bile in intestinal digestion and ion will readily explain why decided pathological alterations unctions of the gastroenteric tract follow disturbances in its n.

his connection we must premise that there may be two condi- the determination of icterus: either a lessened flow of bile intestines because of some obstacle in the bile passages, or a cretion of bile, or an increase in its density. We must, there- stinguish between *acholia* and *polycholia* or *intestinal pleio-*. This question will be taken up more at length in connec- h the pathogenesis of icterus; it has been referred to here for the sake of pointing out the intestinal phenomena which accompany jaundice, no matter what its origin.

stinal acholia, or failure on the part of the bile to reach the es, should really be called intestinal hypocholia, as the cases xtreme rarity, in which there is a total absence of bile in the es, there being simply a diminution in the flow owing to some nent or other.

bear in mind the various physiological functions of the bile ntestines, it will be easy to understand what may occur when vented totally or in part from reaching the duodenum.

most important action of the bile in emulsifying fats and thus g them capable of absorption by the chyliferous vessels, will the fact that the most noteworthy symptom of intestinal is the diminished digestion and absorption of the fatty mat- he food, and we find, in fact, that the fæces may contain as seventy-eight per cent. of fat.

her important function of the bile consists in the total sus- of the peptonization of the chyme so soon as it reaches the m, and this it accomplishes by precipitating the pepsin. has demonstrated the physiological necessity for this func- showing that pepsin destroys the pancreatic ferment, which ow has an important action upon the digestion of albumi- ts, and carbohydrates. If then the bile do not reach the s, or be present in insufficient amount, the digestion of the ids and carbohydrates is interfered with, and that of the rther hindered.

he digestive processes are seriously disturbed in intestinal that is to say, there is a constant lack of digestion of the fs ingested, and general nutrition consequently suffers.

dition to this interference with the transformation of alimen-

tary substances by that long series of processes in regard to which physiological chemistry has perhaps not uttered its last dictum, there is a disturbed condition of the absorptive powers of the intestinal mucous membrane. Physiology has shown that osmosis occurs most readily when the membrane separating the two fluids is saturated with bile, so that if during the period of digestion the intestinal mucosa is not thus moistened, absorption is obstructed and this further tends to reduce the nourishment of the organism, even though an abundant and nutritious diet be taken. Absorption is also interfered with by the fact that in intestinal acholia there is lacking the stimulus to peristalsis of the biliary acids. These normally act in such a way that the muscle fibres of the villi contract at intervals, thus emptying the contents of the lymphatic spaces into the larger lymphatics, so that the former are thus prepared for the absorption of more food (Schiff). At the same time it would seem that even the muscle fibres of the intestinal walls receive some stimulus from the bile, possibly through the mesenteric plexus, and it is easy to understand how the peristaltic movements exert a favorable influence upon the intestinal absorption.

Constipation is another important result of a deficient or suppressed flow of bile into the intestines; it is due to the lessened normal stimulus imparted by the bile to the muscle of the intestinal walls.

The bile, furthermore, possesses an antiseptic action, by means of which it prevents or lessens intestinal fermentation. For this reason, in intestinal acholia, abnormal fermentation occurs with the production of gas, causing meteorism, which may lead to the most serious complications on the part of the intestines. These conditions are aggravated by other factors already mentioned; the insufficient emulsion of fats and the disturbed digestion of the albumin, owing to the non-action of the pancreatic ferment, allow these two alimentary substances to undergo decomposition and fermentation, which are most injurious to digestion. The constipation, moreover, which is a constant symptom in acholia, adds to the putrefaction of the ingested matters by the retention in the intestines of the products of the disordered digestion.

The tongue is clean in exceptional cases only; as a rule it has a whitish or grayish-yellow coating. There is marked anorexia with nausea, the mere thought of food tending to cause vomiting. There is often a bitter taste in the mouth, due perhaps to stimulation of the gustatory papillæ by the bile circulating in the blood. Putrid eructations and fetid rectal flatulence are frequent. Intestinal meteorism is more or less marked, and constipation is constant.

e appearance of the fæces is of importance; they are pale in and may even be of the grayish-white chalky aspect, which has l them to be called argillaceous. The light color is in part due absence of biliary pigment (to which the fæces owe their usual sh-brown appearance), but even more to the presence of a large t of undigested fat, which constitutes a veritable stearrhœa. searches of Müller have shown that in normal conditions from ten per cent. of non-absorbed fats are found in the fæces, but hen there is complete absence of bile in the intestines the t of undigested fat may rise as high as fifty-two to seventy- per cent. That the whitish color is chiefly owing to the pres- f fats may be demonstrated by the fact that when we give such s a diet almost devoid of fats, as did Strümpell, the fæces are ger white, but light brown. The microscopic examination of es will be of great value in these cases.

a normal condition drops of undigested fat are not found, but, ller showed, in healthy adults the fat contained in the fæcal s is in the form of vitreous plaques, irregularly polygonal in either colorless or yellowish, chiefly composed of calcareous In intestinal acholia, however, as Nothnagel[37] has demon- in his valuable monograph, the fat is often found in the fæces form of needles; Gerhardt found an enormous amount of crys- organic matter in acholic evacuations, which he supposed to be . One of his pupils, however, Oesterlein, determined by the al properties of these crystals that they were composed of the lime and magnesium and of the higher fatty acids, and von is of the same opinion. Chauffard[38] divides the fatty crystals o groups, the first formed of free fatty acids, occurring in the long slender needles gathered together in fine bundles, or in form; these are soluble in ether, and are reduced by heat to rops. The second group is composed of saponified fats, and in the form of short crystals, rectilinear and terminating in nts, resistant to the action of ether and of heat. A chemical ation, which on account of the necessarily long and compli- rocesses is practically very difficult, shows that in acholic ere is an absence of biliary pigment together with the pres- a large amount of free and saponified fatty acids.

ld the icterus be accompanied by polycholia instead of acho- ntestinal symptoms will of course be entirely different from st described. An amount of bile greater than the normal, and more dense from the presence of pigments and biliary acids romia), is poured into the intestines; the fæces as a conse- instead of being pale, are of a dark color, sometimes of an

intense greenish-brown, and so soft as to constitute a true biliary diarrhœa. These phenomena are due to the fact that the increased stimulus to the mucous membrane by the quantity or the quality of the bile induces increased peristalsis and thus diarrhœa. A chemical and microscopic examination of the fæces shows a large amount of bilirubin and an absence of fat crystals.

Secretory Symptoms.

All the secretory functions being necessarily in relation with the blood crasis, it follows that in all varieties of jaundice there are marked symptoms on the part of the secretions of the body, since the starting-point of icterus is the presence of the component parts of the bile in the blood. The most important chemical modifications are undergone by the excretions whose function it is to eliminate from the body by the medium of the blood whatever abnormal substances happen to be present. We refer to the protective power exercised by the excretory organs of the body. The bile compounds represent foreign and injurious elements in the organims, and are therefore eliminated by the most important excretions, the urine and the perspiration.

For this reason one of the most marked and constant symptoms in icterus, of whatsoever origin, is the presence in the urine of biliary pigments. This change may be said to precede the staining of the skin, as in a number of cases it has been met with several hours previous to the pigmentation of the sclerotic. This fact will be readily understood, if we bear in mind that before the cells of the skin become jaundiced they present a certain amount of resistance to their invasion by the pigment, and that during this time the kidneys, whose function it is to relieve the blood as rapidly and thoroughly as possible of the bile pigments which have accumulated in it, represent the most direct way of elimination.

The presence of bile pigments in the urine, if of any extent, is revealed by its color; this may vary from orange yellow to a reddish brown like Malaga wine, or even to coffee color; in a few cases it is of a greenish-yellow, which always indicates the profound disturbance of nutrition which accompanies injurious processes in the liver. It is clearly dichromic, because by reflected light a greenish reflection may be seen, especially at the margin. If icteric urine be exposed for a while to the light it becomes greenish. Blotting-paper, silk, or cotton impregnated with this urine becomes yellow, and it is usual to find small yellow stains upon the shirt, the result of drops of urine which have fallen after micturition. If icteric urine be shaken, there will be formed a greenish-yellow scum which is very persistent.

pecific gravity of the urine is increased, unless there should be conditions (hypoazoturia) which tend to diminish its density. action is acid. It is usually decreased in amount, this diminu- aving an important bearing upon the prognosis.

ie most characteristic point about icteric urine is the presence e pigment. Gmelin's test is used to detect it. A few cubic cen- res of nitrosonitric acid are poured into a test tube, and the urine examined is poured in very slowly so that by reason of its lesser ty it may float upon the acid.

pigment be present, a series of colored rings will gradually be d at the point of contact, which from above downwards are , blue, violet, and red, the predominant and most important eing the green, which represents the transformation by oxida- f bilirubin into biliverdin. It is worth mentioning that these ons cannot be applied to urine to which alcohol has been added, uppert has shown that alcohol in the presence of nitric acid rise to a beautiful blue-green ring. Rosenbach has introduced ification of Gmelin's process, which consists in filtering a certain it of the urine, and in letting a drop of the fuming nitric acid pon the filter paper, when the colored rings will be seen sur- ing it. This reaction is a delicate one, but, as von Jaksch ob- , it can be used only when the filter paper is absolutely white ure, for the coloring matters contained in impure paper may, treated with the acid, give colored rings identical with those bile pigments.

urechal suggests a reaction which is easy and sufficiently exact: urine in a test tube a few drops of the tincture of iodine are , and if bile pigment is present a fine emerald green will appear. ippert's test is delicate and valuable for the purpose of reveal- mall traces of pigment. Ten to twenty cubic centimetres and one-half to five drachms) of urine are precipitated with vater; the precipitate is filtered out, put into a test tube, and l with alcohol containing sulphuric acid. The filtrate, which e success of the test should be acid in reaction, to which end e sulphuric acid may be added to it, is heated to the boiling If bile pigments are present, the precipitate becomes colored ie filtrate assumes a greenish tint.

cording to the recent researches of Hayem the spectroscope e utilized in the endeavor to find bile pigments in the urine, cause they possess any special spectrum, but because they color ole of the right side of the spectrum.

positively identify the presence of bilirubin in the urine, we have recourse to the chemical tests given above, because more

or less dark coloration may be given by other substances, and were we to judge by color only we might easily be misled. Thus rhubarb and senna both cause the appearance of chrysophanic acid in the urine, which imparts a dark red color; santonin gives a greenish-yellow color of varying intensity. The addition of liquor sodæ, however, produces a reddish tint, which serves to distinguish these cases from those in which there is bile in the urine.

In addition to the bile pigments, icteric urine may contain the bile acids, although in slight amount. They may, however, be absent, as they may be reabsorbed and transformed by the blood. To demonstrate their presence in the urine we use Pettenkofer's test as modified by Strassburger. A bit of cane sugar is dissolved in the urine to be examined, into which a piece of blotting-paper is immersed and then allowed to dry. A glass rod is dipped into pure concentrated sulphuric acid and touched to the dried paper, and at the point of contact a distinct carmine violet-purple coloration is seen, which is especially visible by reflected light. In addition to the bile compounds proper we may find in the urine a special pigment, or perhaps we should say, we may find an increased amount of a pigment which in normal conditions is found in the urine, namely, urobilin.

In light cases of jaundice the appearance of the skin is caused by the presence in the blood, not of bilirubin, but of urobilin, which later is found in large amount in the urine. We shall describe the mechanism of the process farther on.

Jaffé's method of testing for urobolin is the most exact; an acid alcoholic extract is made of the urobilin, which is then subjected to the chemical and spectroscopic tests. Biliary pigments should first be removed from the urine. Hoppe-Seyler advises treatment of the urine by lime water, the passing of carbonic acid through the mixture, and its filtration; Riva prefers the alkaline solution of chloride of barium, which he introduces into the urine until there is no further precipitation, after which he filters and tests the filtrate by Jaffé's method.

It is important to bear in mind that there have been some cases of marked icterus in which neither bilirubin nor urobilin was found in the urine. Mya reports three well-authenticated cases in which bilirubin was found in the blood, but in which the most delicate tests failed to reveal any abnormal pigment in the urine. The reason for this clinical fact is not known; it may perhaps be due to some special condition of the renal epithelium, which renders it unequal to the elimination of the pigments which have entered the blood.

The urine of icteric patients may also contain other pathological

cts which are the direct consequence of the morbid secretion. there may be albumin, renal epithelium, or even urinary casts. count of their yellow color these casts are sometimes called icteric

The importance of renal lesions and of albuminuria in the osis of hepatic diseases can scarcely be overestimated, and they rise from various causes. The albuminuria may be due to a of the kidneys secondary to the elimination of large amounts of y pigment which would act as an irritating agent, and this occurs ere icterus of long standing; or it may point to a grave dyscra- e to autotoxæmia, or to a primary infection. In the latter case ears with great rapidity, and indicating a serious renal lesion course, a bad prognostic symptom. According to the most researches in regard to the influence of the liver upon serum- in, it might also indicate grave functional disturbance of that whether it be in relation with the jaundice or not; in fact, esence of albumin in the urine as a complication of icterus may arded as a sign pointing to the true origin of the disease, espe- in such cases as seem from the onset to be due to retention than to hypercholia.

occo has, however, called attention to an occurrence which some- eads to error in testing for albumin. In some cases of icterus sual tests give a positive result, and yet there is no albumin it, or it exists in smaller amount than would be perceptible by st. When the urine is heated after treatment with acids, a floc- precipitate very similar to albumin is produced, the urine also the same reactions as those of albumin to Tanret's and Roberts' and sometimes to acetic acid and to potassium ferrocyanide; s Grocco has pointed out, the precipitate obtained is due to din, because it is soluble in alcohol and does not give the reaction. To avoid this cause of error, he advises treating ine to be examined with a fiftieth part of its volume of concen- acid, to let it stand for several hours, then to filter it and sub- ie filtrate to the usual tests for albumin.

t the urine only, but other secretions eliminate pigment in ice, so that the skin may be considered an organ of elimination e bile constituents which are abnormally present in the blood. *erspiration* is of a more or less pronounced yellow color, stain- nen which comes in contact with the skin. Cheyne describes se of a patient who first became aware of having jaundice by ct that the handkerchief with which he wiped his forehead was d yellow, and Andral reports a case in which the sweat was v, although the skin and the sclerotic were perfectly normal in rance. It is difficult, if not impossible, to account for these

clinical facts, which are yet beyond dispute in view of the reliability of the observers. We can only suppose that owing to a certain amount of resistance on the part of the Malpighian cells to the infiltration of bilirubin, and owing to the ease with which this is eliminated through the kidneys and the skin, the pigment as it is poured into the circulation finds at once a means of exit and thus does not accumulate in the epidermis. However this may be, the clinical fact is certainly a quite exceptional one, and it is doubtless owing to this rarity that further researches have not been made with a view to a fuller understanding of their pathogenesis.

Bile pigments have been found in the *milk* of nursing mothers, and for this reason nursing should at once be suspended if the mother becomes jaundiced.

The intimate nutritive exchanges between the maternal and the fœtal blood will fully account for the fact that the fœtus is so frequently found to be jaundiced when the mother is affected with the disease; it has been estimated that for this to occur the icterus must have lasted at least fourteen days.

The coloring matter of the bile is entirely lacking in the saliva (although Frerichs and Eichhorst hold the opposite opinion), nor does it appear in the lacrymal secretion. In persons affected at the same time with icterus and with bronchopulmonary disease, the *sputum* has in some cases been found to be colored yellow, even markedly so. It would appear from what has been said that, while all the secretions and excretions may be stained by the bile pigments, the urine is the one which most constantly contains them, and which represents the true physiological compensation of icterus.

Symptoms of Autointoxication (Cholæmia).

The presence of the constituents of the bile in the blood stream, by means of which it is carried to all the organs and tissues, is the essential cause of the various toxic phenomena constantly observed in jaundice. The bile has been considered poisonous since the time of Diedier in the eighth century, and has been proved to be so by the valuable researches of Bouchard, to which allusion has already been made in these pages. Of the different substances which enter into its composition, some, such as cholesterin, appear to be innocuous, the injurious ones being the bile salts and pigments.

According to Bouchard and Tapret, the glycocholate of sodium is toxic in doses of 0.54 gm. per kilogram, the taurocholate in doses of 0.46 gm., and bilirubin 0.05 gm. The researches of de Bruin, which otherwise confirm those of previous investigators, give a slightly dif-

result. According to his calculations the amount of bilirubin sary to produce death is from 0.026 to 0.103 gm. per kilogram, le salts being from three to five times less active. From these figures, which have received ample confirmation from other in- ators, it is easy to see why, when under various conditions the tuents of the bile enter into the circulation, they necessarily ce the functional disorders which from their pathogenesis we nolæmia.

the organism itself, however, are found important compensatory s, without which indeed the slightest disturbance causing the rance of bile in the blood would prove fatal. In other words, here no natural means of elimination, the gradual accumulation principles which are constantly being poured into the blood attain the proportions of a lethal dose, with the inevitable re- We have already seen that the secretions and excretions, pre- ntly the urine, offer an important compensation for the abnor- ndition under consideration. In fact, the urine may perhaps nsidered the only true means of compensation, since from the eginning of the invasion of the blood by the bile it is found tain bile principles, and in this fact may be seen the close func- relation between the kidneys and the liver. It is interesting that when the jaundice is at its termination there is a great se of urine loaded with bile pigment, a fact which has an im- t bearing upon the prognosis of the disease. There are some conditions which militate against the accumulation of bile ples in the blood. From the researches of Schiff, who was the demonstrate the enterohepatic circulation of the bile, and hose of Tarchanoff and Wertheimer it appears that the liver separates, and eliminates from the blood the bile pigments have abnormally become mixed in it. This fact is best seen ans of an ingenious experiment devised by Wertheimer; he ined that sheep's bile injected into the veins of a dog is eli- d unchanged by the liver of the animal experimented upon, dog's bile gives in ten minutes the characteristic spectrum of ep's bile. We can thus understand that the liver in itself af- ompensation by eliminating those biliary products which its rangement has caused to accumulate in the blood. Finally, cipitation within the intestines of a small amount of insoluble gment and the combustion of bile pigments in the blood with bsequent transformation into innocuous products, contribute ce the bile in the blood to an amount compatible with life.

must we forget that the organs and tissues, by becoming im- ted with bile, subtract a large amount of it from the blood and

fix it within their protoplasm, thus contributing to the physiological compensation, whose chief factor, however, remains the urine.

In spite of all this cholæmia does occur, and is made manifest by important and somewhat constant symptoms. The toxic action of the bile products is shown with most emphasis in the disturbances of the cardiovascular function. The pulse is markedly slowed, being reduced to forty or fifty pulsations a minute, or even less, sometimes not more than twenty or twenty-one. This pronounced bradycardia, first studied by Bouilland, is one of the most constant symptoms of icterus; it is present in acute icterus, but in grave icterus of long standing it is absent, and as we have several times had occasion to observe, there may even be tachycardia. The slowed pulse is rather full, but that there is always some lowering of arterial tension both Chauffard and ourselves have demonstrated by estimating the tension with Basch's sphygmomanometer. The graphic tracing by the Marey sphygmograph shows the slowing of the pulse, and also shows that the up-stroke is rapid but rather low, the down-stroke being very oblique, and it may also show some secondary oscillation. In other words, it shows the lowered arterial tension and a certain weakening of the arterial walls, which yield readily to the impulse given to the blood by the cardiac systole, and during diastole return slowly by means of their elasticity to the systolic diameter.

The pulse may in some cases present certain irregularities. Eichhorst has in some cases observed a *pulsus trigeminus*, and variations in the frequency of the beat often occur at short intervals.

Many painstaking researches have been made with the object of ascertaining the cause of these disturbances in the cardiac functions. Röhring, Feltz, and Ritter hold that there is some action of the bile acids upon the moderator system of the heart. Spallitta believes that the intracardiac ganglia alone are affected. De Bruin produced artificial circulation in a frog's heart, and found that bilirubin first slowed the heart's action and then accelerated it, at the same time lowering the pressure. The taurocholate slowed the pulse, the glycocholate increased its rapidity and diminished pressure; but these salts were less active than bilirubin and their action was extended to the whole of the cardiac apparatus, that is to say, to both the muscles and ganglia. Finally, he showed that if the pulse in jaundice is full and strong, it is because bilirubin has a stimulating influence upon the tenth pair of nerves.

In the endeavor to gain some light upon this very interesting question of physiopathology, extensive and interesting experiments have been made by Sorrentino, under the direction of Gioffredi in Semmola's institution.

he results reached are that the taurocholate of sodium is a car-
remedy, having a decided influence upon this organ. It slows
eat and lowers pressure; if used in large doses, the pressure
nues to be lowered, but the frequency of the beat is markedly
ased. The slowing of the pulse is due to stimulation of the in-
ory ganglia, and its acceleration by toxic doses is due to their
quent paralysis. The diminished pressure is related to stimu-
ı of the vasomotor system together with the vasal dilatation, as
arly demonstrated by artificial circulation in extirpated organs
Mosso's apparatus; this shows at the same time that the
dilatation is due to the peripheral action of the poisonous sub-
e. The toxic dose lowers pressure by paralyzing the muscular
of the heart. As a result of these researches, it is easy to
stand how in chronic and severe icterus we have a more or less
ed acceleration of the heart beat, instead of its retardation.
may be owing as much to the exhaustion of the vasomotor
n which has undergone a long-continued stimulation from the
alts accumulated in the blood, as to the paralyzing action of
doses of these compounds upon the cardiac muscle.
ese researches further enable us to understand the lowering of
al pressure in jaundice, and the causes which produce the special
mographic tracings already described; the first is due to the
of the bile salts upon the vasomotor system, the muscle fibres,
ıe walls of the blood-vessels, the second to paresis of the arterial

e injurious and toxic action of the biliary products poured out
ıe circulation is not limited to the cardiovascular system, but
ls to other organs and tissues.
e blood undergoes changes the significance of which will readily
lerstood if we recall the importance of the biological functions
it fulfils. In the first place the blood serum of an icterus
t is rich in pigment, and all the serous effusions which may
in the course of the disease will always have a yellow or green-
pearance from the presence of bile pigment. Many investiga-
ıave been made to demonstrate the presence of the bile acids in
ood, and Pettenkofer, Kühne, and Huppert have succeeded in
so; they must be in very small amount, however, for they are
t eliminated by the urine, and in part oxidized and transformed
her substances.
om many recent experiments it has been proved that jaundice
ts, especially if the disease have lasted some time, sustain a
e diminution of red blood corpuscles, and a diminution in the
ning powers of the hæmoglobin. The specific gravity of the

blood is unaltered. Gioffredi demonstrated that the prolonged use of the taurocholate of sodium causes a notable diminution of the red blood corpuscles and of the hæmoglobin. By the injection of large doses of the biliary acids in animals, the blood corpuscles are dissolved, and hæmoglobinuria and hæmaturia are produced (Hoppe-Seyler, Huppert).

It seems to us of importance to observe that although these investigations have shown the bad effect of biliary acids upon the blood and the red blood corpuscles, care should be taken not to exaggerate these effects; in other words, the changes in nutrition and the anæmia, which may result in true cachexia, should not be entirely attributed to the lesions caused in the blood by the biliary acids. They doubtless contribute to the result, but we must not forget that other and perhaps more important conditions, such as disturbed digestion and absorption, possible lesions of the hepatic cells, etc., may be far more potent causes of the complex nutritive disturbances usually called the cachexia of icterus. The patients in chronic icterus become progressively weaker and more emaciated, and profound depression follows, so that we may meet with true physical or psychical asthenia, or else a condition of great irritability and melancholy.

When the jaundice persists for a great length of time, a tendency to hemorrhages of the mucous membranes may be noticed; this may be due to the blood lesions caused by the presence of biliary principles as well as to the depraved general condition. More or less intense epistaxis, gastrorrhagia, and enterorrhagia may be noticed, and in exceptional cases may be the cause of death (Murchison).

Grave nervous symptoms may occur in icterus, such as acute delirium, stupor, coma, convulsions, muscular tremors, paralysis of the sphincters, a dry and brown tongue, and other symptoms of the typhoid state. The origin of these nervous phenomena is still under discussion.

It would seem that nervous symptoms relating exclusively to the brain should be attributed to involvement of the meninges, and for a time this was supposed to be the case. But autopsies seldom revealed any lesion of the brain or of the meninges. The symptoms were then attributed to the presence of bile compounds in the blood, that is to say, to cholæmia, and many experiments were made upon animals to prove that the bile pigments and acids are excessively poisonous. The injection of bile in animals causes death, but this may perhaps be due rather to the mucus contained in the bile than to its essential principles. Frerichs and other investigators have injected bile from which all the mucus had been removed into the veins of a dog without occasioning nervous phenomena. Moreover, in

ice we find that there is no well-defined relationship between the ty of the icterus and the intensity of the nervous symptoms, for may be present in light cases of jaundice, and altogether absent ve icterus. Should the explanation of this important fact be he organism becomes accustomed to biliary intoxication and he products of the bile are eliminated in the urine, it is evident e cannot connect the nervous symptoms consecutive to icterus he cholæmia. It would therefore seem that they must be due same causes as the jaundice itself. In fact they almost always in icterus gravis, which is a true autointoxication. We mean he autointoxication may be the direct cause of the nervous phe- na as well as of the icterus.

e must not forget that the nervous symptoms may be due to ry or secondary lesions of the hepatic cells, which by interfer- with their protective action would allow of the accumulation of compounds in the organism, and these in their turn would have terious effect upon the nervous system. The latter would t functional lesions, due less to the influence of the toxic prod- f the bile than to the accumulation of substances only partially ormed by the ecbolic activity of the hepatic cells. We might herefore, that they are due, not to cholæmia, but to acholia, thus ng out the idea advanced when treating of the physiopathology liver.

ere are other phenomena which must be considered as symptom- f autointoxication. The temperature of the body in jaundice normal, unless some other cause be present to elevate it; as periments of Legg demonstrate, this is due to the action of the ids and pigments upon the thermogenic centre.

e visual function is apt to be affected in icterus; xanthopsia ccur, and has been accounted for by the supposition that it is a diffusion of the bile pigments in the refractive media of the ore especially in the aqueous and vitreous humors; but there also seem to be some nervous influence, as the symptom is intermittent in character. There may also be nyctalopia and alopia, and these would seem to be due to a functional disturb- of the nervous system, although the recent investigations of indicate that they are related to the general organic changes atic disease rather than to the jaundice.

tients suffering from icterus complain of a bitter taste in the , a symptom which has been interpreted in various ways. At it would seem to be due to the eructation of biliary matter from omach, but when there is obstruction of the bile ducts this is ssible. The phenomenon may then be considered as a conse-

quence of the cholæmia. The bile acids which are very bitter (the pigment is not) by their absorption in the blood would stimulate the terminal nerve fibres which preside over the gustatory function and might lead to a parageusis due primarily to the cholæmia.

This explanation, however, although based upon sound physiological principles, would seem to be inexact, or at least not to represent the only cause; many patients suffering from disease of the liver unaccompanied by icterus, complain of the bitter taste in the mouth, but in these cases other conditions present may account for the symptom, such as the gastric disturbances always found in hepatic disorders, and which are capable of producing this form of paræsthesia.

A word as to the action of the bile upon structural elements. Frerichs, Lebert, Budd, and Virchow have shown that the kidneys undergo important alterations on account of the continual passage of bile constituents. There may, for instance, be fatty and pigmentary degeneration of the epithelium, especially that of the convoluted and straight tubules of the pyramids. Werner, after the injection of small amounts of biliary acids, has seen the cells of the convoluted tubules become clear and vesicular in their internal half, and then become detached and fall into the lumen of the tube. In addition, true crystals of bilirubin may be found in the kidneys, especially in those of the newly born, and may be so abundant as to constitute veritable infarcts.

The symptomatology of icterus, although complex and variable, is of the greatest importance in the study of hepatic disease, and merits the closest attention of the practitioner. The various disturbances caused should be well understood in their entirety in order that we may know the appropriate treatment and give a correct prognosis.

Pathology and Pathogenesis.

Icterus, the nosology of which we have been studying, together with the theories in regard to the various phenomena which may arise in the course of the disease, has always attracted the attention of pathologists and of practitioners, because both of its general characteristics and of the mechanism of its production.

Were we to describe the historical development of the various theories in regard to the pathogenesis of icterus, it would lead us into the discussion and criticism of many facts acquired by a long series of experiments at the hands of investigators of indisputable authority; but in so doing we should trespass upon the limits allowed, and stray from our main purpose, which is to render aid to the practitioner at

ide of his patient. For this reason we shall speak only of ories as have been proved to be true, and will, while en- g to present them in complete form, be as brief as possible, group all the varieties of icterus in a classification which in harmony with what is known of their pathogenesis.

oloring matter of the bile comes from the coloring matter of d. It is generally admitted that the hæmoglobin is first ned into hæmatin, and then into bilirubin; the two last- ıbstances have intimate chemical relations, differing only by cules of water:

$$\underset{\text{Hæmatin}}{C_{32}H_{32}N_4O_4Fe} + 2H_2O = \underset{\text{Bilirubin.}}{C_{32}H_{36}N_4O_6} + Fe$$

s not yet been possible to obtain bilirubin from hæmoglobin of the organism, but by the action of bromhydric acid upon we get an isomer of bilirubin, hæmatoporphyrin.

the intimate relation between hæmoglobin and bilirubin, t be led to think that the transformation of the first into the ould easily occur in all the tissues, instead of being a func- ıliar to the liver, and experimental research has seemed at support this fact upon which is based the whole theory of genous icterus. Latschenberger states that, after injecting l of a horse into the subcutaneous connective tissue of the mal, the tissue surrounding the hæmatic focus becomes satu- th bilirubin. Bilirubin has sometimes been supposed to be old hemorrhagic foci, and this it was which first gave rise to osition that the transformation of hæmoglobin into bilirubin cur, not only in the liver, but in other tissues as well. We hink, however, that this fact has been satisfactorily demon- by the experiments, because it is a very easy thing to con- ıe bilirubin with hæmatoidin which is often present in old agic foci.

r experiments of great interest demonstrate that the transfor- f the hæmatic into bile pigment takes place in the hepatic y. Extirpation of the liver in birds (which survive the opera- eral hours) does not cause the production of icterus, which be produced if the hæmoglobin were transformed into biliru- ther organs and tissues. In a few cases a small amount of appeared in the renal excretion of chickens, ducks, and fter the extirpation of the liver, but this was probably due act that the bile present in the intestines was absorbed and longer arrested by the liver, was eliminated by the kidneys vski, Naunyn).

Further numerous and important researches clearly demonstrate that the hepatic cell only is capable of producing bilirubin from the hæmoglobin of the blood. Hæmoglobin has been injected into the veins of dogs with a biliary fistula, and the amount of bilirubin has been seen to increase; the same increase was noticed after the injection of distilled water, which, as we know, sets at liberty the hæmoglobin of the red blood corpuscles. Birds whose livers were completely removed, were subjected to the action of hæmatolytic substances, such as sulphuretted hydrogen and toluylenediamine, and no jaundice followed, although there was a large amount of hæmoglobin dissolved in the blood, which was afterwards found in the renal excretion.

Researches have been instituted of late to ascertain what changes hæmoglobin undergoes if placed in contact with hepatic cells outside of the organism. It has been noticed that the leucocytes and parenchymatous cells transform the hæmoglobin into another compound, which, however, after an interval of twenty-four hours, again resumes the character of the blood pigment. The hepatic cells permanently transform the hæmoglobin into another pigment called hepatic pigment, which is not absolutely similar to the bile pigment because it does not give Gmelin's reaction; it is dissolved with difficulty in alcohol, insoluble in water, soluble in chloroform and in liquor sodæ. Klein and Hoffman have shown that this transformation occurs only in the presence of carbohydrates, especially glycogen and glucose. This would appear to be due, not to any vital property of the hepatic cell, but to a true protoplasmic digestion, for hepatic tissue pounded into pulp in which the microscope is unable to reveal any whole hepatic cell, behaves in precisely the same way. These researches, although they throw much light upon the origin of bilirubin, also show that other conditions, as yet unknown, must exist for the transformation of hæmoglobin into bilirubin. From what has been said it is evident that the production of biliary pigment is a function belonging exclusively to the hepatic cell, which elaborates it from the blood pigment. This is an important fact, and must constantly be borne in mind to the right understanding of the pathogenesis of icterus. We can easily understand that when biliary pigment exists in the blood, there must be some disturbance of the hepatic function, and as we cannot admit that the hæmoglobin can undergo this transformation in the blood itself or in the tissues, we can exclude icterus of hæmatic origin. We may therefore state positively that icterus is always connected with a disturbance in function of the liver.

Having established the preceding fundamental facts, we are now in a position to consider the various pathogenic factors of icterus,

ll begin with the description of the most common and the old-
wn form, that produced by some mechanical obstruction to the
bile into the duodenum. Its mode of origin is easily under-
Any intrinsic or extrinsic obstruction of the ductus choledo-
f the hepatic duct and its larger branches, or even of one of
all bile ducts, increases the biliary pressure, causing the ab-
n of bile by the lymphatics, and its subsequent appearance in
od. A very simple experiment illustrative of the production of
e in this manner was made by Saunders as early as 1795;
a of the ductus choledochus in dogs produces after a few hours
w coloration of the blood in the suprahepatic vessels, and even
arkedly of the lymph. It is therefore evident that if any ob-
e interposed to the flow of bile into the intestines, whether a
mucus, or a biliary calculus occluding the ductus choledo-
r the hepatic duct, or even a simple swelling of the mucous
ane, or a tumor, or external pressure diminishing the lumen,
onstriction of a number of small ducts, the bile vessels become
rged with bile, and the endobiliary tension is consequently
ed. As soon as this tension has reached a certain limit, the
tics begin to absorb the bile, which is carried through the
duct, thence into the veins, and finally into the arteries.
gh the resorption of bile occurs almost entirely through the
tics, experimental data are not wanting (Lépine) to show
radicles of the suprahepatic veins may also take part in the
. Thus for the production of retention icterus two special
ns are necessary: (1) Persistence of the normal bile-produc-
ction of the hepatic cells; (2) increase of pressure in the bile
s, owing to which the obstructed bile penetrates into the
tic vessels and the suprahepatic veins. The reabsorption of
pends upon the bile pressure being relatively greater than
d pressure in the portal vein, either by an actual increase in
pressure from obstruction to the flow of bile, or by a diminu-
pressure in the portal vein. The latter condition is supposed
to account for certain special forms of icterus. It may be a
icterus of the newly born, because after ligature of the um-
ord the blood no longer flows from the umbilical to the por-
. The jaundice of fasting may also be explained in this
as in a state of prolonged inanition the pressure in the portal
is diminished owing to insufficient intestinal absorption
Bernard, Voit, Naunyn). Finally, among the cases of stasis
we must include those produced by increased pressure in the
system of the liver, in other words, by passive hyperæmia of
gan. We can easily see that distention of the walls of the

veins due to stasis of mechanical origin may compress the contiguous capillary vessels, and that the consecutive constriction of the bile duct would cause an increased pressure of the bile, which would therefore be absorbed. This reasoning, while applying to cases of slight icterus due to hepatic stasis, does not explain the frequent clinical occurrence of a marked degree of stasis unaccompanied by the slightest jaundice. It would seem that in an advanced stage of stasis the secretory pressure of bile is diminished, and that this is compensated for later by an increased pressure due to compression in the bile ducts; in other words, the bile pressure would not be increased relatively to the blood pressure, which, as we know, is the condition essential to the reabsorption of the bile.

Stasis icterus, demonstrable in practice and substantiated by experimental researches, has long been recognized, nor has any question arisen as to its pathogenesis. But both practice and research show that all forms of icterus cannot be explained by this special form of mechanism. We frequently meet with patients who are more or less jaundiced, and yet in whom there is no reason to suppose the slightest constriction of the bile ducts, and, should death supervene, the autopsy will show that there was none.

Investigations of the greatest value have recently been made with the object of ascertaining the origin of such cases of icterus, more especially those due to poisoning. Many drugs, such as sulphuretted hydrogen, toluylenediamine, phosphorus, arsenic, naphthol, etc., act upon the organism to produce jaundice without, however, causing any constriction of the bile ducts sufficient to occasion increased biliary pressure and consequent reabsorption of the bile.

Stadelmann,[39] to whom we owe the most important researches into the physiopathology of jaundice, and which have completely changed our former ideas upon the subject, has demonstrated that icterus may also be due to qualitative changes in the biliary secretion depending upon functional alterations in the hepatic cells upon which toxic agents act with especial virulence. He has in fact shown that certain poisons, toluylenediamine, phosphorus, etc., cause a qualitative alteration of the bile by increasing the amount of bile pigment relatively to the other substances, and thus producing *pigmentory polycholia*, better named *pleiochromia*. The bile thus becomes thicker, this causes more or less interference with its flow, and as a consequence we have absorption through the lymphatics, and to a less extent through the suprahepatic veins. To have a clearer understanding of successive phenomena which occur, we will describe what, according to Stadelmann, takes place in poisoning by toluylenediamine, which has moreover been confirmed by other investiga-

Schmiedeberg, Afanassiew, etc.). Anatomically, the lesions in the following order: dissolution of the blood corpuscles, degeneration of the liver without inflammation of the connec- ssue, acute parenchymatous nephritis.

first creating biliary fistulæ in dogs and then noting the es which occur in the secretion of the bile, the following facts been ascertained. In an initial period which begins two hours he administration of the poison, there is hypersecretion of the nore especially of the pigment (pleiochromia). In the second which begins fourteen hours after the poison has been injected, le is diminished in amount, and appears turbid, discolored, and ucous nature. The jaundice appears in the first period, attains ximum in the second, and then tends to diminish.

e course in phosphorus poisoning is almost the same, except period of two to three days intervenes between the first and the stages, during which the bile returns almost to normal, but it ndergoes the same alterations as those noticed in poisoning by nediamine, and icterus becomes gradually established. The le explanation of this fact is that it is not the phosphorus it- t one of its combination products formed in the intestines, alters the bile and destroys the hepatic cell. It is not only in ndice due to poisoning that the altered functioning powers of patic cells play the chief part, but also in those numerous net with in practice in which icterus appears from exaggerated olysis, and which for a long time were considered to be hæma- us. In grave infective conditions, in which there is always an us destruction of the red blood corpuscles, in marked impair- f nutrition, and in diseases of the blood itself we often find a r less icteric appearance, and the urine reacts unmistakably to s for bile pigment. We shall return later to the subject of the f jaundice formerly considered to be of hæmatic origin, in this ion desiring merely to call attention to the fact that they are of hepatic origin, due to the altered biliary function of the lls with the consequent inspissation of the bile. Numerous ients have been made for the purpose of verifying this nt, but on account of the purely practical character of this e must content ourselves with a mere mention of some of

jections of blood be made in the subcutaneous connective tissue g, bilirubin will be found in the circulation and in the urine , Hayem), but at the same time the bile will become inspis- ecause loaded with pigment, thus producing *pleiochromia*. obtained the same result by the injection of a large amount

of a solution of hæmoglobin into the intestines or serous cavities, and Stadelmann by injecting hæmoglobin directly into the veins.

Still more convincing, because more nearly approaching the pathogenic conditions of icterus from exaggerated hæmatolysis, are those which by various methods cause an artificial hæmoglobinæmia which in its turn produces icterus. Hayem for this purpose injected distilled water into the veins of a dog, Naunyn and Stadelmann used inhalations of hydrogen arsenide, and all obtained as a constant result hæmoglobinæmia, hæmoglobinuria, and icterus accompanied by inspissation of bile, so that there was a true *pigmentary polycholia*. Clinical medicine has also contributed to the understanding of these facts. In the course of severe paroxysmal hæmoglobinuria, more or less marked jaundice may occur in the period directly following the paroxysm (de Renzi).

It is evident, then, from what has been said, that there is a second series of cases of icterus, which must be considered as due to a functional alteration of the hepatic cell, resulting in the production of pigmentary polycholia, or pleiochromia.

In this series we must include another large group, first studied by Gubler, and by him called hæmaphæic icterus in accordance with a pathogenic theory of his own. When for any reason there is extensive destruction of red blood corpuscles, the liver becomes insufficient to the transformation of the resulting hæmoglobin into bilirubin, and the bile pigment therefore accumulates in the serum, and in the blood becomes transformed into pathological pigments to which Gubler gives the generic name of *hæmaphæin;* hæmaphæism therefore means the accumulation of hæmaphæin in the blood.

Dreyfus-Brisac, a pupil of Gubler, points out the two essential conditions to the production of hæmaphæism: (1) exaggerated destruction of red blood corpuscles (relative hepatic insufficiency); (2) functional changes in the liver (absolute hepatic insufficiency). Even in its symptomatology hæmaphæic icterus differs from other forms of jaundice in the following particulars: a pale-yellow coloration of the skin without any greenish tint, absence of cardiovascular symptoms, fæces at times only slightly discolored, at other times highly colored. This variety of icterus, according to Gubler, occurs in lead poisoning, alcoholism, febrile conditions, infective diseases, chronic affections of the liver, and in other no less important conditions; and a distinction may be made between *absolute hæmaphæism*, in which the liver has lost the power of transforming into biliary pigment the hæmoglobin derived from the destruction of the red blood corpuscles, and *relative hæmaphæism*, in which the primary lesion is in the blood, the liver remaining normal.

is ingenious theory of Gubler, although it has contributed
y to the progress made of late in the understanding of the origin
rus, is no longer tenable. In the first place hæmaphæin does
ist, at least it has never been isolated in the urine or in the
; in the second place, the blood pigment does not merit the
tance given to it by the theory of hæmaphæism, the chief rôle
ing to the liver cells, which alone are capable of transforming
ıy sort of pigment the hæmoglobin of the red blood corpuscles
have become old and incapacitated for their work.

e theory of hæmaphæic icterus was opposed by many pathol-
and clinicians, and received its death-blow when it was finally
strated that in these cases of jaundice a special pigment, uro-
vas to be found in the urine.

obilin ($C_{32}H_{40}N_4O_7$) was discovered in 1868, in fever patients,
é, who after many researches also found *chromogen* from which
actions of acids or even prolonged exposure to light urobilin
oduced. Fresh urine, giving no sign of the presence of this
ıt, soon developed it by mere exposure to the air for a few

'é's method is still the most reliable for the finding of urobilin
urine, and consists in the preparation of an alcoholic extract
is used in the chemical test with chloride of zinc, as well as
spectroscopic test, which Hayem has shown to be of such great
In the spectroscope, the acid alcoholic extract of urobilin gives
cteristic absorption band between the green and the blue, just
ı the lines *b* and *F*, nearer to *h*.

ıe urine contain normal biliary pigment this must be removed,
it interferes with the spectroscopic observation, and for this
e we may use Hoppe-Seyler's method, which consists in treat-
urine with milk of lime, passing carbonic acid gas through it,
n filtering; or Riva's, in which the urine is treated with the
solution of barium chloride, the filtrate then being used.

question of the origin of urobilin has been the subject of the
interest to urologists as well as to practitioners, and the
advanced have been many and various.

n the results obtained in their researches, Maly, Robin,
ski, Riva, and Reale hold that the urobilin may be entirely of
al origin, and derived from the bilirubin in the alimentary
der the special influence of the nascent sulphuretted hydrogen,
s almost always formed during the fermentation (especially
vric) of food-stuffs.

cond theory is the hæmatic, according to which hæmoglobin
formed directly into urobilin, the demonstration of this being

found in the fact that in extravasations of blood this transformation may occur *in loco*.

Pathologists are at the present time divided between two theories as to the pathogenesis of urobilin, namely, the *pigmentary* and the *hepatic* theories.

The first has been chiefly upheld by Quincke, Kiener, Engel, Mya, and others, and it is believed to apply principally to the urobilin which precedes and follows absorption icterus. According to these authorities the urobilin is produced by transformation of the bilirubin, brought about by the direct reducing action of the tissues in which it is deposited, especially the renal epithelium. According to this theory, when the bile pigment poured into the blood is of small amount, as in the beginning and at the end of stasis icterus, it is all transformed by the renal epithelium into urobilin, and appears in this form in the urine; but when it is in large amount, as at the height of stasis icterus, it is eliminated unchanged, the renal epithelium not being adequate to transforming it all. Urobilinuria therefore would be the urological expression of a light degree of icterus.

The hepatic theory, established mainly by the investigations of Hayem, is the one accepted by the majority of practitioners and pathologists at the present time. According to this view, urobilin is always derived from the hepatic cell. Under normal conditions hæmoglobin gives rise to bilirubin, but when it is in a state of insufficiency it gives origin to urobilin. The product therefore is the expression of impaired secretory power of the liver, which may, however, be merely relative. When there is an excess of coloring matter from the blood to be transformed by the liver into bile pigment, the hepatic cell may be unequal to the increased work, and will then give origin not only to the normal pigment, but to the abnormal pigment, urobilin, as well.

The question cannot be said to be settled beyond dispute, for many contradictory and obscure facts are met with in practice. It seems to us that no one theory can be found to give a satisfactory explanation of these clinical occurrences. Leaving theoretical discussions to others, we will merely record a few facts which are indisputable, and which will aid in interpreting the phenomenon.

1. At the beginning and end of stasis icterus we may find urobilin in the urine, while bilirubin is found in the blood serum. 2. Urobilinuria appears when there is extensive destruction of the red blood corpuscles (paroxysmal hæmoglobinuria, toxic hæmobilinuria) as especially demonstrated by de Renzi and Reale. 3. Urobilinuria occurs in diseases of the liver in which there is a more or less severe structural or functional lesion of the hepatic cells.

ıe first of these facts shows that the origin of urobilin might be ıated to the reducing action of the renal epithelium. The others, ˈer, clearly prove that urobilin may be of hepatic origin and ıent insufficiency on the part of the cells.

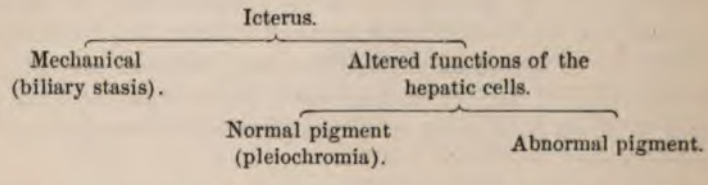
ɜ do not wish to reject altogether the intestinal theory. It is probable that this would explain the fact, demonstrated by that urobilin or its chromogen exists normally in the urine. æmatic theory, however, cannot be entertained. Even if true robilin may be found in old subcutaneous hemorrhagic foci, ot conceivable that hæmoglobin should be directly transformed robilin in the circulating blood. According to some patholo- other modified pigments, as yet but little known, may be found blood and the urine of icteric patients. The most important se is the brownish-red pigment isolated by Winter, which comes reduction of bilirubin. Tessier calls it *bilirubidin*, and it is ıneously formed in a solution of bilirubin or of urobilin by t with the air. This pigment would seem to be capable of ting a yellow color to the tissues, but further study is neces- efore its properties are thoroughly understood.

ving shown the physiopathological significance of urobilin, ointed out the possible presence of bilirubidin, there still re- to us to ascertain what relations exist between the forms of ɜ with normal biliary pigment and those with modified pig- and if there is any form of jaundice which can properly be *urobilinous*.

ɔm the standpoint of hæmatic and urinary conditions we may guish many forms of icterus. It often occurs that during the of jaundice bilirubin may be found in the blood serum and in ine with or without urobilin. This occurs in icterus by ab- ɔn. Or it may happen that the serum contains normal biliary nt and traces of urobilin, the urine containing urobilin only. ally this form of icterus is light, and the fæces are not at all y slightly discolored. In a third series of cases of icterus, in and bilirubin are found in nearly equal amounts in the while in the urine urobilin alone is present. Finally, it may n that the serum and urine both contain only urobilin, but case the color of the skin is unchanged, because the urobilin ses too weak a coloring power to affect the tissues (Kiener ngel) and is at the same time so diffusible as to be rapidly ated by the kidneys. Thus there may be abundant and pro- urobilinuria without the slightest trace of icterus, and this ex- the fact that in cases of jaundice when only urobilin is found in ne, normal or modified bile pigments are present in the serum.

From the foregoing statements we may conclude: 1. That the presence of urobilin in the urine indicates hepatic insufficiency, or the presence in the blood of a small amount of bilirubin which is reduced to urobilin by the renal epithelium; 2. That there is no such thing as urobilinic icterus, in the sense that the presence of urobilin in the blood is the cause of the yellow coloration of the skin; 3. That there are forms of jaundice due to the presence of modified pigments in the blood.

Having synthetically described all that is known in regard to the pathogenesis of icterus, avoiding useless theoretical discussions, and having endeavored to give a clear and exact idea based upon the latest experimental and clinical discoveries which might be of real practical value to practitioners, we now proceed to classify the various forms of icterus according to their pathogenesis into two chief groups, namely, mechanical icterus (from stasis) and icterus due to altered functional power of the hepatic cells, the latter being further subdivided into icterus with normal pigment (pigmentary polycholia or pleiochromia) and icterus with abnormal pigment, as shown in the following diagram:



This new classification of icterus which we advance seems to us to be in harmony with what is positively known about the origin of the disease.

A few words remain to be said in regard to icterus of hæmatic origin, a large group of cases which, recognized until recently by all pathologists, is perhaps even yet believed in by some, although from our standpoint it belongs only to medical history.

Until a few years ago, as may be easily seen in all works on pathology not of recent date, cases of icterus were divided into hepatogenous and hæmatogenous. By the former was meant those due to intrahepatic reabsorption of the bile from some restriction in the bile passages. In the second class were included all cases of jaundice due to direct transformation of hæmoglobin into bilirubin within the circulating blood, exclusive of any action whatsoever on the part of the hepatic cells. Types of this group were found in the icterus caused by toxic agents, by infective processes, or by grave dyscrasiæ; in short, all cases of icterus produced by extensive destruction of

ood corpuscles from whatsoever reason were considered to be
natic origin.

e most ardent advocate of this theory was Leyden, who even
ata for the differential diagnosis between the two forms, which
repeat in order that we may see whether they are of sufficient
o sustain this theory of the pathogenesis of icterus, so com-
opposed to all that we have previously said in regard to it.

den's criteria are the following: 1. In hepatogenous icterus
low color of the skin is in relation with the amount of pigment
ted by the urine, while in the hæmatogenous it is relatively
than the degree of coloration of the urine. 2. In hæmatog-
cterus we have symptoms denoting a dissolution of the blood
s and interstitial hemorrhages, cardiac weakness, loss of
h, delirium, stupor, coma, etc.) which are entirely absent in he-
ous icterus. 3. In hepatogenous icterus a microscopical ex-
ion of the liver will reveal important lesions of the organ, espe-
hose due to obstructed flow of the bile, whereas in icterus of
c origin there are no indications whatsoever of any involve-
the liver. 4. The contents of the intestines are decolorized in
of hepatic origin, or, in other words, there is true intestinal
, while in hæmatogenous icterus the fæces are not only not
ized, but are often more higly colored than normal. 5. In
genous icterus the grave alterations of the red blood cor-
frequently cause fatty degeneration of the liver cells, the
pithelium, and the cardiac muscle, conditions which are en-
bsent in hepatogenous icterus. 6. Finally (Leyden lays espe-
ess upon this fact), in icterus of hepatic origin the biliary
e found in the urine, but in icterus of hæmatic origin they are
because they are formed only by the action of the hepatic

irst sight these differential criteria would seem to be clear
lisputable and convincing as to the two classes of jaundice.
the undoubted weight of the eminent pathologist's opinion
at first, and still seems to many, to be a sufficient guarantee
accuracy. If, however, in a candid and unprejudiced spirit
ll the clinical and experimental facts already given in this
, we shall see that this differential diagnosis is contradicted
liscoveries of physiopathology.

first argument is, in fact, anything but absolute, for it is not
to define the exact amount of biliary pigment eliminated by
ne which should correspond to a given degree of jaundice.
lculations must vary too much with the differing conditions
ividuality to be taken as a guide to the differential diagnosis.

Moreover, whatever the origin of the icterus, the condition of the kidneys will explain the fact that bilirubin is present in the blood by reason of its diminished elimination in the urine. Even in jaundice of purely mechanical origin, as from some structural or functional lesion of the kidneys, we may find a scanty elimination of bile pigment in the urine with an intense yellow coloration of the skin, and cannot from this fact infer that the icterus is of hæmatic origin.

Leyden's second argument is refuted by the result of all the researches made in regard to icterus due to poisoning which, as we have already stated, originate from the qualitatively altered conditions of the bile (pigmentary polycholia, or pleiochromia). In these cases, even should the icterus be of hepatic origin, there is marked dissolution of the blood, due to toxic agents, which is the true cause of the chemical alteration of the bile. Moreover, when grave hepatic lesions (biliary cirrhosis) produce an intense cholæmia, we may have the occurrence of secondary conditions which attest to the alteration of the blood, probably determined by an accumulation of pigments and bile acids in the circulation, or by the hepatic insufficiency itself; so that neither in this class of cases can the jaundice be considered to be of hæmatic origin.

Nor can we attach much importance to the hepatic lesions found post mortem as a means of differentiating between hepatogenous and hæmatogenous icterus. It has in fact been demonstrated, especially by the experiments of Stadelmann given above, and by those of de Luca, that in icterus from poisoning (toluylenediamine, phosphorus), which may be taken as the type of hæmatogenous icterus, there are structural and functional alterations of the hepatic cells followed by chemical changes in the biliary secretion. The argument relating to the coloration of the fæces had weight until the mechanism of production of some forms of jaundice was fully understood. It has been demonstrated that icterus is often determined by inspissation of the bile with relative increase of pigment, so that the bile, by reason of its increased density, is absorbed by the lymphatics, and, the bile ducts being pervious, it flows freely into the intestines, and because of the greater amount of pigment contained stains the fæces a darker color. This occurrence may serve to distinguish between jaundice of mechanical origin and that derived from functional alterations of the hepatic cells, but will not sustain Leyden's theory.

Nor should we attach importance to fatty degeneration of the liver and kidney cells and heart muscle, said to occur only in icterus derived from dissolution of the blood. Icterus from poisoning by toluylenediamine and phosphorus, which, as shown by experiments cited above, is of hepatic origin, demonstrates the fallacy of this

As to the importance of the presence of bile acids in the urine, n this can be taken as an absolute criterion. In small amount of demonstration, these acids often exist in the urine, as y Stadelmann, an authority upon the subject. It is certainly understand that the acids may by the oxidation processes to hey are certain to be subjected in the circulating blood, be so l in their chemical composition as not to be met with in the Moreover, they have occasionally been met with in the urine nts suffering from icterus which was of hæmatogenous origin. himself found mere traces of them in the urine of a patient with croupous pneumonia, in whom, as the disease was of e origin, the icterus would, according to this theory, be of e origin.

alled hæmatogenous icterus is to be explained in an entirely t fashion. When for any reason exaggerated hæmatolysis is d, a larger amount than usual of raw material is brought to to be transformed into bile; the hepatic cell is overworked, becomes inspissated from excess of pigment (pleiochromia), increased density produces a retention of bile in the biliary s, causing reabsorption by the lymphatics and its consequent into the blood current.

nay finally mention a few of the terms which have been pro- or the distinction of the variety of icterus due to grave dis- s of the blood.

assiew suggests the name of *hæmohepatogenous*, wishing thus ate that although of hepatic origin its essential condition is the altered state of the blood. But, as Quincke observes, d contains an unpardonable pleonasm. We know that bili- derived from the hæmoglobin of the blood and that it cannot y other origin; therefore, having once admitted that this nation can only occur in the liver, it is needless to change e *hepatogenous* into *hæmohepatogenous*. Neither is the name l by Quincke, *anhepatogenous*, even admitting that bilirubin rmed from a hæmoglobin in other tissues than those of the yond criticism. For admitting this to be true (but it is), it is formed in such small proportions, is so slowly absorbed, quickly eliminated by the kidneys (Stadelmann), that it could cumulate in the blood in sufficient amount to cause icterus.

ly, Liebermeister proposes that we consider hæmatogenous as dependent upon the fact that while many hepatic cells ain capable of performing their biliary function, a large is unable to prevent the diffusion of the bile (contained in in other cells) into the blood and the lymph, and to this

form of jaundice he would give the name of *acathectic*. But this view of Liebermeister's is mere hypothesis, unsustained by any clinical or experimental facts.

We may, therefore, consider it to be an established fact that jaundice is always of hepatic origin, and that in the present condition of our knowledge it is never possible to admit the existence of an icterus of hæmatic derivation. As biligenesis represents the normal function of the liver, so icterus stands for its pathological functioning.

GENERAL THERAPY OF HEPATIC DISEASES.

In the course of our studies upon the general pathology of the liver, we have taken up analytically the various functional disturbances which occur in hepatic disease, disturbances not of the liver alone, directly dependent upon an anatomico-pathological lesion, but more or less complex functional alterations of other organs and apparatus connected with the functional condition of the hepatic gland. Owing to the close connection between the various organic functions, which is especially evident in hepatic diseases, treatment should be addressed not only to the liver but to other organs as well. It should endeavor at the same time to compensate the impaired functioning powers of the liver cells, and other more or less impaired functions due to the disease. This is the more necessary because complications of other organs following hepatic disorders sometimes overshadow the symptoms of the original disease, and by aggravating it become transformed from effects into causes of the affection.

Physiological Therapy.

In diseases of the liver, even more than in those of the stomach and intestines, physiological treatment is of the first importance, being indeed more absolutely essential to success than any form of pharmaceutical treatment. In the vast and ever-increasing catalogue of drugs, we find no chemical product capable of directly influencing the hepatic cell and of restoring its normal nutritive or functional condition. But with the knowledge which we possess of the various functions of the organ and the conditions in which these may be altered, and of the physiopathological laws which govern its deviation from the normal type, we can by rationally taking advantage of these conditions cause a more or less marked modification in the functioning of the organ, and thus indirectly accomplish the more important object of restoring the hepatic cell to its normal condition. Physio-

therapy is the treatment *par excellence* to be adopted in he-
lisorders, and by this term we mean a treatment consisting of
tely physiological measures, with the direct object of modify-
functional conditions of the hepatic cell.
ere are two main factors which regulate the functioning of the
the quality of the food, and mental influences. Of these the first
more important, and the one to be borne in mind by the practi-
n the treatment of the disease. The catarrhal hyperæmia which
bly occurs during the period of digestion, the hyperbilifica-
nich accompanies the process of chylification, the hyperfunc-
of the hepatic cells following the absorption of the digestive
ts, which is directed to the modification of the albumino-
es, to the formation of glycogen, and to the protection of the
m from noxious products formed in the complicated proc-
f intestinal chemism, are all facts which explain the impor-
f a proper alimentation in the restoration of the normal he-
nctioning powers. The second, recognized since the earliest
medicine as a factor in the causation of diseases of the liver,
tantly assuming a more and more important place in the
enesis of the disease, and is explained by the effect of nervous
e upon the circulation and functioning powers of the hepatic

cute diseases of the liver, the process being an inflammatory
nilk diet and absolute rest in bed form a method of treatment
is usually carried out without objection on the part of the pa-
The greatest difficulty experienced is in chronic diseases (pa-
natous or interstitial hepatitis, etc.), and yet the *sine qua non* of
is an absolute milk diet, above all of ass's milk, which is the
rest in fats. This diet, which is the basis of all treatment of
sease, was recognized and appreciated by the ancients, but its
eat value has been insisted upon especially among modern
by one of us (Semmola).
influence of a milk diet upon hepatic disease is varied and
ated, but consists chiefly and finally in absolute rest of the
Milk is the one food which does not produce any irritation of
stive tract, and which therefore reduces to a minimum the flow
to the intestinal walls and with it the digestive hyperæmia of
r, since the portal blood is merely blood carried from the in-
. The diet has a direct effect as well upon the circulation
n the liver function, by causing a lowered functional activity
epatic cell. The proximate principles in milk are more fully
ed than in other foods, and require less chemicobiological
to so change them that the nitrogenous molecule derived

from gastric and intestinal digestion can be assimilated and undergo the changes necessary for its reduction to urea, the increased production of which, under the influence of a milk diet in chronic morbid processes of the liver, was first demonstrated by us, and must be regarded as the best proof of the efficacy of the treatment. The greater facility with which this nitrogenous molecule, derived from the digestion of milk, is transformed into urea certainly demonstrates the beneficial influence of the diet in hepatic diseases by sparing the urea-producing function of the liver.

Nor should it be forgotten that the products of the digestion of milk possess no irritating properties, and that, therefore, when they are absorbed into the portal system they exert no injurious local action upon the parenchyma of the liver, and thus tend to decrease the active hyperæmia of the organ. The production of bile is of course reduced by the diminution of the chemical processes of digestion and by the lessened difficulty of absorption.

Finally, a milk diet constitutes the best method of intestinal disinfection. By it we obtain a reduction, if not the disappearance, of the abnormal fermentations which so frequently arise in the intestines of patients suffering from liver troubles, the products of which when carried into the portal system induce a state of overactivity in the liver, one of whose functions is the protection of the organism from intoxication of intestinal origin. All these effects of a milk diet enable us to understand how it assures a condition of rest to the liver; this rest being, as a rule, the most important therapeutic compensation for the disordered state of the organ. It is worthy of note that however little importance some may wish to attach to the influence exerted by the *febris digestiva*, it certainly does constitute a daily recurring obstacle to the cure of chronic inflammatory processes by the stimulation of constantly renewed causes of irritation and of functional intermittent congestion.

Not only does a milk diet secure rest to the liver, but it also compensates for the structural and functional disturbances of the digestive system which follow hepatic lesions. Stasis catarrh, which we have already mentioned as a constant result of affections of the liver dependent upon chronic interstitial processes, finds its chief compensation in a milk diet by a mechanism so simple and so direct that we shall not stop to analyze it. Moreover, the formation, under a milk diet, of an albuminopeptone of easy transformation into serum albumin, which is carried into the intraorganic circulation, enables us to understand the fact that in a milk diet nutrition is more easily accomplished and the evil consequences of a hepatic lesion to the general condition of the patient are averted.

he lessened production of toxic substances in the intestine pre- overactivity of the urinary apparatus, the integrity of which is est guaranty of a cure in diseases of the liver. We see, there- that a milk diet is the best physiological compensatory agent patopathies, because of the rest which it gives to the liver, and medial nature of its influence upon the disturbance of the func- of other organs following lesions of the liver.

to mental influences, these are, as we stated above, a necessary in the cure of chronic affections of the liver, disturbing emo- acting powerfully by influences as yet unknown upon vaso- innervation, and the active hyperæmia which they induce being stant obstacle to the restoration of the capillary circulation in ver, with which, in some way or another, the chronic irrita- ocesses are connected. To place the patient amid peaceful sur- ings, away from all cares, to avoid all causes of emotion, and to withdraw him from ordinary life in order to secure a serene iet existence, unmarked by great pleasures or disquieting oc- ces are the conditions essential to a cure which as yet have not d the attention they merit from the medical profssion.

ese two physiological therapeutic measures are so all-important uently to bring about a cure of rebellious cases of chronic he- , which remain uninfluenced by pharmaceutical treatment. It firm conviction that without them all remedial measures will ıd to be irrational and useless.

hird therapeutic condition of importance in chronic morbid ses of the liver concerns the relation between the visceral and ripheral circulation, the treatment of these affections being ıpon the restoration of a disturbed hepatic capillary circula- Whatever interferes with this equilibrium retards or even pre- he cure of chronic liver diseases. So that what we stated above rd to digestion and to mental influences we repeat here in re- the influence of the cutaneous capillary circulation; when this ılated to its utmost degree of activity, the effect, on account of stant and inverse relation existing between the external and l circulation of the organism, will be to diminish the conges- ıdition of the parenchyma and to favor the rapid cure of sub- chronic processes of the liver. Hydrotherapy alone is ca- f inducing this peripheric stimulation, and for this reason we t the nature of the diet, mental influences, and hydrotherapy ıte the fundamental triad of therapeutic measures to which the ın can resort with full confidence of success.

ortunately, hydrotherapy in its true sense is not always easy ication, because the condition of the patient's muscles and

his debility as a consequence of the chronic liver disease are such that general hydrotherapy cannot be resorted to. This does not, however, interfere with the application of local hydrotherapy, by means of which we may without fatigue to the patient stimulate the peripheric capillary circulation, and this measure should always, without exception, be adopted. It should be given in the form of Priessnitz's pack applied daily or twice a day around the abdomen, and left on a variable length of time, according to the reaction induced.

It is evident that great importance should be attached to the restoration of the circulation of the liver in the treatment of hepatic disorders, this importance being the logical result of the enormous blood supply of the organ. Abdominal conditions which might influence the hepatic circulation must be carefully noted. One of these is the action of the bowels, for habitual constipation by favoring abdominal stasis contributes largely to the retardation and sometimes prevents the cure of chronic irritative processes in the liver. For this reason, although in the large majority of cases physiological therapy is all that is necessary for a cure, drugs have sometimes to be resorted to in order to overcome constipation.

Another condition which tends to the development and continuance of chronic irritative processes of the liver is a certain organic disposition, considered of great importance by the ancients, and which, after a long period of oblivion, has been restored to a position of consequence. We refer to the *venous temperament*, better known under the name of "*abdominal phlebosis*," which was mistakenly believed to be due only to a sedentary life, to constipation, etc. Abdominal phlebosis is a congenital condition, and consists in a tendency to a predominance of the venous system, or, if the term be preferred, it may be called an hepatic temperament, from the point of view that considers the liver as the most important centre of the abdominal venous circulation. For this reason a fourth therapeutic indication in chronic inflammatory processes of the liver consists in methodical muscular exercise, for, if sedentary and indolent life is capable of causing abdominal stasis in persons of any temperament, so much the more must it occasion and aggravate the condition in those of a "venous" temperament suffering from hepatic disease. We have said enough to demonstrate the importance in affections of the liver of a rational physiological therapy, without which no other treatment can succeed, and which in many cases is of itself sufficient to bring about the desired result.

But, although the chief element consists in a milk diet, we must not neglect such pharmaceutical or hygienic measures as may modify

come the cause of the disease, and influence the structural or
nal condition of the hepatic cells.

Etiological Treatment.

nany cases of hepatic disease, etiological treatment will de-
he first place, because by removing the cause it may prevent
ct. Antisyphilitic treatment by means of mercury and the al-
odides in hepatic syphilis, cinchona and quinine in malarial
e our chief resources in this form of treatment, and will be
) give good results when appropriately employed.
1 these exceptions we possess no true specifics, although it is to
d that with the progress of therapeutic knowledge some will
d. Treatment, as a rule, will have to be addressed to the
process itself.

Antiphlogistic Treatment.

great number of hepatic diseases there is present a constant
n which, although it is not the fundamental lesion, serves to
te it and to complicate the symptomatology; this is hy-
1.
ute exacerbations occurring in the course of chronic interstitial
ations, in infective processes of the liver around new growths,
eased flow of blood to the hepatic gland determines an increase
norbid phenomena, since even in the case of circumscribed
of inflammation the vascular lesions extend throughout the
nd thus accentuate those functional hepatic disturbances which
ave been compensated for by the portions of parenchyma which
l unaffected, had it not been for the occurrence of circulatory
Moreover, hyperæmia is one of the conditions most essen-
flammation, for, if it do not constitute the cause, it is at least
constant and the primary lesion in all inflammatory proc-
For this reason antiphlogistic measures are the first to be
in the most diverse hepatic affections, and this not so much
to affect the inflammatory reactions of the parenchyma as in
y to compensate for the hyperæmic condition of the organ.
older physicians had more confidence in the antiphlogistic
t of hepatic diseases than we in the present state of our
ge regarding inflammation are able to entertain. Hot plain
rd poultices over the region of the liver, vesicants, painting
line, cauterization, leeches, wet-cups, all methods of induc-
taneous hyperæmia or inflammation in order to compensate

for the hyperæmic or inflammatory condition of the liver, were held in great repute, so that it was considered high treason to science to omit the prescribing of them as a matter of routine practice. But when by means of important anatomical and experimental researches in regard to the pathogenesis of inflammation it was found that this consisted essentially in the reaction of the tissue to some phlogistic agent, and that it was not possible to cause a disappearance of the effects without overcoming the cause, faith in antiphlogistic measures gradually became weakened, and at the present day has been almost entirely abandoned. In the cases under discussion, we have the less reason for belief in this form of treatment, because we know that there are no direct circulatory relations between the hepatic gland and the abdominal wall upon which the derivatives are applied, so that these so-called antiphlogistic measures are of no use even in lessening congestion, to say nothing of reducing inflammation.

The antiseptic and aseptic methods of surgery have emboldened practitioners to resort to heroic measures. Thus, in order directly to produce diminution of the hyperæmia of the parenchyma of the liver, O'Leary resorted to bleeding of the organ by puncturing it with a small trocar or aspirator, and by the aspiration of 8 to 10 gm. (2 to 2½ drachms) of blood. This procedure he especially recommends in the idiopathic forms of hyperæmia. If this delicate operation be undertaken with due regard to all aseptic precautions, it will probably have no untoward consequences, and yet it seems to us a method to be absolutely avoided, both because it causes traumatism, which cannot but be the cause of irritation and increased hyperæmia of the liver, and because we possess other means for the diminution of the flow of blood to the liver, which can be applied with less difficulty and with more hope of success. Moreover, although our experience in this class of operations is limited, since our faith in it is small, we cannot resist the conviction that, especially when it is used in cases of severe active hyperæmia of the organ, there is the possibility of serious and immediate consequences, more particularly of intraperitoneal hemorrhage, since the abundance of the blood supply, the dilatation of the vessels, and the distention of the capsule greatly diminish the chances of the prompt arrest of the bleeding by a thrombus.

The most efficacious and absolutely innocuous method of lessening hepatic congestion at our disposal are anal bleeding and intestinal derivation. The relation which exists between the inferior hemorrhoidal veins and the portal system permits of our depleting the latter by applying leeches to the anal orifice. This procedure meets with great success in many cases of hepatic affection, especially in

hyperæmias. But it is not a form of treatment which can be
d daily, since it would induce a condition of general anæmia,
must be avoided. We must, therefore, resort to intestinal
tion by means of saline purgatives, which have a true dialytic
and, by producing a marked serous transudation into the
of the intestines from the small venous radicles, cause a de-
of the portal system, and thus diminish the flow of blood
liver.

se therapeutic measures are of the utmost importance in the
inflammatory processes in the liver, especially in circulatory
rs, in which by reducing the hyperæmia of the organ they in
ure cut off one of the essential conditions of inflammation.

mel has been recommended as an antiphlogistic. Trousseau
at this and all mercurials had a beneficial action upon inflam-
processes of all parenchymatous organs, by reason of their
eristic alterative action. With all respect to the renowned
clinician, we do not believe that his opinion can be sustained
present day on account of our modern views in regard to in-
tion. The endeavor has been made to explain the action of
reparations by their cholagogue properties (which have not,
, yet been demonstrated by pharmaceutical researches, as we
e later), an action which by improving the biliary circulation
osed beneficially to influence the hepatic circulation and the
ing powers of the cells. Harley, when this cholagogue action
longer be maintained, endeavored to modernize Trousseau's
sis by imputing to calomel an antiphlogistic action upon the
es of the liver, modifying or abolishing vascular congestion,
s diminishing the mechanical pressure upon the hepatic cells
revented them from secreting bile.

ever that may be, it is certain that in chronic inflammations,
primary or secondary hyperæmic conditions of the liver,
produces a good effect and is perfectly reliable. It is our
owever, that it acts chiefly as an intestinal derivative, and to
tent as a stimulant to the secretion of bile, by increasing the
l peristalsis which facilitates the flow of bile. Its antifer-
e and antiseptic action must not be overlooked, but of that
speak later.

best to administer calomel in small divided doses, and every
ys to suspend the treatment for two days, in order to avoid
lization. Powders of 3 to 4 cgm. ($\frac{1}{2}$ to $\frac{2}{3}$ gr.) each are to be
ee or four times in the course of the day, the patient being
watched to see that mercurial gingivitis does not occur.

Resolvent Treatment.

In the chronic forms of the disease, resolvent treatment, by which we mean iodine treatment, is of great importance. Although its use had been previously known in hepatic diseases, we were the first to demonstrate its great efficacy, to give the interpretation of the mechanism of its therapeutic action, and by clinical statistics to prove that cases of chronic interstitial hepatitis could be cured by a milk diet and iodine treatment.

Iodine, absorbed as an alkaline iodide, and in smaller amount as an iodate, acts principally by stimulating tissue changes, and in this way affects chronic inflammatory processes of the parenchymatous glands, more especially the liver. By stimulating the mechanism of the processes of nutrition and disassimilation and increasing cellular oxidation, newly formed tissue, which, especially when in the stage of embryonal proliferation, represents a pathological tissue and requires a greater amount of nourishment, is absorbed and finally disappears. This special action is more marked in the lymphatic glands and in the liver, because in these organs the absorbed iodide is deposited and sets at liberty the iodine, which in a nascent state, according to Binz, exercises its oxidizing powers.

Thus interpreted, the beneficial action of iodine and iodides in chronic inflammatory processes of the liver can be counted upon to give the best results. The practitioner must not be content with the administration of small doses, but must give large doses of the iodides (4 to 5 gm.=60 to 75 gr.) a day, according to individual susceptibilities.

The chloride of ammonium has been recommended for chronic congestions of the liver, by the English physicians especially. According to Harley, it acts as a stimulant to the nervous and the circulatory systems, but this action is not sufficient to account for its effects in hepatic affections.

Cholagogues.

From the descriptions given of the action of certain pharmaceutical agents, and especially of the coloration of the fæces after their use, the idea was suggested that cholagogues might excite the liver cells to a hypersecretion of bile, and by this increase of functional power produce an improvement in all the other functions of the cells. It was thought that a certain compensation would thus be afforded for hepatic insufficiency, whether due to structural lesions or to functional disturbances.

ut, as we may easily see, this therapeutic indication was not a
al one, it was not in harmony with our present knowledge of
mal functional or structural processes of the organs and tissues.
if we admit the stimulating action of certain drugs upon the
ells, which is far from the truth of the case, it is very certain
this would not cause a restoration of function. All forms of
lation are combined with hyperæmia from increased work, and
bute to the increase of the irritative or inflammatory lesions
ich they are supposed by this theory to compensate. For ex-
, it is what occurs when we administer epithelial diuretics in
itis; the inflammatory condition of the epithelium is aggra-
and the result is the very opposite to that desired. Nor
there is extensive anatomical destruction of the hepatic paren-
is much reliance to be placed upon cholagogues, which may
pon the normal cell, but not upon one which is altered or
yed.

e have already seen under the head of physiological treatment
he most important rational indication is to secure as much rest
liver as is compatible with life, while the use of cholagogues
lead directly to the opposite extreme, that is to say, to over-
which is absolutely to be avoided.

oreover, the cholagogue power of substances previously consid-
o possess it is now far from being accepted by all investigators.
ld be beyond our scope to quote at length the experimental
ches conducted by Handfield Jones, Röhring, Rutherford,
l, Gamgee, Bennet, Baldi, Gueneau de Mussy, etc. We will
state that the cholagogues which have been most highly rec-
ded are calomel, rhubarb, podophyllum, senna, aloes, and
kaline salicylates. The mode of action is not interpreted in
me way by all; some hold that there is an increased excretion
ot an increased secretion, others believe that there is a real
ation of the hepatic cell. The increased excretion is supposed
e to be due to the action of the drug upon the muscular fibre
f the large bile ducts, by others to the increased peristalsis
they cause, being nearly all purgative in their action; the in-
of the cellular function is said by some to depend upon a
al hyperæmia of the gland, and by others to be due to an
e preference for the cell. But even if the laboratory does not
onstant results, clinical experience teaches us that certain
do possess an undeniable cholagogue action, which is probably
tion to increased excretion.

do not believe, however, that cholagogues are efficacious in
e diseases, with the exception that in cases of a removable

obstruction of the large bile ducts they may by increasing the bile pressure overcome the obstacle.

Alkalies have a well-deserved reputation in the treatment of hepatic disorders, not because of a purgative or cholagogue action, but because they act upon the hepatic cell, upon the circulation of the liver, and the composition of the bile in such a way as to restore the normal functional powers to the hepatic gland. Martin-Damourette and Hyades have demonstrated the elective action of alkalies upon the liver functions, having noted the increased elimination of urea which follows their administration, urea, as we know, being chiefly formed in the liver.

However this may be, the alkalies form an important part of the treatment in hepatopathies, not only when given internally, but also when used in thermal baths, as at Vichy, Carlsbad, Montecatini, Castellamare, and Ischia, near Naples. It is indisputable that in thermal "cures" we not only have the beneficial action of the alkalies taken internally and absorbed by the skin, but there are present other conditions favorable to the cure or the improvement of the hepatic disorder, either by improving nutrition and hæmatopoiesis, or by a beneficial action upon the renal function.

Treatment of Gastroenteric and Peritoneal Disorders.

We have already learned how close is the bond between the gastrointestinal function and structural or functional disorders of the liver. It is therefore of the utmost importance that gastric and intestinal affections due to hepatic disease should receive appropriate treatment, since they react injuriously upon the condition of the liver, considerably aggravating existing disease.

Constipation due to muscular inaction of the gastroenteric tract, and which is a decided obstruction to the portal circulation, merits our chief attention. To overcome this condition, so frequent in hepatic disorders, it is not enough to regulate the diet—indeed the milk diet so necessary to the treatment of disease of the liver, when well supported, increases the constipation by the formation of fæces of small volume and unirritating to the intestinal mucous membrane; neither are the ordinary methods resorted to in these cases sufficient (cold water in the morning on an empty stomach, two or three spoonfuls of olive oil, of linseed, preserved fruit, etc.), but measures better calculated to accomplish the desired purpose must be adopted. The best of all measures is the daily morning use of enemata of not more than half a litre (one pint) of cold water, to be retained for at least half an hour, for the purpose of softening the

ccumulated in the rectum. In some cases this treatment is ly useless, because the accumulation of excrementitious mat- rs in the colon instead of in the rectum. In such an event we ave to resort to purgatives, which are also of benefit in case ites is present. The salines and the drastic purgatives are t in use in cases of hepatic disorder, because they expel the and at the same time cause a serous transudation which o deobstruct the portal circulation and to cause reabsorp- any peritoneal transudation. Should there, however, be no symptoms of stasis of the portal system, the myokinetic pur- such as rhubarb and senna, are to be preferred, the long- ed daily use of salines and drastics having the effect of in- ; constipation and aggravating the stasis catarrh of the ntestinal mucosa.

rapeutic intervention is also demanded by disorders of the n, which often take the most prominent place in the symp- ogy of liver disease, and aggravate the pathological condition. k of appetite, the lessened activity, and the altered chemism of nach should therefore be symptomatically and carefully treated use of bitters, tonics, preparations of strychnine, acids (hy- ric, lactic), digestive ferments (pepsin hydrochlorate), and according to the indication.

following has often given us the best results, and is of value treatment of constipation as well:

℞ Neutral sulphate of strychnine, . . 0.05 (gr. i.)
Tincture of rhubarb, of calamus, of ginger, and of
cascarilla, ... of each, 40.0 (℥ x.)
S. Forty drops in half a cup of cold water one hour before meals.

stinal meteorism and diarrhœa should receive special atten- the treatment of hepatic affections; for the first we should be aromatic waters possessing absorbent powers (anise water, vater, etc.), for the second astringents, and we recommend the ng:

℞ Dover's powder, ... 0.4 (gr. vi.)
Powdered alum, ... 0.6 (gr. ix.)
Bismuth subnitrate,
Calcium carbonate, ... āā 1.0 (gr. xv.)
M. et ft. pulv. No. iv. S. One to be taken every hour.

have already studied the effect of infective processes and of oxication of intestinal origin upon the functions and the gy of the hepatic cell, and it is evident that a rational therapy ases of the liver will seek to limit or to prevent the fermenta-

tive processes which abnormally occur in the chemism of digestion, and to create conditions unfavorable to the life of the pathogenic micro-organisms which find in passive congestion of the mucous membrane the most favorable conditions for their development.

Although these forms of autointoxication and intestinal infection may occur without occasioning symptoms of importance when the liver is in a normal state and able to exercise its protecting influence, in the case of structural or functional insufficiency of the cells the slightest accumulation of intestinal toxins, or the slightest interference with the eliminative function of the kidneys may be the starting-point of serious and even violent phenomena.

Intestinal antisepsis is almost obligatory, especially in certain cases of hepatic disorders. For this purpose Bouchard has proposed the use of insoluble antiseptics, such as naphthalin, naphthol, betol, salol, etc., given in small and repeated doses; these remedies have had and still have the confidence of many physicians for their antiseptic and antifermentative action in the stomach and intestines. Very recently, however, in the Therapeutical Society of Paris a long and learned discussion took place as to the use and usefulness of these remedies, and Bardet and Huchard in especial maintained that they were useless and often harmful—useless because the insoluble antiseptics when they reach the intestines are included in the fæces and thus eliminated, injurious because they often give rise to symptoms of poisoning. The best intestinal antiseptic, because it improves the digestion, benefits the catarrhal condition of the intestines, creates conditions unfavorable to the development of the microbes, and limits abnormal fermentations, is a milk diet. This view, sustained by Semmola, has been further confirmed by Charrin and Roger, who demonstrated that with this *régime* the urotoxic coefficient was diminished. In addition to this form of treatment we may use the hyposulphites of sodium or of magnesium, which, as Semmola[40] demonstrated as early as 1864, reduce abnormal fermentations in the intestine. They should be prescribed in doses of from 8 to 10 gm. a day (ʒ ij.—iiss.), in 2 gm. (30 gr.) powders, to be taken in the milk; the magnesium salt will be used when a slightly purgative action is desired.

Reconstituent Treatment.

The injurious effect of hepatic diseases, resulting from the action of complex causes, upon the general nutrition of the body has induced practitioners to attempt an intervention capable of improving the nutritive processes in the tissues, and thus to establish a reconstituent treatment in diseases of the liver.

order that such treatment may attain the desired end, it will be
ary to treat the cause of the impoverished condition, and this
ndirectly be done by endeavoring to restore the normal func-
g power to the liver; truth to tell, this object is not easy of
ment, and often remains a mere therapeutic desideratum, be-
the means in our possession, especially when the disease is not
special a character that we can apply specific treatment, are
quate. For this reason the reconstituent treatment in hepatic
es may be only symptomatic.
n, arsenic, and quinine are of benefit in some cases. But the
of potassium or of sodium, according to individual idio-
sy, will best answer the purpose, as we have already stated.
, by accelerating the tissue changes, compensates for the most
tant disturbance of the nutritive processes in disease of the
which, by the lowering of its functional activity, induces a
ation of metabolism, manifested by a diminution in the for-
a of urea, the ultimate product of the digestion of albumin-

e most efficacious means we possess for the improvement of the
l condition of the patient are measures directed to the ame-
on of the gastroenteric disorders, such as the administration
l which is of easy absorption and assimilation—in other words,
diet. To put the patient in the best possible climatic and
ic surroundings is another excellent method for improving his
ve condition. Pure air, full of oxygen and ozone; a mild, tem-
and dry climate; and comfortable, sunny, and well-ventilated
rs are to be insisted upon, for by these means we reduce the
of the micro-organisms and increase the resisting power, thus
ing an intraorganic condition favorable to the resolution of the
l process.

Surgical Treatment.

ce Lister gave the impetus to the enormous progress made by
r, even hepatic diseases have come within the reach of this
f treatment; we may even say that at the present time surgery
nphantly taking possession of this field, so that the treatment
ffection of the liver is in danger of becoming the specialty of
geon rather than of the physician.
do not desire to enter here upon a discussion of the broad
of hepatic surgery, but will merely state that from suppura-
patitis to biliary lithiasis, from hydatid cysts of the liver to
owths, a vast field is daily being invaded and conquered by

surgery, and the successes so far obtained give reason to hope that many diseases now considered incurable may be helped by these measures.

Symptomatic Treatment.

In clinical practice it is often necessary to find therapeutic measures for the relief of certain symptoms of the disease, either because of the suffering which these symptoms cause, because they threaten life, or because they increase the hepatic trouble. As they may arise in the most varied diseases of the liver, we will treat of them here in a general way, in order to avoid repetition when we consider them in the special portion of this work. The most frequent symptoms, and the ones most deserving of our special attention, are ascites and jaundice.

The treatment of ascites consists chiefly in intestinal derivation, in paracentesis, and in drainage of the peritoneal cavity. The first is directed to the deobstruction of the portal circulation and to the inspissation of the blood in the venous radicles of the intestines, thus promoting absorption of the transudate; the other measures are directed to the extraction of the ascitic fluid. The choice of a method will depend upon the special conditions. Diuretics are usually of little service in the ascites of hepatic diseases, but they are of benefit when combined with cardiac stimulants when the ascites seems to depend chiefly upon cardiac weakness.

Apart from the various cholagogues, the symptomatic treatment of icterus is largely addressed to favoring the excretion of the bile, and for this purpose Mosler and Krull have recently recommended enemata of cold water; this has always given good results in our own practice, only we never give as much as two litres (quarts), but keep the amount of each injection below one litre. The patient must be advised to retain the water as long as possible and to repeat the enema two or three times during the course of the day. This treatment improves the rectal circulation, either because it empties the intestines of abnormal accumulations of fæces, or because it induces increased peristalsis, which is propagated to the ductus choledochus and to the other large bile ducts.

Pain is a frequent symptom, and in some affections the most unendurable. The use of anæsthetics and analgesics (morphine, atropine, antipyrin, etc.) is justifiable. In some cases, however, it will be well to resort to the local application of hot poultices, to mild epispastics, or to the ice bag. In paroxysms of pain hot sitz baths are to be recommended.

It will be seen, from what we have said in this section, that the

utic measures for the treatment of hepatic diseases are limited
ıber, but extremely efficacious when appropriately applied.
an etiological treatment is not possible, the physiological
nt, based mainly upon a milk diet and the use of iodine, will
d to give the very best results.

ERATIONS IN THE FORM AND POSITION OF THE LIVER.

Deformities.

morphological anomalies of the liver may be either congeni-
cquired, and, as a rule, are of rare occurrence.
result of abnormal fœtal development, the liver may become
l in its growth or may possess supernumerary lobes, or there
casionally be accessory livers, as we may term those more or
nded portions of hepatic parenchyma, which are met with,
rare anatomico-pathological anomalies, however, in the ab-
l cavities and which sometimes (as in Riegel's case) may lead
ror of diagnosis.
changes in the form of the liver which are the most frequent
ich possess the most clinical interest are the acquired ones,
lt of mechanical compression upon the hypochondria and the
of the ribs by the corsets of such women as seek to enhance
tractions by squeezing the lower part of the chest and most
tically exaggerating, even to monstrosity, the curves of bust
. Direct pressure by the corset, and indirect pressure by the
ted ribs, cause a downward displacement of the liver in the
n of least resistance. Pressure atrophy of the hepatic paren-
causes the formation of a horizontal furrow upon the convex
of the right lobe of the liver, at a variable distance from
rnal margin. The investing peritoneum of the liver mean-
ecomes so thickened that it may finally be seen as a fibrous
ing at the lower part of the furrow and extending along its
ength. It will readily be understood that the production of
omaly is a very gradual process, and that from a clinical
int the advanced stages alone of the lesion are of importance,
t because of possible errors in diagnosis which they may
ı. In the initial stage an objective examination by means
of palpation will show that the liver is displaced downwards,
n its right surface, just beneath the costal arch, may be felt
of greater or lesser depth, according to the case; the diag-
ided perhaps by the history and by possible osseous deformi-

ties and cutaneous lesions which arise from the same cause as the change in the form of the liver, will be both easy and positive. But when this special anomaly is in so advanced a stage that the furrow forms a species of girdle circumscribing a portion of the biliary gland, it is not impossible that a few loops of intestine may be thrust into the sulcus in such a way that the lower movable portion of the liver is found to be separated from the area of liver dulness by a band of tympanitic resonance, in which case an erroneous diagnosis of abdominal tumor might easily be made. By palpation, however, we can readily find the furrow, and deep percussion will cause the tympanitic resonance to be replaced by dulness. These methods of examination will be all-sufficient to determine the special form of alteration present.

Prophylaxis is the only possible treatment; stiff corsets and tight lacing should be forbidden.

Displacements.

Anomalies in position of the liver may also be congenital or acquired. The first are rare, and are composed of cases in which there is a transposition of the viscera, the liver being found on the left side and the spleen on the right. As a rule, when the abdominal organs are inverted, the thoracic organs will be so likewise, the heart being found to the right, pulsating in the fourth or fifth right intercostal space, although a few cases have been known in which the transposition of organs involved the spleen and liver alone. One such case, which was unmistakable, we have ourselves seen in the Hospital for Incurables in Naples. When there is congenital diaphragmatic hernia, a portion of the liver may be found in the pleural cavity; in absolutely exceptional cases small accessory hepatic lobes may be found in this position.

The anomalies in position of the liver which possess clinical importance are, however, the acquired ones, especially cases of migrating or movable liver, or, as Glénard calls them, hepatoptosis. The existence of this special form of disease was demonstrated by Heister in 1754 upon the cadaver, and by Cantani in 1866 upon the living subject.

Etiology.—Movable livers, like movable kidneys, are found most frequency in women, and their occurrence has been attributed to pregnancy and to the wearing of stays, but although these may produce the condition, they cannot be considered to account for it completely, hepatoptosis having been noted in virgins and in women who had never laced. Straining efforts, and flaccidity of the abdomi-

alls after pregnancy have been rated among the causes, but no actory explanation has been given of the mechanism of the first, hat the second is not even an essential condition in the produc- of hepatoptosis is shown by the fact that it is frequently found rsons whose abdominal muscles are not in the least relaxed. ni attributed the displacement of the liver to inflammation of eritoneal hepatic ligaments, but as Faure remarks, inflammation l result in thickening of the ligaments rather than in a further tion. It would appear, however, that the cause might be found genital anomalies of the ligaments, consisting in either a greater or a greater extensibility. From this point of view it will be y understood how any influence which would cause a diminu- f abdominal pressure (as repeated pregnancies, etc.) might in- displacement of the liver. This theory of a congenital anomaly ligaments is further confirmed by the fact that in some cases idneys as well as the liver are found to be movable. For this we do not share the opinion of those who consider the displace- of the liver consecutive to that of the kidneys, the kidney not a support to the liver; neither do we believe that a movable is at all likely to cause displacement of the kidney, since the organ is attached to the vertebral column much more firmly o the liver.

e *pathological anatomy* of hepatoptosis is little known, inas- as the condition is not a fatal one. The liver is usually found entirely displaced in a downward direction, the right lobe the lowest and slightly directed towards the median line.

mptoms.—Hepatoptosis may occur without giving rise to any ard symptoms, and the condition may be discovered only by nt, in the course of an examination of the abdominal cavity for other disease. In other cases, however, the subjective symp- may become so painful and of such severity as to assume a great tance. The usual symptoms of which the patient complains sense of weight in the epigastrium and right hypochondrium, a or less intense pain of a neuralgic nature in the region of the sometimes radiating to the shoulder, an increase of these symp- in the erect position and their decrease in the supine. In an- class of cases the clinical picture is far more marked. The , which have all the characteristics of hepatic colic, become and more severe, and may give rise to reflex phenomena, being panied by a more or less intense jaundice caused by torsion of uctus choledochus, and ascites from portal stasis due to tension ompression of the portal vein. These symptoms may, of course, dily confounded with those of other hepatic lesions, for which

reason the objective examination will have to be depended upon to illuminate the question. Towards the inferior portion of the right side of the abdomen will be found a tumor resembling the liver in its shape, smoothness, and consistence, while upon its sharp margin, if the abdominal walls are sufficiently thin, may be recognized the two fissures of the organ. In some extremely rare cases one may even feel the falciform ligament like a slender fibrous band. The tumor is usually freely movable, so that, the patient being placed in a recumbent position, the physician may, by using both his hands, replace the organ in its normal position. Tympanitic resonance instead of liver dulness in the hepatic area will greatly assist in the diagnosis. In the case of repeated pregnancies the abdominal walls may be relaxed, and symptoms of gastroenteroptosis may sometimes be present.

The *diagnosis* is frequently difficult. Frerichs and Müller have described two cases in which carcinomatous degeneration of the omentum gave all the objective symptoms of a movable liver. Errors in diagnosis may easily be made with reference to abdominal tumors, especially tumors of the right kidney, the uterus, the ovaries, etc. Of the greatest diagnostic importance are the form of the tumor, its movability, the possibility of its reduction to the normal position, and the information obtained by percussion of the liver area before and after such reduction.

The *treatment* is purely symptomatic. An attempt may be made, after returning the organ to its original position, to fix it there by the use of pads kept in place by a wide abdominal bandage, but, as a rule, little success is obtained by this means. The only rational therapy would consist in surgical measures for the fixation of the liver. Billroth, in the course of a laparotomy for supposed abdominal tumor, having found a lobe of the liver separated from the rest of the organ and displaced in the abdominal cavity, sutured it to the walls of the abdomen and was thus the first to perform *hepatopexy*. Marchand in 1891 performed a true hepatopexy for movable liver. But we question seriously whether such surgical intervention is justifiable for the relief of hepatoptosis. The operation being a major one, and liable, even in the event of operative success, to cause serious alterations in the liver, it would seem that it should be reserved for those exceptional cases in which the functional disturbances are so severe as to render existence unbearable. As a rule, we shall have to be satisfied with relieving the symptoms as far as possible by medical measures.

CHANGES IN THE CIRCULATION.

e enormous amount of blood contained by the liver in compari-
th the other organs, its double system of capillaries, the varia-
n circulation which it undergoes during the phases of respi-
and of the cardiac cycle are the most direct causes of the
ıcy of the occurrence of important alterations in the local cir-
n of the liver, which constitute a veritable disease. For this
we are able to devote a special section of considerable clinical
ance to the circulatory disturbances caused by hyperæmia of
gan, whereas in the consideration of other organs, however
the circulatory disturbances may be, they cannot be treated of
ut as symptoms dependent upon the same lesions. From this
view the liver may be compared to the brain, in which a study
irculatory disturbances is of the greatest interest, with this
ce, however, that whereas in the cerebrum the importance of
ration in the circulation is due to the high character and the
icity of its functions, which if disturbed manifest such dis-
e at once more or less locally, in the liver a study of the dis-
f circulation derives its chief interest from the frequency with
hey may occur, in spite of the abundant supply of blood-
and amount of blood which they contain, from the digestive
ances to which they almost invariably give rise, and finally from
omico-pathological lesions which may develop. In the study
ic congestions, although from the standpoint of pathological
it is not always possible to distinguish differences and estab-
ecial types, it is important from a clinical and a therapeutic
view that we recognize two forms, viz., active hyperæmia and
hyperæmia.

Active Hyperæmia.

Etiology.

ng the period of digestion there is a physiological active
nia of the liver. This periodical normal congestion is due
to the fact that there is an increased flow of blood to all the
organs during digestion (since all increase of function is
by an increased blood supply) as it is to the absorption of
ucts resulting from the chemical processes in the intestines,
e carried through the portal system to the liver, where they
tate the hepatic cells; the latter are at this time in a con-
overwork, so to speak, from the necessity they are under of

transforming the various products of digestion absorbed from the intestine. It will, therefore, be easily understood that the degree of physiological active hyperæmia will be in direct relation with the amount and the quality of the substances ingested and absorbed. Thus when food of an irritating nature, or which is liable to cause abnormal fermentation in the intestines is taken, the degree of hyperæmia will be high, and may even go beyond physiological bounds, as may be seen in the case of persons who overindulge in the use of wines, liquors, spices, etc., or who eat a quantity of indigestible food. If these habits are persisted in, the condition of portal congestion becomes permanent and a state of pathological active hyperæmia is induced which is subject to aggravation during the digestive periods.

In addition to these gastrointestinal causes of the disease, which, truth to tell, are the most frequent and claim the careful attention of the clinician and the therapist in their treatment, there is another series of causes of a general pathogenic nature, namely, dyscrasias, infections, and toxæmias.

Among diathetic diseases gout is the chief cause of active hyperæmia of the liver, which may occur as a premonitory symptom of the attack, or during the attack, or even independently as a symptom of visceral gout. In this event the hyperæmia of the liver possesses all the characteristics of active hyperæmia, because it represents an increased activity of that organ which is endeavoring to reduce the uric acid accumulated in the blood to its ultimate condition of complete oxidation, urea (see the section on General Pathology). Exceptionally, syphilis and scrofula may be considered as factors in the causation of the hepatic congestion, the first when secondary to a gastrointestinal catarrh, the second by causing fatty degeneration of liver.

When there is infection, the liver in order to fulfil its function as a protective organ becomes more or less hyperæmic; thus in typhoid fever, in malaria, and in dysentery hyperæmia is constant and may even go beyond the limits of a simple circulatory disturbance and become a veritable inflammation.

Among the toxæmias which may cause hepatic hyperæmia we may mention poisoning by alcohol, carbonic oxide, mercury, carbolic acid, phosphorus, arsenic, nicotine, etc.

Nervous causes should not be omitted from the list, as the first condition leading to hyperæmia, instead of being a direct irritation of the hepatic cells, may be a functional alteration of the vasomotor nerves. On this hypothesis alone can we understand how the suppression of menstruation or of an hemorrhoidal flow by sudden chilling, can result in active hyperæmia of the liver.

inally, we may say that climate has something to do in the pro-
on of this disease. It is a fact noted by all pathologists that it
frequent occurrence in tropical countries, so that the name of
al hyperæmia has been given to it, but opinions differ as to its
genesis. Some authorities lay emphasis upon the elevated tem-
ure of the atmosphere, while Kelsch and Kiener[11] attribute the
se to an infection. We, however, believe with Chauffard that
a rapid change is made from a temperate to a hot climate, in
ion to the general disturbance of the organic functions, there
e a slight overproduction of the biliary secretion, which may
rified by the fact of an exaggerated elimination of biliary pig-
in the fæces (Rattray). Thus it is easy to see how a slight ac-
tal occurrence (such as a nocturnal chill, a too abundant diet,
cess of alcohol) may cause a congestive condition of the liver, so
he infective germ of dysentery or malaria will find prepared
d, as it were, and that serious lesions may be the result.

Pathological Anatomy.

e structural alterations produced by active hyperæmia are those
on to all hyperæmias, and consist in an engorgement of the
vessels. There is also an increase in the volume and weight of
gan; the increase in volume is uniform, so that the liver is en-
but in nowise deformed. Its substance is homogeneous and
ly at all increased. The surface of a section appears smooth,
llows of the escape of a greater amount of blood than normal.
psular ecchymoses are sometimes noticed. Under the micro-
there is seen to be a considerable increase in the diameter of the
aries which are filled with blood corpuscles, and in the case of
rhages red blood corpuscles will be found in the connective-
spaces, and even within the acini. The cells of the paren-
are swollen, partly from cloudy swelling and partly from en-
nent with the yellow granules of bilirubin. In the more severe
such as may be considered the first stage of hepatitis, we find
ginning of a radicular angeiocholitis, and a fatty-granular in-
on of the ganglion cells.

Symptoms.

e symptoms of hepatic congestion may be of gradual appear-
ut as a rule they occur somewhat acutely. In the first case
tient becomes aware of a sense of tension in the epigastrium,
d by more or less severe symptoms of dyspepsia. In the sec-

ond case, there may be a slight chill and febrile movement, which will usher in a sharp pain in the right hypochondrium, a severe feeling of tension radiating to the right shoulder; symptoms of dyspepsia, which is one of the causes of the disease, may precede the pain, and will go on increasing, resulting in nausea, vomiting, and bilious diarrhœa, a group of symptoms closely resembling the initial stages of an infective disease. After two or three days there will be slight jaundice, especially to be noticed in the sclerotic conjunctiva, which may be followed by true icterus, of no great severity, and originating from pleiochromia, since it is accompanied by deep coloration of the fæces.

The urine undergoes important modifications. It is as a rule reduced in volume, of a higher specific gravity, and is intensely yellow or even icteric. A chemical examination will show bilirubin or urobilin, according to the amount of biliary pigment thrown into the circulation, and to the existence or non-existence of insufficiency of the hepatic cells. An important diagnostic point is the amount of urea excreted; it is always increased, even to 40 or 50 gm. (600 to 750 gr.) in the twenty-four hours. This increased elimination, of course, bears a relation to the increased work of the hepatic cells, and will therefore almost serve as a criterion for determining the extent of active hyperæmia of the liver.

In severe cases it is usual to find swelling of the spleen, but not to any marked extent; it is due to the stasis caused by the obstruction to the portal circulation. We may in addition have general symptoms of varying intensity, such as weakness, emaciation, etc.

Of great aid to the diagnosis are the objective symptoms, especially when the liver has increased in size through congestion. Inspection will sometimes reveal signs of some importance. In cases of very intense hyperæmia the enlargement of the organ may cause a relative prominence of the right hypochondrium as compared to the left. The lower ribs of the right side are slightly pushed above the enlarged organ, and may even be displaced in their longitudinal axis, as the upward movement occasions a movement of rotation which brings their external surface upwards, and their internal surface downwards, while the inferior margin tends to become external and the superior margin internal. At the same time the intercostal spaces become somewhat decreased in size.

Palpation is, as a rule, of the greatest assistance in the diagnosis. By its aid we become aware of a certain sense of resistance in the right hypochondrium beneath the ribs, and by continued palpation in a downward direction we may reach the inferior margin of the liver, which will be found projecting two or three fingers' breadth or more

m the costal arch, while it reaches beyond the median line between base of the ensiform cartilage and the umbilicus.

Pain in the hepatic region is usually increased by palpation, some- es to such an extent that it can be performed only with the great- care. This pain, which may constitute one of the most prominent ıptoms of the disease, is due to the rapid distention of the peri- eal capsule of the liver which is supplied by the sensory fibres of pneumogastric. If, however, the pain should not be marked in racter, and there should be little tension of the abdominal walls little meteorism, we may by palpation determine the nature of anteroinferior border of the liver, which we shall find to be smooth, mal in shape, of increased consistence, sharp, or perhaps a little e rounded or blunt than usual. Sometimes we may be able to feel two anatomical fissures, especially the longitudinal.

Percussion will reveal the area of absolute and relative dulness of enlarged liver, the size of which will of course depend upon the ınt of the lesion. A point worthy of attention is that the hepatic lling is subject to sudden variations; these may be spontaneous, as is most frequently the case, due to diet, to the mode of life, or ı to an increase or a decrease in the alvine discharges, as the latter se a sort of clearing out of the portal circulation.

In our climate active congestion of the liver produces no other ptoms than these; the gastrointestinal disturbances and fever n intermittent or remittent type, but always limited in degree, perhaps be traced to the intestinal lesion which is the cause of disease, but are the morbid phenomena which claim the chief at- ion of the clinician.

In hot countries, however, hyperæmia of the liver presents a dif- ıt train of symptoms altogether. Icterus is marked and may be mpanied by all the symptoms of cholæmia; diarrhœa and the iting of bile may occur, the patient may suffer from severe head- s and from fever of a more or less intermittent type which may all the features of a malarial miasm or of suppurative fever; the se of the disease may have the regularity of a true infective proc- ınd at the end of a few days terminate in recovery, providing that hyperæmia be not a sign of a suppurative hepatitis.

Duration and Course.

The duration and course of this disease vary greatly according to causation. There is an acute form with a somewhat cyclical se, which resolves after eight or ten days; this is seen more espe- y in the catarrhal hyperæmias of hot countries.

In the temperate regions the most commonly occurring type is the subacute. During the course of digestive disorders, and from the abuse of alcohol, catarrh of the liver is produced with a certain degree of acuteness, but prompt intervention with the appropriate remedies will cause a retrogression of the symptoms, and in from ten to fifteen days the disease will run its course.

The chronic form of the malady deserves the careful attention of the physician. In certain diathetic conditions, and in the frequent intestinal disorders produced by alcoholic and dietary excesses, a chronic congestion of the liver is established which is liable to acute exacerbations by repetition of the exciting cause. This form is the more worthy of attention in that it may run an insidious course unattended by symptoms of any special annoyance to the patient, while at the same time it signifies that the liver has already begun to feel the effects of the functional or anatomical changes in the intestines, and that the point has been reached where physiological compensation can no longer be expected to be effective. Both physician and patient should realize that the biliary gland is worthy of serious care and attention in order to obviate the fatal results which are certain to follow if the hygienic and dietetic conditions are not radically changed. Therefore when alcoholic patients seek the physician's advice for disorders of the stomach and intestines, the liver is the organ which should be subjected to the most rigorous examination.

Diagnosis.

The diagnosis, as a rule, is simple, and there is slight possibility of error. The etiological data are, however, extremely important, and careful inquiry should be made in order to ascertain whether digestive disturbances or alcoholic excesses exist which might justify the inference that there are circulatory lesions of the liver. When, in a patient who has used alcohol to excess, or who has suffered from repeated attacks of malaria, we are able to recognize hepatic lesions in addition to functional phenomena and increase in size of the organ, we shall still have to diagnose between active hyperæmia of the liver and chronic interstitial hepatitis unaccompanied by much atrophy. A rapid development of the disease will be in favor of a simple circulatory disturbance; of more importance, however, is a careful examination of the urine, for we shall find an increase in the daily amount of urea in the case of active congestion of the liver, and a decrease when a cirrhotic process is present. This differentiation is less important from the standpoint of therapy than from that of prognosis. Still, it is well to bear in mind that unless intervention is

mpt hepatic hyperæmia may lead to a chronic interstitial hepati- From this fact it is easy to understand that the steps from the form of disease to the other are very gradual, and that they are to be discerned clinically with ease, for they can scarcely be ap- iated by the pathologist. In the hepatic hyperæmia of hot coun- the acuteness and gravity of the symptoms are such that it is to mistake the disease for the suppurative hepatitis which is of uent occurrence in those regions. The diagnosis can be based upon the severity of the symptoms, the elevation of the tempera- , the general condition of the patient, and above all upon the se of the disease.

Prognosis.

n active hyperæmia of the liver the prognosis is usually good; as ave to do with a disturbance of the circulation, and as the causes easy to eliminate, a rational treatment will secure a rapid dis- arance of the morbid phenomena. We must remember, how- , that if the patient will not or cannot remove the etiological rs, a train of successive changes may occur in the liver, the cir- tory disturbances often being the note of alarm which will call hysician's attention to disturbances caused by digestive disorders the abuse of alcohol. This will of course serve to modify the nosis which is good in the case of the disease itself.

Treatment.

nly by addressing our treatment first of all to the etiological rs can we obtain a positive result and obviate the fatal conse- ces which are so liable to occur. The most important indications o regulate the diet in hearty eaters, to forbid absolutely all al- lic liquors in those addicted to their use, to treat an existing y diathesis, to regulate hemorrhoidal circulation by means of es applied to the perineum, to cure malaria, and to remove the nt from a tropical climate. Treatment addressed to the process will consist in the institution of a rigid milk diet. We know during even normal digestion an active hyperæmia of the liver s, this functional hyperæmia being more marked according to mount of work required and according to the nature of the ex- entitious products of digestion which are absorbed by the portal and, by irritating the hepatic cells, help to increase the flow of l to the liver. Now milk is an aliment already half digested, luces the work of digestion to a level below the physiological, origin to products which are quite innocuous to the hepatic

cells, and enormously diminishes the amount of toxic matters produced during the chemical work of digestion, thus proving itself to be the best of all intestinal antiseptics. By this means the periodical digestive hyperæmias become notably reduced, and the liver, from the functional rest which it enjoys, is in the best condition to enter into the period of recovery. In addition to the milk diet, it is of the greatest importance to remove all obstruction to the portal circulation, by the abstraction of blood at the anus, and the administration of saline purgatives, among the best of which are the natural mineral waters, such as Hunyadi János, and the like; or calomel may be used as a cholagogue.

The thermal cures of Vichy, Carlsbad, Montecatini, Castellamare di Stabia, etc., are adapted to the prolonged cases with frequent exacerbations, and to the enlarged liver of malaria and of dysentery. A third therapeutic indication is the antiphlogistic treatment, which, however, should be resorted to only in those cases in which we have an acute form of the disease with severe local symptoms, an intense pain, and a sensation of tension in the right hypochondriac region, these symptoms perhaps constituting the chief suffering of the patient. Copious leeching in the right hypochondrium, dry cupping, or better yet, scarification, will materially aid in reducing the painful sensations. For the same purpose we may apply cold to the hepatic region, in the form of compresses wrung out in iced water, or of an ice bag, according to the severity of the case.

We must not forget that many clinicians make use of intestinal disinfection by means of insoluble antiseptics in divided doses (naphthol, betol, etc.). We have already expressed our opinion of this form of treatment, and will only repeat that we consider milk to be the most valuable intestinal antiseptic.

Passive Hyperæmia.

Etiology.

We have already studied the etiology of passive congestion of the liver in the section on general pathology (page 411), and therefore we shall here merely enumerate the causes of stasis, which consist chiefly of mechanical obstructions to the general circulation.

Hepatic hyperæmia is a common sequel of any of the valvular heart lesions, especially such as are connected with the venous apertures, when a disturbance in compensation finally causes increase of pressure in the veins with its decrease in the arteries. Somewhat less frequently hepatic stasis is produced in cardiac asystole due to le-

n of the pericardium or of the muscular tissue of the heart (myo-
ditis, degenerations, pericarditis with exudation, pericardial ad-
ions), to diffuse arteriosclerosis, senile marasmus, deformities of
thorax, aortic aneurysm, or aneurysm of the large arteries, etc.
their influence upon cardiac activity, and by themselves as well,
ections of the respiratory tract frequently cause passive hyper-
ia of the liver, which may result from acute forms of disease
eurisy with effusion, croupous pneumonia) or from chronic ones
eurisy, empyema, bronchial asthma, chronic diffuse bronchitis,
erstitial pneumonia, pulmonary emphysema, etc.). In some rare
es the hepatic stasis is due, not to disturbances of the general cir-
ation, but to some obstacle to the circulation in the suprahepatic
as. Thus Frerichs found a formation of valves in the hepatic
as, and Gee describes a case in which the openings of the hepatic
as into the ascending vena cava were closed by a membranous sep-
n. Bridles of connective tissue, the result of pericarditis, are
metimes developed around the suprahepatic veins, diminishing the
men and thus interfering with the circulation.

In the development of disease of the liver from stasis, when dis-
bances of the general circulation exist, there are other factors
ch must have something to do with the production of this local
turbance of the circulation, since even when marked asystole of
atever nature is present, it is not produced to the same degree in
patients. This variability in individuals may be due to a vary-
resistance in the blood-vessels in certain portions of the body, so
t in some patients the most serious disturbances will be caused in
ne special organ, in other patients in some other tissue or ap-
atus.

Occasionally the history of the patient will throw light upon this
iability in resistance of the hepatic veins. Alcoholism, malaria, or
quent digestive disturbances may cause this "cardiac liver" by
ating a *locus minoris resistentiæ*, that is to say, a predisposition to
ons of the circulation.

Pathological Anatomy.

The anatomico-pathological lesions vary according to whether the
gestion is or is not of recent date. When it is recent, the entire
an is uniformly increased in size, is of a dark-red color, of greater
sistence, and its anterior margin is smooth and rounded. Upon
ion a large amount of blood oozes from the surface of the cut por-
n, the veins are seen to be greatly dilated, and the middle part of
acini, which sometimes are elongated and resemble oak leaves, are

of a dark bluish-red color, their periphery forming a sharp contrast, as it is of a much paler hue, a brownish-yellow or even yellowish-white, according to the degree of fatty degeneration present. This form of lesion has been named *nutmeg liver* from its resemblance in appearance to a nutmeg; the name of "variegated liver" has also been given to it. From its etiology it may be called *cardiac liver*.

A microscopic examination will show the central vein to be dilated and thickened; the hepatic cells towards the centre of the lobule are flattened and deformed, with a granular protoplasm which is filled with brown pigment or with crystals of hematoidin or of bilirubin, while the peripheric cells are swollen and more or less filled with fat globules.

When, however, we have to do with a passive hyperæmia which is of long duration, other and successive alterations occur, leading to the *cyanotic atrophic nutmeg liver*, otherwise called *cardiac cirrhosis*, or, according to Virchow, *red atrophy* of the liver.

The organ in this case is not much enlarged, and is sometimes even reduced in volume below the normal size, is of a dark-red color, harder in consistence, with a granular or nodulated surface, and is resistant to the knife.

Many researches have been made with a view to an understanding of the anatomical lesions observed in this special form of cyanotic atrophy. Thus Rendu and Ziegler believe that we have to do with a bivenate cirrhosis, while Legg and Talamon think that in the initial stages there is periarteritis. Sabourin has demonstrated that around the suprahepatic veins stasis produces a sclerotic periphlebitis, which involves the hepatic lobules that are undergoing atrophy and imparts a peculiar areolar appearance to the sections, thus causing the cirrhosis with small subcapsular granulations, or with large fibrous bands circumscribing less extensive areas of parenchyma. The arterial lesions which are sometimes met with in cardiac liver should be attributed to alcoholism, which will undoubtedly be found in the patient's history.

Lesions of the other organs will of course be found in connection with cardiac liver, more especially of the spleen, kidneys, stomach, and even of the pelvic organs.

Symptoms and Course.

When in the course of a disease of the cardiac or respiratory apparatus, marked disturbances of the general circulation occur, the congestion of the liver which is produced with that of the other organs may be a morbid process totally devoid of interest from the point of

of either prognosis or therapy. That is to say, that in connec- with the œdemas, ascites, and other serous effusions present, and the stasis of other parenchymatous organs, stasis of the liver also be found and may oftentimes even be taken for granted with- an objective examination. It may also be passed over, on account ie prominence of the other lesions due to cardiac asystole.

But there is another class of cases in which the whole attention of physician will be attracted to the hepatic lesion. Valvular dis- of the heart which is latent for a long period will produce circu- y disturbances which react with special force on the liver, the ent suffering from pain in the right hypochondrium, a sensation nsion in the epigastrium, and more or less marked digestive dis- ances. After having established the fact that there is enlarge- t of the liver, the physician, by a careful examination of the heart, find that the primary affection is in the circulatory system, the tic lesion being entirely secondary. This condition may become emely grave owing to insufficiency of the heart action, while, as sult of the circulatory disturbances, only the liver is found con- ed, there being no œdema, no congestion in other organs, no us effusion in the pleural cavities. In this way arise the cases o-called *hepatic asystole* (Hanot), and these are the cases which for especial care upon the part of the physician, because the rational treatment, the only one which will secure good results, at which is directed to increasing the force of the heart's action restoring compensation. Especial care must be taken not to mit the error of supposing that the cardiac lesion is a result of hepatic lesion. (See the section on General Pathology—rela- between the liver and the heart.) An accurate clinical history an exact appreciation of the cardiac affection will serve as aids correct diagnosis.

From among a number of cases of this description observed in tice, we may mention one seen in the Hospital for Incurables in les, in which the notable diminution of the hepatic area, the great lopment of the collateral circulation, and the large amount of as- without symptoms of stasis in any of the other organs and with- edema, caused us to make a diagnosis of chronic interstitial hepa- in the cirrhotic stage, the patient giving a history of alcoholism. reful physical examination showed that there was stenosis and fficiency of the mitral valve, with an atrophic cyanotic nutmeg . The symptomatology of hyperæmia of the liver from stasis es first of all to such modifications of the organ as can be ascer- d by physical examination. The liver is enlarged and protrudes greater or lesser extent beyond the borders of the ribs; its sur-

face is smooth and somewhat hardened in consistence, the inferior margin is rounded and easily felt unless the amount of ascites present should cause a marked increase of the intra-abdominal tension. In this case, palpation combined with succussion would be useful. The tumefaction of the organ may reach such a point as to induce a dilatation of the right hypochondrium which will be visible on inspection. To these objective symptoms may be added the subjective symptom of pain, which is persistent and oftentimes notably increased by palpation. It is heavy, dull, and deep in its nature, and when there is no hepatitis present, is due to the distention of Glisson's capsule. When we have to do with hyperæmia from stasis, with organic or functional insufficiency of the tricuspid, we shall find in addition to other symptoms the venous hepatic pulse, due to the pathogenic conditions of which we have already spoken.

The hepatic stasis may cause symptoms of gastrointestinal catarrh, consisting of anorexia, vomiting, eructations of gas, and alternating constipation and diarrhœa. In severe cases of obstructed hepatic circulation we have hemorrhoidal or intestinal hemorrhages, which are in the nature of a physiological compensation, since after their occurrence we note a reduction in volume of the liver.

In cardiac cirrhosis, however, the symptoms are quite different. The increase in size of the liver is not marked, and in advanced cases we even find a notable diminution of the hepatic area; the organ is of a harder consistence, the anterior margin is neither smooth nor uniform, and the surface is slightly granular. Ascites, which is found to a greater extent than in cardiac hyposystole, may be the direct expression of impeded hepatic circulation, all the more because in this case it is accompanied by increased development of the collateral circulation, shown clearly upon the abdominal wall in the form of the *caput medusæ*. In hepatic congestion it is usual to find a slight icterus of the conjunctivæ and mucous membranes, and we may even have generalized icterus, due probably to dilatation of the intrahepatic venous system by the increased pressure in the biliary vessels, which induces increased intrabiliary pressure with consequent absorption of bile by the lymphatics. Lépine, however, holds that it is due to spasmodic contraction of the biliary ducts. As a rule, the urine of these patients contains a large amount of urobilin and uroerythrin, and when the jaundice is at all marked, we may also find a certain amount of pure biliary pigment.

In addition to these conditions relating to jaundice, the urine presents other important symptoms; its color is deepened, its amount diminished, the specific gravity is increased, urea is reduced with the daily diminution in volume of the urine, and increased whenever, by

ration of compensatory processes, the urinary secretion is in-
ed; uric acid and the chlorides are diminished, while the phos-
es are considerably increased, sometimes even to double the usual
unt (Parmentier). Finally, alimentary glycosuria is very liable
cur.

n addition to these phenomena, which are more or less directly
ed to the disturbed condition of the hepatic circulation, we find
ective and functional symptoms which should be laid to the
ge of the primary lesion rather than to that of the hepatic stasis.
noea of more or less marked severity, a weak pulse, albuminuria,
are symptoms which are almost always present, but which are
d by the primary affection of the heart or the respiratory ap-
us. We will merely recall the fact that when cardiac insuffi-
y is made manifest only by symptoms on the part of the liver,
ne subjective and functional symptoms caused by the cardiac
n constitute the *hepatic asystole* of Hanot.

Prognosis.

n congestion of the liver which together with stasis in the other
ns is a symptom of hyposystole, the prognosis will depend en-
upon the primary disease. But when, on the other hand, long
tion of the stasis has caused anatomical changes in the hepatic
nchyma leading to cardiac cirrhosis, this latter condition will be
hief one to be considered, even if the functional power of the
ac muscle is improved and compensation is established. The
al nutrition becomes progressively worse, cerebral and pulmo-
disorders appear, the kidneys become insufficient, and finally
xia leads to death.

n hepatic asystole, the result will depend absolutely upon the
of the cardiac function; improvement or death will occur ac-
ng to whether appropriate therapeutic measures succeed or not
mpensating for the strain upon the circulation.

s a rare result of cardiac liver we may have the occurrence of
ed jaundice, followed by death. The blood stasis, the interfer-
with the flow of the bile, and the congestive catarrh of the
tive tract enable us to understand how a secondary icterus may
which is caused either by a primary infection of the biliary
, facilitated by the slackened flow of the bile, or by an auto-
ication due to the condition of the digestive tract. In these
the slight existing jaundice rapidly increases, a somewhat high
appears, which is occasionally of an intermittent type and ac-
panied by severe chills, the urine is diminished in amount, the

urea is enormously decreased, albumin and hyaline casts appear, a typhoid condition is established, and in a few days death ensues.

Diagnosis.

The diagnosis of passive hyperæmia of the liver is easily made when we can demonstrate the etiological factor, since, as we have already seen, it is never idiopathic. Thus if there is increased size of the organ and we find either a cardiac affection or a pulmonary lesion or any other condition which could satisfactorily account for the condition, we may conclude that it is a case of congested liver. Difficulties in the way of a diagnosis may be found when the etiology is not clear, but in such case we may by the course, the history of the disease, and by exclusion arrive at a correct diagnosis.

In cases of hepatic asystole the most easily made error in diagnosis is the supposition that the cardiac lesion is consecutive to that of the liver. The rarity of such an occurrence, a careful investigation of the morbid symptoms, and a proper estimation of the anatomico-pathological condition of the liver will be of the utmost assistance in the diagnosis.

Treatment.

The therapy of passive hyperæmia of the liver consists almost entirely in treatment addressed to the primary cause, or hyposystole of the heart, so that the most efficient remedy will be such as compensates for the functional disturbance of the heart. We therefore use cardiac stimulants (digitalis, adonis vernalis, etc.) which excite the cardiac muscle, thus raising arterial pressure and indirectly increasing diuresis; in short, we treat the cardiac insufficiency with all the known remedies.

In addition, it is essential that we should use means to diminish the periodical digestive increase in the circulation, and to produce a depletion of the portal circulation (absolute milk diet, saline purgatives, cholagogues, alkaline mineral waters)—these have all been referred to in connection with active congestion of the liver. When in hyposystole the liver symptoms predominate, and marked ascites exists, producing an injurious effect upon the abdominal circulation, and when cardiac stimulants and diuretics have been used without success it is permissible to have recourse to paracentesis, which, we may be sure, will be of great advantage. In the case above referred to, paracentesis was repeated certainly ten times, and to this was due the fact that the life of the patient (whose heart did not respond in the least to the various cardiac stimulants) was prolonged a year and

f. After each operation a marked improvement was noted, the t's action became stronger, the urine was increased in amount, a better effect was obtained from direct diuretics which were administered (diuretin, calomel).

INFLAMMATIONS OF THE LIVER.

Perihepatitis.

The capsule of the liver is composed of two layers; a thin external peritoneal layer, and an internal connective-tissue layer, from which are given out prolongations of connective tissue which form the interlobular spaces of the hepatic tissue. From this peculiarity of formation it will be easily understood that a perihepatitis may arise either from the external peritoneal layer or from the internal layer, the latter easily becoming inflamed from primary inflammatory lesions of the connective tissue of the liver. This explains how a perihepatitis consecutive to a primary hepatic lesion may occur, if the lesion is an interstitial one or a periangiophlebitis, and that it is absent when the primary alteration is in the parenchyma, excepting when it is propagated by means of the contiguous capsule of the liver. Thus while it is usual to find perihepatitis in venous cirrhosis, it is never met with in hepatic degenerative processes, which, as we know, originate in the parenchymal cells.

Perihepatitis may assume different aspects and follow a varying course, thus it may be acute or chronic, dry or exudative; in the last case it is sometimes accompanied by a development of gas, has a different causation and different symptoms, and deserves therefore a special description.

Dry or Adherent Perihepatitis.

Etiology.

This form of inflammation of the capsule of the liver is rarely idiopathic, and is usually the result of traumatism, which includes the pressure of a tightly laced corset, as it is often found in connection with the deformity produced by this cause. It may also be due to syphilis, which very probably causes a primary inflammation of the capsule without hepatic lesion. Whatever anatomical alteration may reach the surface of the liver during its progress will cause an inflammatory reaction in the capsule with fibrinous exudation. The most frequent causes are neoplasms, echinococcus cysts, abscesses, etc. Perihepatitis may also be found in interstitial inflammations of

the liver and in venous cirrhosis, in which case it is caused by propagation through continuity of tissue.

Peritoneal affections almost always involve the peritoneal layer of the liver. Finally, affections of the stomach, the duodenum, the colon, and the kidneys are in rare cases propagated to the capsule of the liver along the gastrohepatic, hepaticoduodenal, hepaticocolic, and hepaticorenal ligaments. More often perihepatitis is found in connection with pleurisy of the right side, which is explained by the intimate lymphatic connection between the serous layers of the diaphragm.

Pathological Anatomy.

The surface of the liver is found to be of a grayish or yellowish color, covered either completely or in parts by more or less thickened and resistant false membranes, which are easily removed if the perihepatitis is of recent date. In a chronic case of long standing, however, they are solidly attached, and form adhesions between the liver and the costal walls, the diaphragm, stomach, intestines, and kidneys, constituting a fibrous and resistant involucrum for the liver.

In some cases, in consequence of an atrophic process at the transverse fissure of the liver, there occurs narrowing or occlusion of the portal vein or of the secretory bile ducts, or following a similar process near the openings of the suprahepatic veins there is stenosis of the latter and even of the ascending vena cava.

Symptoms.

Adhesive perihepatitis may in many cases run its course without giving rise to any morbid symptoms which would lead to its discovery; this occurs especially when, as is most often the case, it accompanies other hepatic affections. In other cases, the only phenomenon produced which is of any assistance in the diagnosis is a more or less intense pain in the right hypochondrium accompanying disease of the liver. Thus when in the course of an hepatic affection pronounced pains occur in the hepatic region which are increased by palpation, and when they cannot be attributed to increased tension of the peritoneal capsule of the liver, we must conclude that a perihepatitis has set in. This is rendered all the more probable if the preëxisting disease is of a nature liable to extend towards the periphery of the organ. There remains a small number of cases in which during life there are symptoms of occlusion of the portal vein, chronic icterus, stasis, etc., which at the autopsy are found to have been due to a chronic fibrous perihepatitis.

The clinical symptoms which point to an adhesive perihepatitis,

ally chronic in its course, may be divided into subjective and ob-
ive. The first consist of sharp pain in and about the right hypo-
ndrium, which is increased by the respiratory movements, by
ghing, and by pressure. Sometimes, in cases in which the pain
ery marked, we may notice a respiratory immobility of the right
of the thorax, which is rendered the more conspicuous by
on of the exaggerated movements of the left side. At times the
atic pain radiates to the right shoulder or towards the angle of
scapula; this is owing to the fact that the phrenic nerve sends
ll branches to the capsule of the liver, and that it starts from the
th cervical, which sends sensory fibres to the shoulder. In addi-
to the pain, a distressing subjective symptom consists in an un-
rollable hiccough, due to extension of the inflammatory process
he diaphragmatic peritoneum, and consequent reflex irritation of
phrenic nerve manifested by clonic spasm of the diaphragm.

The most important objective symptom from a diagnostic point
iew is perihepatic friction, revealed by palpation and auscultа-
. It coincides with the respiratory act, during which the anterior
ace of the liver grazes the abdominal wall. The inflammatory
ess of the capsule, propagated by continuity of tissue along the
toneal lining of the abdominal walls, may as an ultimate result
e fixation of the liver, which effectually prevents the descent of
nterior border of the liver during inspiration.

Prognosis.

The prognosis of chronic adhesive perihepatitis is, as a rule, favor-
. Very rarely does it cause the grave results above described,
the prognosis will usually depend upon the primary disease of
h perihepatitis is the direct consequence.

Treatment.

side from the treatment of the primary disease, therapeutic in-
ntion in adhesive perihepatitis will consist simply in alleviation
e pain, which is often so extreme as to claim the whole attention
e physician. Heat in the form of linseed poultices, or cold in
orm of applications of ice, according to individual idiosyncrasies,
lull the pain if it does not cause its entire disappearance. Occa-
lly, however, we shall have to resort to revulsion by means of
isms, painting with tincture of iodine, cupping, scarification,
which often greatly relieves the patient's sufferings. Morphine,
ver, is the sovereign remedy in cases rebellious to other treat-
. An injection of one, or with the necessary precaution 2 cgm.
($\frac{1}{6}$ to $\frac{1}{3}$) of morphine will almost always control the pain.

Suppurative Perihepatitis.

Etiology.

This affection (Pyoperihepatitis; Pyopneumoperihepatitis; Subphrenic Abscess) also may be either primary or secondary to other lesions of the liver or of neighboring organs.

Traumatism is to be considered as a primary cause, for (as is the case with abscesses of other organs and tissues) it acts as determining cause of a streptococcal infection, to which suppuration of the liver is always due.

The hepatic affections which may lead to a secondary inflammation of the capsule of the liver, terminating in a suppurative process, are of the most varied nature; superficial hepatic abscess, suppurative echinococcus cysts pointing towards the periphery of the organ, perforation of one of the bile ducts or even of the gall-bladder by a gall-stone, etc., may all with greater or less frequency cause pyoperihepatitis. But, as we have already noted, this may also follow other affections of the abdominal cavity of varied nature and situation; thus there are cases in which the collection of pus between the convex surface of the liver and the inferior surface of the diaphragm must be attributed to ulceration or cancer of the stomach or duodenum, to inflammatory paranephritic or parasplenic processes, to perforations of the small intestine, of the transverse colon, the cæcum, or the vermiform appendix, to preceding perityphlitis or appendicitis, or to suppuration of the right pleura which is propagated to the diaphragmatic peritoneum by means of the lymphatic connection between the two serous membranes. It is difficult to understand how pus which is formed in other parts of the abdomen, especially at a lower level, as in the case of typhlitis or suppurative appendicitis, can extend upwards; possibly this may be due to the peristaltic movements of the loops of the intestine combined with respiratory action of the diaphragm. Finally, suppuration of the hepatic capsule with the formation of a subphrenic abscess may be connected with an infective general disease, more especially with pyæmia and puerperal septicæmia.

Pathological Anatomy.

Suppurative hepatitis may, as we can readily understand, be either supra- or infrahepatic; the latter form, however, as it resembles rather an encysted suppurative peritonitis, we shall not dwell upon, but will study the suprahepatic form only.

When we have not to do with the early stages of the disease, in ich the symptoms are merely those of an ordinary inflammation, h a scanty fibrinopurulent exudation, but with an advanced condi-, the fundamental anatomical lesion consists of a collection of between the upper surface of the liver and the diaphragm.

The size of the abscess varies as does the amount of the contents. nay be the size of a pigeon's egg or may be large enough to con- a pint or more of pus. The latter may be creamy and odorless, but nore frequently ichorous, filled with necrotic tissue, putrid, fœtid, dark red or green color, according to whether there is an effusion lood or of bile. The walls of the abscess are, as a rule, irregular, sometimes throw off prolongations of false membrane which di- the cavity into loculi. In a few rare cases the walls are smooth lined with a pyogenic membrane (Zuber). Together with the there may be found more or less gas in the abscess cavity, whose in it is easy to account for when there is perforation of any part he intestine; when, however, there is no perforation, the idea utogenous origin from the pus naturally suggests itself, but is far n being substantiated. The expansion of gas against the walls of capsule causes decided enlargement of the tumor. This condition stitutes *pyopneumoperihepatitis*.

The lesions accompanying suppurative perihepatitis relate to the r, and also to other organs, according to the etiology. The dia- agm is always pushed upwards, and is more or less adherent to internal surface of the ribs from propagation of the inflammatory ess, the costodiaphragmatic space being thus partially or com- ely obliterated.

The lesions of the pleura are of varied nature and pathogenesis. urulent pleurisy may exist which may be considered either as e or effect of the perihepatic lesion.

Exceptionally, we may have a purulent pericarditis by propaga- through the opening for the vena cava in the diaphragm. As a lt of the perihepatitis we may sometimes have perforation of the hragm and emptying of the pus into the pleural cavity, or, more ly, into the lung substance, if there have been previously formed sions between the diaphragmatic and the visceral pleura.

Symptoms and Course.

The development of the lesion may be rapid and accompanied by nomena which attract attention to the liver, or it may be so in- ous and latent as to cause no noteworthy symptoms until it has ined very considerable proportions.

The most important subjective symptom is in this case also the pain, which is more or less severe in the hepatic region and which often radiates to the shoulder and interferes with respiration, making it short, superficial, and of the costal type. Peritoneal symptoms may also arise: meteorism, vomiting, hiccough, general depression, a weak pulse, etc. Fever of a remittent or intermittent type is often present, ushered in by chills and ending with profuse sweating. We may also have all the symptoms of a general pyæmic infection.

The objective symptoms are of great importance, but in describing them we must be careful to distinguish those relating to pyoperihepatitis from those belonging to pyopneumoperihepatitis, which have been carefully studied by Leyden, and form a clinical picture which he calls *subphrenic pyopneumothorax*, a name which is anything but appropriate to the seat and nature of the process, but which reminds us of the easily made error in diagnosis of confounding this affection with pyopneumothorax.

The objective symptoms of subphrenic abscess consist mainly in the finding of a more or less prominent swelling in the region of the liver; this may, however, be absent. The right hypochondrium appears to be dilated, the ribs assume a more horizontal position, the intercostal spaces sometimes protruding. By means of palpation we may occasionally appreciate the perihepatic friction when there are foci of dry hepatitis; and very rarely we may feel a sense of deep fluctuation in the dilated and protruding intercostal spaces. Percussion of the tumefaction will give a dull resonance which may extend upwards as far as the fourth rib, and which is in direct continuation with the liver dulness. The liver is found farther down than usual, which is a symptom of great diagnostic importance. The upper line of dulness usually extends from before backwards and from above downwards, which forms a distinction between this affection and purulent pleurisy. The various clinical types present variations as to the form and regularity of the upper limit of dulness, but as a rule it is regular and presents a convexity towards the thorax, thus following the curve of the diaphragm.

An important point in the diagnosis of subphrenic abscess, which serves to distinguish it from a purulent collection in the pleural cavity, is that discovered by Pfuhl in 1877. Upon puncturing the abscess by means of a trocar and inserting a water manometer, the pressure will be found to increase during inspiration and decrease during expiration; in other words, it follows the curve of endoabdominal pressure. The exact opposite occurs in cases of a purulent collection in the pleura; pressure is increased during expiration and decreased during inspiration, thus following the modifications of

ssure in the thoracic cavity. Zaffe states that when we have ıctured the abscess, insertion of the manometer is unnecessary, force with which the fluid issues from the trocar in the two piratory acts serving as a criterion of the variations in pressure. ıuffard calls attention to the fact that, this symptom being in ect relation with the function of the diaphragm, when the latter is ed upwards by a long-seated and extensive collection of pus, it lergoes a certain amount of paresis, and then Pfuhl's symptom y be entirely absent.

More recently Feletti[42] has noted that in a case of subphrenic ab- s the reverse occurred; pressure increased during expiration and reased during inspiration; according to this author this is to be lained by the fact that in this case a communication existed be- en the abscess and the bronchial tubes through an opening in the phragm. However that may be, the diagnostic sign described ve can be discovered only by means of an operative procedure of rtain importance which is in reality a therapeutic measure. In opinion, the fact that it is a surgical operation of some difficulty not devoid of danger greatly detracts from its value as a means iagnosis.

The usual result of abscess when death does not occur from pyæ- is perforation; this may occur exteriorly, into the abdominal ns through the peritoneum, or into the thoracic cavity. The rior opening is the rarest, and a few cases only have been re- ed. Gioffredi operated upon one case in the military hospital Naples in which a shirt-stud abscess of the posterior wall of the ax opened externally in the eighth intercostal space in the scapular region. It is more usual for the abscess to open into pleura, giving rise to symptoms of acute purulent pleurisy. The occurrence of an aperture into the bronchi may be diagnosed by purulent pulmonary cavity caused, which may or may not be mpanied by hæmoptysis. When the pus fills the pericardium, h is a most exceptional occurrence and due to perforation of the ral tendon of the diaphragm, sudden death will take place from iac paralysis. Rupture into the peritoneum causing a fatal sep- eritonitis, perforation of the stomach, the small intestines, or colon, attended by vomiting or purulent diarrhœa which causes ediate relief to the patient, who is, however, exposed to the ter dangers of a secondary septicæmia of intestinal origin, are of the possible results which almost invariably lead to death.

Pyopneumoperihepatitis is from an anatomico-pathological point ew very similar to pneumothorax, and may readily be confounded it because it may present all of the signs supposed to be charac-

teristic of the latter disease (amphoric respiration, metallic tinkling, a succussion sound, metallic resonance, which alters with a change of position, marked dyspnœa, dilatation of the right side of the thorax, etc.). The differential symptoms are, in addition to the clinical history of what the patient remembers to have suffered with reference to the abdominal organs: 1. In pneumothorax the whole right side of the chest is enlarged and protruding, while in pseudopneumothorax only the inferior portion is dilated; 2. In right pneumothorax the heart is pushed to the left, while in pseudopneumothorax it is only displaced upwards; 3. In pneumothorax the vesicular murmur is absent on the whole of the right side, while in subphrenic abscess with gas, it is heard at least in the infraclavicular space when a deep inspiration is taken (Leyden).

The *duration* of purulent perihepatitis is somewhat short (from fifteen days to several months), and the result is almost always fatal, although energetic remedial measures may cause recovery.

Treatment.

Nothing is to be expected from medical treatment. Surgical measures alone can afford relief and will consist in opening and antiseptic irrigation of the abscess. Details of the treatment belong to treatises on surgical therapeutics.

Tuberculous Perihepatitis.

Perihepatitis may be of tuberculous origin. As a rule, it is never primary, but always secondary to tuberculous infection of the liver or other organs. More often it is the result of a general tuberculous peritonitis, or is an anatomico-pathological development of acute miliary tuberculosis. In the latter case its importance is overshadowed by that of the disease producing it, and it may remain latent without causing symptoms sufficient to attract the attention of the physician. At the autopsy, however, upon the hepatic serous membrane will be found a great number of tubercles of various sizes.

There are, however, some cases in which a primary tuberculous perihepatitis exists and results in caseous degeneration, as has been pointed out by Lannelongue. The anatomical lesions of this form of disease are similar to those of ordinary pyoperihepatitis; in addition, we note only that corresponding to the purulent collection we find a cavernous excavation in the liver of varying size and of a tuberculous nature.

The *symptoms* of this lesion are those of subphrenic abscess; the course is, however, slower, the pain less intense, the fever less high.

patient's age is of importance to the diagnosis, the lesion being
ıd almost altogether in children.

It is so grave a form of perihepatitis that Lannelongue reports
e deaths out of seven cases.

Treatment.—The therapeutic measures are limited entirely to sur-
l intervention, and are practically similar to those resorted to in
rculous abscess of other organs and tissues.

Suppurative Hepatitis.

Suppurative hepatitis (hepatic abscess) may be taken as the type
nflammatory affections of the liver. Although known to the an-
ts, as evidenced by the fact of its description being given by
n and Celsus, it is only of late that it has been studied as to its
ils, the pathogenesis in especial. English physicians in India,
ehead, Twining, and Annesley in particular, have contributed im-
ant symptomatological descriptions; the observations of French
sicians in Algeria, among whom Cambay, Laveran, Hospel, and
é especially deserve mention, as also the careful etiological
ies made in Europe by Behier, Gallard, Harley, A. Bindi, etc.,
contributed greatly to the understanding of this important part
ıe pathology of the liver.

Etiology and Pathogenesis.

A study of the etiology and pathogenesis of suppurative hepa-
is of great importance, since it may suggest indications for both
hylaxis and treatment. We shall first consider the conditions
h predispose to inflammation of the liver resulting in suppura-
taking up later the determining causes and their mode of action
ving rise to the lesion.

Suppuration of the liver is found more often in men than in wo-
, the proportion being as 30:1. It is chiefly a disease of adult
being rarely met with in children, although more often found in
ıewly born. This point in the etiology is easy of comprehension
recall the fact that adult men are the ones most exposed to the
es which determine hepatic inflammations. They are also more
ect than women to syphilis and alcoholism, which create a con-
n of minor resistance in the parenchyma of the liver which is
favorable to the development of any infective process. Children
ıaturally exempt from these predisposing causes. Malaria acts
e same way by causing serious damage to the functions and to
tructure of the hepatic cells. The occurrence of hepatic abscess
e newly born is due to the frequency of suppurative phlebitis of

the umbilical vein, which may induce a purulent portal metastasis leading to hepatic abscess. Traumatism in the hepatic region may be regarded as a predisposing cause, excluding penetrating wounds which would, of course, be a determining cause. Circulatory disturbances, and even the more or less extensive lesions to the parenchyma following local traumatism, may induce a condition of minor resistance to infection by any cocci which may be present in the circulation, and becoming arrested in a spot favorable for their development cause grave nutritive and structural changes. This variety of traumatism develops a peripheric abscess. Penetrating wounds, on the other hand, cause a suppurative process in the substance of the liver by direct infection. Traumatic suppurative hepatitis is, however, of rare occurrence, the thoracic walls forming a secure protection to the liver. Budd in 60 cases of hepatic abscess found only 1 which was due to traumatism, and Morehead out of 318 cases found only 4 (1.3 per cent.).

Climate is the most important predisposing cause, so much so that a large number of hepatic abscesses are called *tropical abscesses*, being very frequent in hot countries and extremely rare in cold ones. They take conspicuous place in the list of diseases peculiar to the tropics, and have occasioned many researches into their symptomatology and pathogenesis. As to the geographical distribution they are frequent in Algeria, Egypt, and Syria, even more generally found in Turkey, Cochin China, and India, and in Senegal constitute almost a common disease. An important clinical fact is that Europeans are more subject to suppurative hepatitis than are the natives of these countries, possibly because the latter are so continually exposed to the predisposing causes that they acquire a certain immunity, and also because the Europeans do not observe the hygienic precautions necessary in these regions. Thus in Bombay the mortality is from seven to eight per cent. in the European hospital, and three per cent. in the Indian hospital. The foreigners most liable to the disease are those who have recently arrived from a temperate climate, rather than those who have become accustomed to the tropics, and, like the natives, have perhaps acquired immunity. It has also been noticed that the abrupt change from a tropical to a temperate climate may cause abscess of the liver in Europeans who return to their own country.

This great frequency of hepatic abscess in warm climates has been variously explained by different authors. Larrey, who accompanied the French army in the Syrian and Egyptian campaign, attributes it to thermic influences, to excessive fatigue, to alcoholism, and to the abrupt transitions from heat to cold which occur in those regions and

h give a too sudden check to cutaneous transpiration. Annesley to these factors chronic dyspepsia, habitual constipation, and rbances in the secretion of the bile.

Malaria has been considered to be a determining cause of abscess, now that we know the biological properties of Laveran's hæma-a, and that they are incapable of causing suppuration, this ry should be at once rejected. Capable of producing only nodu- hepatitis, malaria may perhaps be an important predisposing e by putting the hepatic tissues in a condition favorable to the lopment of an infection by cocci, but it cannot be regarded as direct cause of suppurative hepatitis.

Recent studies, especially those of Dutranbau and of Kelsch and ier,[1] have shown the close relation which exists between dysen- and suppurative hepatitis. These two endemic diseases de- e a parallel curve, and in all latitudes any influence which, like famine, etc., determines an increase of dysenteric troubles, will cause an increase of hepatitis.

The two last-named authorities give most convincing statistics in valuable work. In 314 cases of hepatic abscess dysentery was d to coexist in 268. We may, therefore, conclude that dysentery ie chief etiological factor in the production of suppurative he- is of warm countries, and that the heat, the rapid changes of erature, habits of life which are unsuited to the climate, etc., are ly accessory causes. We shall see later in what manner hepatic ess is related to dysentery.

Suppurative hepatitis, however, is usually of secondary and most ently of metastatic origin. The hepatic gland possessing two llary systems, emboli may be either venous or arterial. In the case they occur within the territory of the portal vein, in the nd within that of the hepatic artery. We therefore make a dis- ion between metastatic portal suppurative hepatitis and metas- arterial suppurative hepatitis.

The first, which is the more frequent and possesses the greater cal interest because it may give rise to special symptoms, may used by any purulent inflammatory process in the trunk of the al vein or in the region of its radicles. Thus in operations of soever nature upon the kidney, in suppurating hemorrhoids, in ne and ovarian affections, in the rare cases of suppuration of the reas or spleen, in ulcerations of the stomach and intestines, in urating typhlitis or paratyphlitis, and in purulent inflammation ie portal vein, emboli may be arrested in the terminal branches e vein, being formed either of fragments of necrotic tissue or of or less large masses of pus or even of micro-organisms alone.

Whatever their nature, when they reach and are arrested in the liver they give origin to those anatomico-pathological changes which constitute an abscess.

Intestinal ulcerations are the most important of all these causative factors. Tuberculous ulcerations never produce a suppurative hepatitis, probably because they cause obliteration of the veins. Typhoid ulcers rarely produce hepatic abscess. Dysenteric ulcerations, however, as we have already stated, are the true etiological factors in tropical abscess. But how shall we explain this intimate relation between dysentery and abscesses? Do they result from the same cause, or is one the effect of the other? This question has interested and still interests many investigators, but as yet no definite solution has been reached, neither is the etiology of dysentery fully understood. The bacteriological data of hepatic abscess are obscure and often contradictory. Kartulis in 9 cases of idiopathic abscess in hot countries isolated in 4 the staphylococcus aureus, in 1 the staphylococcus albus, in the remaining 4 he obtained negative results, as did also Netter and Laveran in their investigations.

In abscesses with dysentery Kartulis in 2 out of 13 cases found the staphylococcus aureus, in 1 the albus, in 1 the bacillus fœtidus, and in 8 obtained a negative result. Laveran found complete absence of bacteria in many cases. The same uncertainty prevails in dysentery. Kartulis has succeeded in cultivating upon potato a special micro-organism which causes dysentery in cats to which it is administered, and he believes that this amœba produces suppurative hepatitis by travelling through the radicles of the portal vein and stopping in the radiating capillaries of the hepatic lobules, where it develops inflammation, either by its own properties or because of the digestive detritus and pyogenous cocci which it may contain. Chantemesse and Widal believe in a special bacillus which causes the special anatomical lesions with foci of coagulation necrosis in the liver.

Following out this idea, suppurative hepatitis would seem to be due to the same cause as dysentery, the two affections being merely different localizations of the same infection.

Another theory, which to us seems more reasonable because corresponding better to the clinical history of hepatic abscess, is that dysentery is caused by some special micro-organism, and that it causes secondary development of the abscess, the ulcers opening the way for microbic embolism of the portal vein. This interpretation of the connection between the two diseases has been confirmed by a fact recently and statistically demonstrated, that hepatic abscess is not so frequent as dysentery and that many cases of dysentery occur which are unaccompanied by abscess, which would be difficult to understand if the

diseases were the result of one and the same infection. Another istical fact which to us seems to be of importance and to confirm above-mentioned theory, is that while these two endemico-epidemic ases describe a parallel curve, that of suppurative hepatitis follows of dysentery, with a slight delay. This demonstrates that the bid processes are of successive occurrence and not manifestations he same infection. The micro-organisms which determine sup- tive hepatitis by penetrating through the dysenteric ulcera- s, may be of various sorts; the streptococcus, the diplococcus, bacillus fœtidus, and even the bacterium coli, which is found eat numbers in the intestines, may all play a part, as has been onstrated in a number of cases, although for the most part bacteriological examinations have given negative results. This be due to the fact that if the process is of long duration the ative agents of suppuration disappear, or because they are only onstrable in the walls of the abscess, or most probably because hepatic cells, by some as yet unknown chemical action, destroy yogenic micro-organism or reduce its virulence. The researches auffard and Widal tend to sustain the last theory, which is in dance with that function of the liver which consists in the pro- on of the organism from infection and autoinfection.

rterial metastatic hepatitis occurs with greater rarity than the going; it may originate in any condition in which there is sup- tion in any part of the body, especially such as concerns the (septic endocarditis) or the lungs (gangrene, pulmonary ab- , fœtid bronchitis, etc.), because the embolus, being carried from pulmonary veins into the left auricle and thence into the left icle, may without hindrance reach the network of capillaries in iver, and so pass into the capillaries of the lobules. Here minute obic emboli are found which by contact or by secretory action swelling or proliferation of the vascular endothelium on the one , and on the other precipitation of granular fibrin, stasis of leu- es, and obliteration of the lumen of the vessels, thus causing ss. The ancients noted the relation existing between suppura- processes of the head and hepatic abscesses; but of this and of matters connected with the origin of hepatic abscesses from ial metastasis, we have already spoken in the section on general logy.

ppuration of the bones is a special cause of suppurative he- is, due to the fact that the veins of the spongy tissue, being un- to collapse, tend to form tubes filled with infective matter, from the emboli originate. Finally, Virchow has called the atten- of pathologists to the fact that after intense and prolonged osse-

ous suppuration marasmic thrombi in the vesical and prostatic or uterine plexuses are developed which, owing to the state of general pyæmia, undergo purulent disintegration and give origin to infective emboli in the portal system. In this manner a suppurative process in the general circulation is able to cause an hepatic abscess by means of portal emboli.

More recently Widal [1] has demonstrated that pyæmic abscesses of the liver in puerperal infection originate around the suprahepatic veins. The endophlebitic nodule of infection invades the external walls of the veins, and forms a perisuprahepatic abscess which tends to invade the radiating capillaries of the neighboring lobules, leaving the periportal tissues intact. The microbes might in this way be carried to the liver by the blood and become engaged by a retrograde process in the suprahepatic veins, this being rendered possible by the adynamic condition of the heart and the consequent venous stasis.

Besides the metastatic origin of hepatic abscess, its biliary origin is of importance, and represents a morbid series of events which is not infrequently met with in biliary calculus (pyocholic abscess). The stagnation of the bile, owing to the presence of a calculus obstructing a large or a small biliary duct, forms a condition most favorable to a secondary infection of intestinal origin; this in time will, at the point where the calculus has produced more or less marked alterations of the duct, give rise to a suppurative process which will suffice to produce an abscess. We shall later consider the subject more in detail. If the calculus is latent, as may easily be the case, the etiology of a suppurative hepatitis may be obscure, although it is merely the expression of an infection secondary to the retention of bile and to the lesion produced by the presence of the stone.

Finally, purulent hepatitis may occur by diffusion from neighboring organs; this takes place in destructive disintegrating processes, more especially in cancer of the walls of the stomach, the detritus travelling along preëxisting adhesions and becoming diffused into the hepatic tissue.

Pathological Anatomy.

The study of the anatomico-pathological lesions of suppurative hepatitis is as complicated as the etiology is varied. We will first describe the pyæmic liver in which suppuration is more or less diffused, and which is found in pyæmia or septicæmia due to the most varied causes.

The liver is large, softened in consistence, with a surface which is

sh in color, and often irregular by reason of protuberances which rarely larger than an almond, and of a grayish-yellow color; if they it they give issue to a certain amount of thick and creamy yellow

If an incision is made across the diameter of one of these pro- rances we shall find upon the cut surface scattered ovoid or rical spaces, from a pinhead to a pea in size, and filled with

A large abscess is rarely found in the pyæmic liver, because the se of the disease is short, but occasionally the fusion of several nese small abscesses results in a large collection of pus which, ig to the manner of its formation, is of irregular and broken out-

By studying the successive anatomical lesions which give rise to e small metastatic abscesses, we may almost understand the ogenic history of an hepatic abscess. The stoppage of the pus obes in the capillaries of the hepatic lobules and their subse- t reproduction cause a certain amount of thrombosis, and the tic cells, probably through a specific chemical action, become d with granulations, the nucleus disappears, and coagulation osis occurs, followed by complete destruction. With the subse- t emigration of leucocytes from the engorged capillary vessels ormation of pus begins.

Not including suppurative hepatitis of metastatic origin, hepatic sses have, from an anatomico-pathological point of view, been ed into three varieties: *single phlegmonous abscesses*, *multiple us abscesses*, and *alveolar abscesses*. Kelsch and Kiener named the two, and Chauffard the third.

Single phlegmonous abscess is the variety ordinarily met with in the urative hepatitis of hot countries, related to dysentery. The sses are sometimes superficial, at other times deeply seated, and nore often found in the right lobe than in the left, which is prob- owing to its greater size.

The size of the abscess depends upon the stage of its progress and the greater or less amount of resistance possessed by the sur- ling tissues, and may range from that of a hazelnut to that of an , or even to that of a fœtal head. Some cases of enormous ab- have been reported. Toman, of Liverpool, describes a case in the purulent collection weighed ten pounds, causing almost lete destruction of the right lobe. As a rule the pus is *bonum et bile*, but in some cases it may possess a fetid, putrid odor. In present stage of our bacteriological knowledge, we cannot tell ner this is due to secondary infection of intestinal origin, or to herent and intense virulence of the pyogenous micro-organism. lor the pus may be green or even chocolate-colored, owing to the

presence of bile from ulceration of some biliary duct. If the blood-vessels are eroded the pus becomes hemorrhagic.

A microscopical examination of the pus will show that it is formed of pus corpuscles, which have for the most part undergone fatty degeneration, of free fat globules, giving it the appearance of a true emulsion, of a granular detritus formed by parenchymatous gangrene, of micrococci (which may, however, be entirely absent for reasons above mentioned), of parenchymatous liver cells which have undergone fatty degeneration, and sometimes of typical plaques of cholesterin.

The abscess walls are often anfractuous; they are formed of embryonal tissue analogous to the tissue of the granulations, and covered with shreds of gangrenous tissue. In a more advanced stage, inflammatory reaction in the surrounding interstitial tissue causes the formation of a fibrous membrane around the pyogenous membrane, which circumscribes the abscess and prevents its further development.

The hepatic tissue in the vicinity of the abscess is usually found to be pale, softened in consistence, and undergoing fatty degeneration. Lepidi-Chioti describes a rare case in which the whole liver had undergone amyloid degeneration. The situation of the abscess is of influence in determining other morbid conditions. When upon the inferior surface, the pressure upon the large biliary ducts and portal vein may cause stasis, resulting in either icterus or ascites. When superficially situated, or when a deep-seated abscess reaches the surface, it causes perihepatitis with subsequent adhesions to the neighboring organs. This is a desirable occurrence, as opening of the abscess usually follows and may lead to a complete cure, as we shall see later. When, on the other hand, there are no adhesions, and the abscess opens into the peritoneal cavity, death is the usual result, from acute purulent peritonitis.

Fibrous abscesses are small and multiple, varying in size from a walnut to a pigeon's egg, and in number from three to twelve or more. They form rounded nodules which are whitish in color, and not very soft in consistence, the purulent softening occurring only in the centre. The pus is creamy; the walls are broken, hard and coriaceous, sometimes infiltrated by lime salts. A histological examination will show the walls to be formed of fibrous tissue, which is stratified and intersected by numerous vessels. The fibrous structure which distinguishes this variety is probably due to an intense inflammatory process of the hepatic connective tissue. The development of the abscess may be completely arrested, and it may remain as a foreign body in the parenchyma of the organ, often invaded by a deposit of lime salts

surrounded by a layer of fibrous tissue, without causing morbid nomena of any importance.

lveolar abscess remains to be described. According to Chauffard, iginates in the deep hepatic tissues in the middle portion of one of lobes, and develops towards the surface in the form of an infarct a central apex and a broad base almost circular at the periphery. section we find a spongy, cavernous abscess with small areolæ d with a pyogenous membrane, milky white in color, and contain- pus which may be thick and creamy or green and viscid, resem- g the muco-pus of advanced coryza. From Chauffard's histo- cal researches it would appear that these abscesses are the result artial suppurative angiocholitis, and are caused by the migration ugh the biliary passages of a septic germ which may travel up- ls and induce the lesions of areolar abscess around and above tever point it becomes fixed upon. As may be readily understood, form of hepatic abscess is difficult to cure, even by surgical inter- ion, for, owing to its areolar construction all of the pus cannot be uated through the incision, nor can the abscesses be thoroughly ned out. It is also extremely difficult to reach the upper surface ie liver with a knife.

Symptoms and Course.

When suppuration of the liver occurs in connection with a general mic process, it may give rise to no noteworthy symptoms, espe- y when the patient's general condition from its more threatening re absorbs the physician's whole attention. Even when the atic disturbance constitutes a true morbid entity, it may pass un- rved. Thus there is frequently a latent period of the affection, should death occur from an intercurrent disease, the abscess of liver appears at the autopsy merely as an accidental occurrence. ther cases the suppurative hepatitis may run its course without ing any symptoms which draw the attention of either patient or sician to the liver, but be accompanied by grave general symp- . An intermittent fever of the quotidian type, occurring at a d hour and accompanied by severe chills, may lead to a diag- s of acute malarial fever, all the more readily that this disease d in a measure account for any swelling of the liver or spleen h might be present. In some patients, however, the course of lisease is absolutely similar to that of typhoid fever; the high of a remittent type, the swollen abdomen, the inconspicuous or of the liver, the swollen spleen, the weakness, diarrhœa, de- m, adynamic heart action, generalized convulsions, etc., suggest

typhoid fever rather than a lesion of the liver. Finally, in a third series of cases, the depressed general condition of the patient, the progressive emaciation, the quotidian fever, and the night sweats may, if the sputum have not been examined and especially if there be a secondary bronchitis, lead to the supposition of pulmonary disease; so much so that the ancients gave to this variety of the disease the name of hepatophthisis.

Hepatic abscess which has a chronic and latent course may cause acute morbid symptoms, to be accounted for only by the presence of suppurative hepatitis. Examples of this are given in the rapid development of fatal purulent peritonitis, purulent pleurisy, and the presence of vomica when there have been no previous pulmonary symptoms, diarrhœa of fæcal matter mixed with pus, etc. Excluding these cases, which are of infrequent occurrence, suppurative hepatitis may present symptoms which attract the physician's attention at once to the liver. Its course is, however, by no means always the same; we may have an acute, subacute, or chronic form.

The *acute form*, which is rare in our climate, but which may be said to constitute the typical form of hepatitis in hot countries, is ushered in by symptoms almost identical with those of the infective diseases. A state of general malaise is followed by initiatory chills and a fever which is sometimes remittent and sometimes intermittent, which is higher in the evening and at night. At the same time the right hypochondrium becomes painful spontaneously or under pressure, and the patient also feels a sense of tension and of weight in the hepatic region. By reflex action through the phrenic and vagus nerves, there may be some dyspnœa or even a slight, dry, hard cough, which increases the pain (hepatic cough of Galen).

As the pain accompanying these phenomena may easily be taken for that of pleurisy, the fact that pressure upon the liver increases it, the increase in size of this organ, and the absence of objective symptoms in the right lung and right pleura will be of great importance to the diagnosis. To these symptoms may be added the vomiting of bile and a slight degree of icterus from retention by pressure upon the bile ducts or from concomitant catarrh of the ducts, but they are not constant and may indeed be entirely absent. If this early jaundice occurs with a high fever, we may easily mistake the disease for acute yellow atrophy; in suppurative hepatitis, however, we do not have the cutaneous or nasal hemorrhages, the albuminuria, or the adynamic condition which make their appearance early in the course of acute parenchymatous hepatitis.

The symptoms go on with increasing severity for eight or ten days; the skin becomes covered with profuse perspiration, the fever

sists, the general condition becomes worse, and if death does not ult at this stage, the hepatic abscess will form. The general symp- s then undergo a slight improvement, the fever diminishing and oming intermittent with exacerbations that last a few hours only, jaundice almost always disappearing, and the pain diminishing, the local symptoms increase in severity. It is doubtful whether purative hepatitis can result in recovery without the formation of bscess, as some clinicians believe; their theory is doubtless due rrors in diagnosis.

The *subacute form* occurs more frequently than the other in our ate. The symptoms are similar to those of the acute variety but ss intensity; there is less elevation of temperature, the pains are so severe, vomiting is rare, and the local manifestations of ab- s formation are of more gradual development.

The *chronic form* is the most insidious and has been shown to ess the greatest clinical variations, for it is the form which runs most latent course and is attended by phenomena which may y divert the physician's attention to other organs. In clear cases, ever, the symptoms are of the same nature as those of the acute subacute varieties, but occur less tumultuously and lead more ly to the formation of a collection of pus.

Whatever the course of the disease, when the hepatic abscess is formed (and its presence is made clearly evident by the signs), objective symptoms as can be found in the hepatic region are hief importance and relate principally to the increased size of rgan.

greater fulness in the right hypochondrium than in the left, a pronounced curve of the lower ribs, and protrusion of the lower costal spaces are signs which may be verified by simple inspec- and serve to demonstrate the presence of a tumor. In some cases nferior border of the liver can be seen projecting and its respira- movements may be followed. In some rare cases, when the ab- has become superficial and the thoracic wall is on the point of g invaded by the suppurative process, the latter will appear to be natous, and the subcutaneous veins will be markedly developed. pleural boundaries of the liver are enlarged and the area of liver ss is increased upwards, so much so that Sachs consider this to diagnostic sign. Frerichs considers it of importance to define uperior limits of the area of dulness, which presents a more or marked appearance of a swelling, and this because of the abscess oped upon the diaphragmatic surface of the liver. In some cases lulness reaches to the third or second rib, and then of course ission will not possess the diagnostic value above referred to.

By palpation we may appreciate perihepatic friction when there is complicating perihepatitis; and we also increase any pain which is present, or cause its appearance. This hepatic pain is extremely important in the diagnosis, and it is in the cases under discussion that it is apt to radiate to the shoulder. Besides showing the increased volume of the liver, which usually extends beyond the transverse umbilical line, and may, if the abscess is developed in front or below, reach to the iliac crest, palpation will enable us to appreciate the indurated elastic consistence of the surface of the liver, which is usually smooth and uniformly projecting. But that which more especially indicates the presence of an abscess is the sense of fluctuation, which, to tell the truth, is but rarely found, and when found is seldom clear and distinct. Occasionally we meet with an extreme amount of tension in the right rectus abdominis muscle; this is a reflex phenomenon caused by the examination, although Boulanger holds that it is due to the hepatic tumor.

Not only objective symptoms but important functional disturbances of the liver may be found. Jaundice may occur, but is not very frequent, the statistics showing that it is found in one-fourth of the cases. It is, as a rule, not severe (except when the result of compression or occlusion of the ductus choledochus), but on the contrary so slight that we call it "icteric pallor." In these cases of icterus the urine usually does not contain bilirubin, but does contain a certain amount of urobilin; in other words, the jaundice is due to urobilin from altered hepatic function. It was formerly believed to be an hæmatogenous icterus from pyæmic infection.

Ascites is even rarer; it is produced by compression of the portal vein, or by some possible peritoneal complication. Functional disturbances of the digestive system are frequent, as they always are in any disorder of the liver, but they possess no special characteristics which would aid in the diagnosis; a coated tongue, a bitter taste in the mouth, anorexia, diarrhœa which in the beginning is often due to polycholia, and vomiting, especially when the hepatic peritoneum is involved, are some of the symptoms observed.

The pain, which is increased during the respiratory movements, and the elevation of the diaphragm, which narrows the respiratory field, may in some exceptional cases cause a dyspnœa entirely out of proportion to the degree of fever present. Of the greatest interest from a diagnostic point of view is the behavior of the body temperature. It may be normal, or there may be merely a slight elevation in the beginning of the disease. More often, however, there is fever present, of varying elevation and type in the different cases. In one there will be slight elevations of temperature in the evening; in another there

be intermittency with a true pyæmic type—chills and intense rigor wed by a rapid elevation which may reach as high as 40–41° C. '–105.8° F.) and which will last for hours and then fall rapidly be followed by profuse sweating, which may give rise to cutane- esions (miliaria, pityriasis tabescentium, falling of the hair). It ables intermittent malarial fever, but does not yield to treatment inine. In other rare forms, the fever assumes a continuous re- nt type with considerable evening exacerbations. The pulse is what frequent, small, and soft.

nportant diagnostic signs may be found by a daily examina- of the urine. In the first stage of the disease there is a certain ase of urea, which is dependent upon inflammatory irritation in iepatic cells. In the second period, that is to say, when the n of purulent foci has occurred from destruction of a consider- portion of the parenchyma, we have hypoazoturia. This is a tom which has always been of especial interest to us, and which in affirm to be constant, as we have found it in every case of ic abscess in our practice, and regard it as a valuable aid to osis. It is of the more importance because the elevation of erature in this affection should produce an increased amount of for which reason the inverted relation between the thermic con- n and the urea must, in connection with other symptoms of liver le, be of great practical value. In the calculation of the daily nt of urea the quantity of urine eliminated in the twenty-four should always be exactly estimated, for as the urine is usually ed in amount, this symptom might not otherwise be appreciated full value. Nutrition is greatly disturbed in suppurative hepa- owing partly to the deranged liver functions, but chiefly to the al pyæmic condition. The panniculis adiposus becomes scanty ount, the skin pale and delicate, the muscles flaccid, the bony iyses protrude, and the patient little by little becomes the victim hexia.

ie position assumed by these patients for the relief of their suf- ; deserves notice. As a rule, when there is sharp pain they lie e right side, the trunk supported by pillows and bent forwards, he right thigh flexed in order, as far as possible, to relax the ninal walls and thus to diminish pressure upon the liver.

Results.

spontaneous cure of suppurative hepatitis may occur from reab- on of pus. This has been proved both by clinical experience y pathological anatomy; the first has shown us cases in which

a collection of pus, clearly appreciable by physical examination, has entirely disappeared without opening outwardly, and the second has demonstrated the existence of true cicatrices in the hepatic parenchyma, or else of encysted masses of dense caseous matter. These favorable results are, however, extremely rare, in fact absolutely exceptional. As a rule, if death do not occur from a marasmus or from pyæmia, we have the usual result in all cases of abscess, an external opening or one into neighboring organs.

The external opening of an abscess is the most favorable and least frequent result, and usually occurs when the abscess is situated anteriorly. From the perihepatitis caused by the disease adhesions are produced between the liver and the parietal peritoneum, and following the ulcerating process extends progressively through the adhesions until the skin is reached, becomes inflamed, ulcerates, and allows egress to the pus. The physical signs which may lead us to hope for this result are œdema, redness, and gradual attenuation of the skin. But even when the abscess opens in this manner recovery does not always follow; infection of the abscess cavity, erysipelas, the continuation of suppuration causing marasmus, may occur and lead to a fatal issue. In some cases the opening does not take place anteriorly, but into the axilla, perhaps the umbilicus or the inguinal ring. Gioffredi observed a case in which a perforation occurred through previous pleuritic adhesions in the infrascapular region in the eighth intercostal space. More frequently the pus is evacuated into the abdominal or the thoracic cavity. In some cases peritoneal symptoms of some importance arise, a few days later the abscess opens into the abdominal cavity, and death occurs from septic peritonitis. The abscess opens less frequently here than elsewhere, however, because the perihepatitis following it usually produces adhesions to the other organs.

Exit of pus into the stomach will cause somewhat intense pain, the vomiting of purulent matter sometimes mixed with blood from the erosion of blood-vessels in the gastric wall. When the opening occurs into the small intestines or the colon, there is profuse diarrhœa of fæcal matters mixed with pus. This, however, is also a symptom of perforation of the biliary passages with a subsequent discharge of pus into the intestines.

Sharp pain in the region of the kidneys together with the excretion of pus-filled urine indicates opening of the abscess into the right renal pelvis. A cure may follow these various results, especially the opening of the abscess into the colon, but they may instead cause serious complications in the intestines or in the urinary apparatus, or secondary infection and sepsis of the abscess cavity.

The pus may work into the ascending vena cava, the portal or the rahepatic veins, causing emboli which are usually followed by den death. Less frequently, the abscess may rupture into the racic cavity. Adhesions between the liver and the diaphragm, foration of the latter, followed by an opening into the pleura, give to sharp lancinating pain and to all the symptoms of purulent irisy. If there be adhesions between the diaphragmatic and vis-l pleura from adhesive pleurisy consecutive to diaphragmatic tonitis, the opening of the abscess will occur into the lungs, and e will be the expectoration of dense matter, chocolate-colored inged with dark green from the presence of bile, or else a true noptysis. The signs of pulmonary infiltration will usually pre- the opening of the abscess, and certainly after this has occurred nay by auscultation find signs of the presence of cavities, while ne same time the liver will be reduced in volume, the abscess ling will diminish notably, the fever will be lowered, and a slight rovement will be noticed in the patient, who may finally recover. the hepaticopulmonary fistula may persist, or if it closes it may dily reopen to allow of the exit of newly secreted pus from the ess cavity. The influence of decubitus upon expectoration is acteristic of this fistula; it is almost entirely absent if the patients n an erect position, but when they assume the horizontal it be- s so abundant that it is almost impossible for them to lie down. after formation of the fistula a cure may occur; the expectora- gradually diminishes and tends to become mucous, the fever dis- ars, the appetite and general nutrition improve, until finally remains to remind one of the disease. Often, however, the re- is fatal from the bronchopulmonary lesions produced, with a equent septic or gangrenous process, from marasmus, or from cæmia due to infection of the abscess cavity.

inally, the opening of an abscess of the left lobe of the liver the pericardium causes an intense purulent pericarditis or im- ate cardiac paralysis.

he *duration* of suppurative hepatitis is as variable as the course; y last for a few days only, or, becoming chronic, go on for years.

Prognosis.

ne prognosis will depend upon the form of disease present, and the occurrence or non-occurrence of the various results mentioned . In the case of a phlegmonous, unilocular abscess, therapeu- easures will be more readily effective. On the other hand, in se of metastatic abscesses we should give the chief considera-

tion to the primary disease. In general the prognosis should be very guarded, as the mortality is high. We cannot hope for a spontaneous cure, but should always resort to efficacious surgical measures.

Diagnosis.

In some cases the diagnosis of suppurative hepatitis is easily made, but in others it presents serious difficulty. In the early stages it may be confounded with active hyperæmia, especially as the latter is also common in hot countries; moreover, as hyperæmia is the principal anatomico-pathological condition in the beginning of hepatitis, the differential diagnosis cannot be made until a later stage.

Diagnostic errors in regard to malaria not infrequently occur when there is fever of an intermittent type accompanied by hypertrophy of the spleen. The fact that it does not yield to quinine will clear up the diagnosis.

The absence of pulmonary lesion, and a negative result of bacteriological examination of the sputum will serve to distinguish the affection from pulmonary tuberculosis.

Other affections of the liver itself may lead to a mistake in diagnosis. Suppurative echinococcus cysts present absolutely analogous symptoms, because they also constitute a suppurative process in the liver. The course of the disease alone and the general history will indicate the nature of the trouble.

Abscess of the liver may also be confounded with non-suppurative echinococcus cysts, with soft sarcoma presenting pseudo-fluctuation and with cystic dilatation of the gall-bladder. In these cases, therefore, it is absolutely essential to resort to exploratory puncture which alone can decide the question of an operation; it is unattended by danger even to a normal liver if performed under strict antisepsis.

Fluctuating projections in the hepatic region may be due to tuberculosis of the ribs or of the vertebral column, or to suppuration of the abdominal walls. But in these cases there is no respiratory displacement of the tumor, which is only moved upwards a trifle, and in the first two diseases there is always localized pain in a rib or in the spine. When suppuration of the abdominal walls is suspected, it is well to follow Sachs' advice and thrust a long needle into the tumor. If the suppuration is in the liver, the respiratory displacement of that organ will cause the needle to describe a to-and-fro movement, the free point being lifted during inspiration and lowered during expiration; in suppuration of the abdominal walls the needle as a whole will move forwards during inspiration.

Hepatic abscess may sometimes be confounded with empyema; the

rse taken by the superior limit of dulness may assist in the diag-
is.

Finally, when masses of pus are suddenly expelled through some
nnel or other to the exterior, we must be careful to ascertain
ther there is any inflammatory process in the organs through
ch the supposed abscess of the liver has discharged itself. In
s of discharge from the air passages the sputum should be care-
y examined, as it may contain hepatic cells or biliary pigment.

Treatment.

Therapeutic agents are of great importance in both the first and
nd stages of suppurative hepatitis. But while in the inflamma-
period medicinal measures may give relief, in the suppurative
od abdominal surgery alone is to be relied upon, and its value is
tantly becoming better appreciated.

The physician's great object should be to reduce the inflammatory
ess as much as possible, for which reason antiphlogistic treat-
t is the only rational one.

Local bathing with ice water, or the application of an ice-bag to
hepatic region, local bleeding, and wet-cups induce a certain
unt of ischæmia of the organ, and at the same time relieve the
, which at this stage is one of the most prominent symptoms.

To reduce the hyperæmia, we must use every means possible to
e depletion of the portal system; four to eight leeches, according
ne indications, should be applied to the anus, and relaxing pur-
es administered, such as the sulphate of magnesium, calcined
nesia, Hunyadi János, etc.

To improve the biliary circulation and at the same time produce
ced serous transudation from the radicles of the portal vein, we
give calomel, aloes, or any other cholagogue cathartic. A milk
is of paramount importance from its beneficial influence upon
omplex mechanism of this grave hepatic disorder. The patient's
ngth must be sustained by every possible means, and we may en-
or to reduce the fever by quinine or other antipyretics, such as
pyrin, phenacetin, etc., given with all possible precautions.

The abscess once found, however, no reliance should be placed
any measure other than its opening and evacuation, even aspira-
of the pus will be of little use.

It is not our intention to trespass upon the field of surgery by a
ission of the various methods which have been proposed for
ing the abscess. We will merely state that owing to the vast
ress made by abdominal surgery, the best method consists in a

large opening directly upon the purulent focus followed by thorough washing out with antiseptic solutions, and drainage according to Lister's method. The manner in which the opening is made will of course depend upon the situation of the abscess; thus, when it is seated upon the convex surface of the liver, we have recourse to resection of the inferior portion of the thorax without opening into the pleural cavity; in an abscess situated postero-superiorly, we follow Kartuli's directions and make an incision through the pleura, resecting one or two ribs to the extent of 6-7 cm. (about 2½–3 in.).

We should never defer treatment in the hope of a spontaneous cure.

Pylephlebitis.

Inflammation of the portal veins constitutes pylephlebitis.

A consideration of the phlogistic processes which may occur in the whole course of the portal vein from its radicles to the intrahepatic capillaries would take us into the wide subject of the various forms of cirrhosis, many of which are due to primary lesions of the veins. We will therefore confine our attention to inflammatory lesions of the trunk of the portal vein, which in point of fact constitute a pathological entity possessing a special symptomatology and course.

At the time when Stahl's theories were in the ascendant and many hepatic lesions were attributed to abdominal plethora, inflammation of the portal vein held an important place. Completely forgotten for a while, it was again taken up by Frerichs, whose descriptions of it were so exact as to serve as a basis for the most recent investigations.

There are two varieties of this affection, the *adhesive* and the *purulent*, the first being formed by a thrombosis leading to occlusion of the vein, the second by a specific phlogistic process resulting in suppuration. The two processes, differing greatly in severity and in their etiology and their clinical course, deserve a separate description.

Adhesive Pylephlebitis.

(Pylethrombosis; Occlusion of the Portal Vein.)

Etiology.

Thrombosis and occlusion of the portal veins may be produced by two causes, the same which act in all cases of coagulation of the blood, namely, slowing of the circulation and changes in the endothelium lining the blood-vessels. The former acts indirectly by causing deficient nutrition of the endothelium. As a rule, the etiology of thrombosis is twofold.

ll retardation of the circulation in the portal vein, of long dura-
or great extent, whatever the pathogenesis, may result in coagu-
ı of the blood in the vein. But slowing of the circulation in
ein may depend upon a slowing of the systemic circulation from
ic disease of the heart, or upon hydraulic disturbance of the
l system. In the first class of cases we have the various
lar lesions, in especial those which cause the greatest disturb-
in the venous circulation, that is to say, lesions of the mitral
, muscular degeneration, or even the hypokinesis accompanying
cachexia (marantic thrombosis). In the second we find those
ions which interfere with the portal circulation by either in-
c or extrinsic, hepatic or extrahepatic compression of the trunk
any of the numerous branches of the portal vein. Cirrhosis,
r, or abscess of the liver, distention of the bile ducts from
, the pressure of a calculus upon a large branch of the vein,
ing of the lymphatic glands in the hilum of the liver, chronic
nitis with the production of cicatricial bridles or adhesions of
er or less strength which may constrict the trunk of the portal
tumors of neighboring organs, such as cancer of the pancreas,
mentum, the retroperitoneal glands, etc., are the more or less
ent causes of adhesive pylephlebitis.

primary lesion of the endothelium of the blood-vessels with the
cutive production of thrombosis constitutes true pylephlebitis.
the result of an infective process, more especially of syphilis,
ı as we know is liable to affect the walls of the blood-vessels.
ppel, out of thirty cases of hereditary hepatic syphilis, found
ıbosis of the portal vein in three.

ecent investigations, such as those of Widal, Vasquez,[1] and
s, tend to demonstrate that the thrombosis caused by cancer,
culosis, etc., is in relation with infective phlebitis from parie-
cterial emboli rather than with the cachexia or the cardiac mar-
s.

Pathological Anatomy.

a few rare cases a complete thrombus of the portal vein may
ıe organized, transformed into connective tissue, and attached to
nterior of the vessel, so that the vein may become transformed
connective-tissue or fibrous cord.

ore frequently, if we cut into the portal vein, we shall find parie-
ırombi which cause great narrowing of the lumen, and which
nvolve the trunk, the branches, and even the intrahepatic rami-
ns, and which may remain limited to some one of these por-
of the circulatory system. The appearance of the thrombus

varies according to its age. When of recent formation it is of a brownish-red color, whereas later it is harder, more intimately attached to the walls, and whitish-yellow in color. The vessel walls undergo certain changes, which may be the consequences of the thrombus, but which in many cases are of primary occurrence. There may be thickening, fatty or calcareous degeneration of the walls, and cloudiness or roughening of the intima to a greater or less extent.

Lesions consecutive to thrombosis are found even in the liver itself. A proliferation of the periportal connective tissue, followed by cicatricial retraction, occurs, constituting a true perilobular venous cirrhosis, a fact which has been demonstrated by histological studies and practical experiments (ligature of the portal vein) by Solowieff, Foà and Salvioli, Leroux, etc.

Owing to the disturbed circulation considerable stasis occurs in the spleen, often accompanied by proliferation of the connective tissue, a stasis which causes hemorrhagic spots on the intestinal walls and serous transudation into the peritoneal cavity.

Symptoms and Course.

The etiological factors which lead to thrombosis often consist of grave disorders, with a marked symptomatology relating to the abdominal organs, so that occlusion of the portal vein may occur and yet escape notice. For instance, during the course of cirrhosis of the liver, of a grave cardiac affection, or of a malignant neoplasm of the abdominal organs, a rapid increase of the ascites with severe gastrointestinal disturbances may be the only symptoms leading one to suspect the formation of a thrombus.

In some cases, however, when the thrombus is almost of primary origin the symptoms caused are clear and unmistakable; as, for example, when it depends upon syphilitic infection or upon connective-tissue bridles due to chronic peritonitis.

The symptoms are those due to alterations in the abdominal circulation, that is, to stasis in the radicles of the portal vein. Of chief importance is the ascites, which, however, may be slight in amount, provided the collateral circulation, owing to slow and gradual development of the occlusion, be well established. The effusion will be aided by profuse gastric or intestinal hemorrhages, which disturb the portal circulation. Of diagnostic importance is a great amount of ascites (which is due to extensive stasis) and the rapidity with which it appears and increases. For instance, if the ascites become marked very soon after an abdominal paracentesis, we are justi-

in concluding that there may be some impediment to the portal ulation, more especially thrombosis.

The network of abdominal veins may be seen as the *caput medusæ*, development of which is in inverse ratio to the ascites, as it rep- nts the most important physiological compensation in disturb- es of the portal circulation.

Stasis in the spleen, an organ which, as we know, discharges its d into the portal vein, is also very noticeable, and will be absent if there be fibrous thickening of the capsule from chronic peri- tis, or again if there be amyloid degeneration of the parenchyma ccount for the increased size of the organ.

As a consequence of the obstructed portal circulation and the re- ing stasis in the gastric and hepatic circulation, we have gastror- gia and enterorrhagia, which may be profuse enough to cause h. Digestive disturbances appear, such as loss of appetite, ea and vomiting, and profuse serous or even sanguineous diar- a.

Jaundice is rarely observed; even when the portal circulation is pletely abolished it seldom appears. If the thrombosis be of duration we may have symptoms indicating reduction of the me of the liver from the perilobular cirrhosis which sometimes ws thrombosis. The urine will be scanty, rich in uroerythrin urates, and nearly always deficient in urea.

Pylephlebitis is sometimes acutely ushered in by sharp pain in right hypochondrium and by abdominal sensitiveness to palpa- ; but, as a rule, it arises in a slow and latent manner and is un- gnized because the symptoms which it occasions are also those e primary disease.

ts course may be short, lasting a few weeks only, especially when curs as a complication of a preëxisting affection, but it may last ears, as when it is of syphilitic origin. Alexander reports a case h lasted six years. The fatal result usually occurs with symp- of marasmus, but may be due to intestinal or gastric hemor- es.

Diagnosis.

he diagnosis is always difficult. The chief errors occur in re- to cirrhosis of the liver and to peritoneal affections, such as culosis, carcinoma, and chronic serous peritonitis, the more ly that it may be a result of these diseases. As aids to the ential diagnosis we have: 1. The etiological factor which we find in any given case, and the exclusion of causes which act to uce the condition, such as alcoholism, malaria, etc.; 2. The

degree of serous transudation in the abdominal cavity and the absolute mobility of the abdomen; 3. The rapid production of ascites.

The *prognosis* is unfavorable. We can only hope for a slow, chronic course of the disease.

The *treatment* is that of atrophic cirrhosis of the liver.

Purulent Pylephlebitis.

Purulent inflammation of the portal vein is chiefly related to suppurative or ulcerative lesions found in the radicles of the vein, from which source only the streptococcal infection, which is the cause of the disease, can reach the trunk and cause specific anatomico-pathological alterations.

Yet the immediate cause of the affection may be found in typhlitis, perityphlitis, or appendicitis, or more rarely in dysenteric ulcers, hemorrhoidal ulcers, or gastric and intestinal ulcers of the most varied nature; in ulcerated carcinoma of the stomach and duodenum, abscess of the spleen, encysted peritonitis, inflammations of the pancreas, mesentery, or epiploon, suppuration of the retroperitoneal glands, and suppurating ovarian cysts.

Infection may occur by extension of the process from primary affections in the liver, the peritoneum, or the neighboring organs, but this is rare. Thus ulcerations of the biliary ducts from calculi, abscess, or suppurating cysts of the liver, acute or chronic, circumscribed or general peritonitis of a purulent character, and cancer of the stomach or œsophagus may be causes of the disease.

Suppurative inflammation of the umbilical vein in the newly born may cause a purulent pylephlebitis, which may also occur in the absence of such inflammation from the passage of specific germs to the portal vein, no trace of the germs being found at the point of entrance. In exceptional cases a primary pylephlebitis has been noted.

A case has been reported in which a fish bone worked its way into the portal vein, causing purulent inflammation in that situation, and another in which an iron filing behaved in a precisely similar fashion.

Pathological Anatomy.

The anatomical lesions are those ordinarily found in suppurative phlebitis.

The internal coat of the vein is brownish-red or yellowish-gray in color, thickened and presenting vegetations, and infiltrated with hepatic cells; the middle and external coats and the periphlebitic connective tissues are similarly affected. The diseased vessel is harder

thicker than normal; upon section its lumen remains open and
vs the contents to be composed of a grayish, purulent, ichorous
, sometimes from the presence of blood resembling urinary sedi-
t. These structural changes are sometimes limited to a part
ıe trunk or a few branches and sometimes extend throughout the
e portal system and the veins through which the infection was
ed.

mportant lesions are always found in the liver, consisting prin-
ly of the collections of pus, which are usually small, but multiple
generally disseminated. Hepatic abscesses may be either em-
or parietal in origin. Small purulent particles or micro-organ-
alone, thrown off from the point of injury, may produce emboli
e hepatic parenchyma, and thus cause an embolic abscess. Prop-
on of the process from the intrahepatic branches to the paren-
ıa gives rise to the so-called parietal abscesses. Cornil and
ier thus describe the successive steps in the process: infiltration
e walls with leucocytes, coagulation of the blood, perforation of
alls, formation of the abscess.

arely, metastatic abscesses of the other organs are formed by ex-
on through the fine branches of the hepatic capillaries.

Symptoms and Course.

ppurative pylephlebitis is insidious in its onset, the symptoms
masked by those of the causative disease. The patients al-
affected with the primary trouble are seized with severe chills,
ı are followed by fever and profuse sweating; the fever may be
tent or intermittent in type, quotidian, tertian, or atypical. It
s somewhat from Charcot's intermittent hepatic fever, which
vs a more regular and constant course. A more or less intense
arises in the vicinity of the right hypochondrium or of the epi-
ium, and may be spontaneous or perceived only upon palpation.
he liver becomes voluminous and sensitive to the touch, the
n is enlarged from blood stasis or from general infection. Jaun-
s almost always present, there is a dark coloration of the urine,
marked reduction in the amount of urea is noted.

scites does not usually occur, and when it does it is never ac-
anied by development of the venous circulation of the abdominal

he gastrointestinal disturbances are very marked, and consist of
xia, bilious vomiting, and diarrhœa, which is bloody or dysen-
Sometimes a peritonitis-like condition occurs (hiccough, me-
m, severe pain in the whole abdomen, cardiac adynamia). The

patient's general condition becomes serious; there is great depression, sometimes delirium which resembles that of a severe attack of typhoid fever. Occasionally there will be signs of a *dissolutio sanguinis*, more or less extensive hemorrhages being produced upon the skin and mucous membranes.

The duration of the disease is nearly always short, the process usually being acute. There may, however, be a duration of several months with a subacute course, and in this case remissions will occur which are very deceptive (Leudet).

Death is the inevitable result, and occurs either from a gradual loss of strength or following general septic infection.

Diagnosis.

Important guides to diagnosis are: The etiological data (the existence of pylephlebitis not being admissible without sufficient and evident cause), a painful tumor of the liver, enlargement of the spleen, fever of a suppurative character preceded by chills, jaundice, and a grave general condition.

The conditions most frequently confounded with pylephlebitis are abscess of the liver, the colic of biliary calculi, and acute malarial infection. In regard to abscess or to suppurative hepatitis with diffuse and multiple foci, the most frequent cause of errors in diagnosis, the enlargement of the spleen and the marked gastrointestinal symptoms are the chief symptoms which point to purulent pylephlebitis.

It may be distinguished from biliary colic by the absence of the specific symptoms of this form of pain, the enlargement of the spleen, the course of the disease, the previous history, the absence of intestinal acholia, the type of the fever, and the rapid emaciation. Icterus, which is nearly always present in suppurative phlebitis, upon the one hand, the constant and decided character of the fever and the marked results from the administration of quinine noted in malaria upon the other, will aid in the differential diagnosis between the two diseases.

Treatment.

There being no etiological or pathogenic indications, treatment must be purely symptomatic. To keep up the patient's strength by a fortifying and suitable diet (milk, broth, grapes, meat, etc.) and by tonics, to reduce the attacks of fever by hypodermatic injections of quinine, to combat the diarrhœa by opium, and to mitigate the patient's sufferings by morphine, these are the only methods of treat-

it open to the physician, nor can he hope by these means to avert fatal termination of the disease.

CIRRHOSIS OF THE LIVER.

General Considerations and Classification.

The chronic inflammatory lesions usually included under the ie of cirrhosis hold an important place in the pathology of the r.

The name originated with Laennec, who used it to indicate the racteristic color of some forms of this lesion, and was afterwards ined in spite of the fact that it does not express any characteristic logical or anatomico-pathological condition of importance.

Although the Greek and Roman physicians may have known the rious effect of alcoholic drinks upon the liver, we may say that study of the hepatic diseases which they induce belongs to mod- times, as it began in the early part of this century under Baillie 3) and Laennec (1819).

It is true that in Fernel there are some allusions to the action of e in causing "scirrhus" of the liver, in Vesale some notes upon phy of the liver in alcoholics, and that Morgagni in speaking of psy describes reduction in size of the liver accompanied by ruction from compression of the small intrahepatic vessels; the relation between ascites and reduction in size of the liver was e clearly recognized by Baillie; and after him we owe to Laennec credit for having called the attention of all practitioners to this bid process, and for having to some extent pointed out the ana- ico-pathological lesions of at least one of its forms. It must be ed, however, that he had not a perfectly correct idea of the lesion, e instead of considering the process to be a sclerotic one, he ight it related to the production of heteromorphous neoplasms. ght, who in 1827 described seven cases of cirrhosis, called atten- to the dependence of the disease upon the abuse of alcohol, de- ped with accuracy the train of symptoms which it occasions, and orrectly as was possible at that time the anatomico-pathological histological lesions caused. Thus he pointed out the perihe- tis, the enlargement of the spleen frequently accompanied by splenitis, and the intestinal lesions present.

After these introductory studies, a series of anatomists, patholo- s, and clinicians began to work in the unexplored region of hepa- liseases, so that our ideas in regard to cirrhosis of the liver have lually expanded, and we now recognize many varieties which in

Laennec's time were unknown. It was first held that the sclerotic form represented the final stage of the disease, but that there was a hypertrophic stage in cirrhosis, an opinion especially upheld by the German school (Neumann, Wagner, Liebermeister, Bamberger, Rokitansky). But the researches of Ollivier, Todd, Charcot, Cornil, Hayem, Hanot, etc., have by degrees succeeded in causing a division of cirrhosis into two quite different types, the atrophic and the hypertrophic; the latter being not an early stage of the former, but a definite form characterized by sufficiently well-marked symptoms (icterus) and by specific anatomical and histological lesions, which differ from those of the atrophic form as regards both etiology and development.

This period of histological research was followed by another quite recent period of experimental study of the mode of production of the several forms of cirrhosis of varied origin. The researches of Meyer, W. Legg, Charcot, Gombault, Foà and Salvioli, and especially those of Maffucci in regard to cirrhosis of biliary origin; those of Solowieff, of Foà and Salvioli, of Strauss and Blocq, Sabourin, Maffucci, etc., in regard to cirrhosis of venous origin, have done much to clear up the question of diagnosis, as we shall see later.

Without entering into a theoretical discussion which would lead to no practical result, we will endeavor to give a clear conception of the present theories in regard to cirrhosis and to present a classification which shall be satisfactory to the clinician and in harmony with the anatomical, histological, and experimental investigations which have been made.

By cirrhosis we now understand proliferation of the connective-tissue stroma of the liver, according to a definite anatomical and physiological type. As we see from this definition, cirrhosis does not imply sclerosis, that is to say, cicatricial contraction of the connective tissue, because although this may represent the ultimate stage of the disease, it is not an essential part of it. A proliferation of the connective tissue throughout the whole or the greater part of the organ is sufficient to constitute cirrhosis. In lesions such as abscess, echinococcus cysts, neoplasms of slow growth, there is an inflammatory reaction of the connective tissue around the focus of the disease, which is manifested by proliferation and cicatrization, but which cannot be called cirrhosis.

Instead of the term cirrhosis, which expresses neither the etiology, the pathogenesis, nor the anatomico-pathological condition, we ought to use the term *interstitial hepatitis*, for this at least describes the most important anatomico-pathological lesion present, that is to say, the inflammation of the interstitial connective tissue of the liver,

hout including the ulterior stages in the morbid process, which y be sclerotic.

Many forms of interstitial hepatitis do not reach this stage of atricial contraction, but remain always in the stage of proliferan of the connective tissue, which only here and there in spots ws any tendency to sclerosis. The lesions of the parenchyma of organ in these forms are always secondary to a primary lesion of connective tissue, and consist of atrophic or degenerative proces, or simply of disturbed nutrition or of inflammation; but it is l that in some cases they may be primary and constitute the nulus to the connective-tissue proliferation. In other words, there ld appear to be a chronic parenchymatous hepatitis which reacts n the interstitial connective tissue. Many pathologists do not nit this interpretation of some of the forms of cirrhosis, yet it not be denied that in the deeply seated and extensive lesions of hepatic cells, as for instance in fatty degeneration, there is nd around the degenerated cells a hyperplasia of the connective ue which sometimes penetrates into the interior of the lobule, stituting a variety of cirrhosis known as *fatty hypertrophic cirris*. The same thing may occur in the case of amyloid degenera-. These forms, however, should be considered as secondary, or he French call them, *bastard* varieties. In the same way we may cirrhosis secondary to other morbid processes. In cardiac liver, e have already seen, the last stage of the disease may consist of iferation and consecutive cicatricial retraction of the connective ue, leading to so-called cardiac cirrhosis.

Of more clinical interest than these secondary forms of cirrhosis the primary forms, which from their frequency, from their anacal lesions, and from their symptomatology constitute perhaps nost important of all the diseases of the liver. The primary form nat which is due to primary inflammation of the interstitial conve tissue of the liver, from whatever cause. Although the anatomathological lesion is of the same nature, the final results of the use are not always the same, nor are the symptoms, the clinical se, nor the consequences. This is because the seat of the proation of tissue varies, as does the pathological development.

A classification such as is necessary for the systematic study of large group of hepatic diseases can be based only upon the primary of the inflammation of the connective tissue, which may arise in part of the vast connective-tissue network which surrounds the nchyma, the venous and the biliary ramifications, and almost s a connecting link between these many and various parts of the

The inflammatory process may begin by involving the whole

of the periphlebitic connective tissue, and thus cause *venous cirrhosis;* it may originate chiefly in the tissue around the bile ducts, causing *biliary cirrhosis*, or it may, rarely, arise from Glisson's capsule, giving us *capsular cirrhosis*. From this standpoint, venous cirrhosis may be otherwise called *periangiophlebitis*, and biliary cirrhosis *periangiocholitis*.

A classification based upon these indications is further sustained by the fact that the ultimate consequences of the morbid lesion are of varied nature, in accordance with the various connective-tissue systems in which the inflammatory process arises. For instance, in the case of venous cirrhosis, the connective-tissue proliferation tends towards cicatrization, for which reason venous cirrhosis is almost always atrophic, and this corresponds to the type first studied by Laennec. When, on the other hand, there is biliary cirrhosis, the proliferation in the connective tissue surrounding the dense network of biliary vessels shows no tendency at all to sclerosis, but on the contrary causes a notable increase of the whole organ, and is therefore called *hypertrophic cirrhosis*. Capsular cirrhosis originates in inflammation of the connective-tissue capsule of the liver, whether there be a general or partial perihepatitis or a generalized chronic peritonitis. The inflammatory lesions of the capsule extend inwards, and result in atrophy of the parenchyma of the liver. This process may be compared to interstitial pulmonitis of pleuritic origin.

Galvagni and Bassi,[45] who have endeavored to ascertain in what manner the inflammatory process is propagated from the capsule to the intrahepatic connective tissue, hold that the toxic products of mycotic origin which are accumulated around the capsule of the liver are absorbed by the lymphatics and the subcapsular vessels, and penetrating into the liver stimulate the interstitial connective tissue to inflammation. Cirrhosis of capsular origin is, however, rare and by no means well established, since we may easily have an advanced grade of perihepatitis without consecutive cirrhosis.

However convenient this classification of cirrhosis may be for the symptomatic description of the disease and for didactic purposes it does not always correspond to the clinical facts. There are some forms of interstitial hepatitis which, originating from a periangiocholitis or a periangiophlebitis, may involve the periphlebitic or pericholitic connective tissue with which they are so intimately connected, and they then constitute the so-called *mixed cirrhoses*, which are constantly growing in importance.

From the foregoing analytical but somewhat concise description of the nature of cirrhosis, we may, keeping always in view the neces-

of practice, give the following classification of cirrhosis which is upon its pathology:

- sis
  - General
    - Primary
      - (*a*) Vascular
        - Venous.
        - Arterial.
      - (*b*) Biliary
        - From retention.
        - From radicular angiocholitis.
      - (*c*) Capsular
        - From chronic perihepatitis.
        - From chronic general peritonitis.
      - (*d*) Mixed
    - Secondary
      - Fatty hypertrophic cirrhosis.
      - Tuberculous cirrhosis.
      - Cardiac cirrhosis.
      - Cirrhosis of Bright's disease.
  - Partial

is classification, based upon the anatomico-pathological condi- appear to us to be the most rational. Chauffard,[36] in his class- on, takes account of both the pathological anatomy and the gy.

hough the researches which have in recent times been made e etiology of the cirrhoses of the liver have thrown some light his somewhat obscure subject, we do not believe that they have reached a point when they can be taken as the basis of a class- n. On the other hand, as Chauffard and Bouchard both state, genic processes are rarely simple, isolated, or independent, but associated with other processes. And if this is true of dis- n general, even more clearly is it the case in this class of affec- the etiological factors and the mode of action of which are so le and varied. In our description of the disease we shall fol- e above-given classification, but shall group together a number ns which have slight clinical importance and do not therefore d a minute description.

ong the primary forms of cirrhosis which possess the most in- we have already spoken of those derived from lesions of the e, and hence called capsular.

arterial cirrhoses are clinically unimportant, and may only overed accidentally at the autopsy. The process is that of a y arteriosclerosis, or it may be secondary to a gouty diathesis, results in a reduction in size of the liver, which becomes hard anular and under the microscope shows the sclerotic process to the portal spaces, and endoperiarteritis.

ous, biliary, and mixed cirrhoses being of great practical im- ce demand a special and somewhat minute description. Of ondary cirrhoses, we have already spoken of the cardiac; the he tuberculous, and that of Bright's disease we shall refer to

Fatty cirrhosis is constituted, anatomically, by a notable

increase in size of the organ, which is large, yellowish in color, and somewhat hard in consistence. A histological examination will show, in addition to the diffuse fatty degeneration of the parenchyma, a form of annular and insulated sclerosis, together with a certain amount of phlebitis of the small portal branches and the suprahepatic veins, and a slight amount of arteritis.

Venous Cirrhosis.

Synonyms.—This, the most frequent form of cirrhosis is that which was first studied by Laennec. It has a varied nomenclature, being known as chronic interstitial hepatitis, atrophic interstitial hepatitis, atrophic cirrhosis, Laennec's cirrhosis, alcoholic cirrhosis, malarial cirrhosis, ordinary cirrhosis, periangiophlebitis, granular atrophy of the liver, and fibrous hepatitis.

Etiology.

The causes are predisposing or determining. The latter are of course of chief interest, since the predisposing causes are nearly always merely conditions which facilitate the action of the determining causes.

The first group of etiological factors in cirrhosis is represented by alcoholic poisoning. The injurious effect of alcohol upon the liver is an established fact, and has been the subject of much research. Alcohol seems almost to have an elective affinity for the liver; cirrhosis has even been known to occur in abstemious persons exposed to the fumes of alcohol, as, for instance, workmen in a distillery. This preference of alcohol for the liver has been experimentally demonstrated by Gioffredi. He found that if a cat was poisoned by alcohol administered subcutaneously, a third part of the injected alcohol was found in the liver, a fifth only in the brain, a less amount in the kidneys and lungs, a trace merely in the muscles and the blood. If chronic poisoning be produced in the animal, alcohol will be found in the liver many days after its administration has been suspended, whereas it will be absent from all the other organs and from the blood. The toxicity of the various kinds of alcohol is in direct relation to their atomic weight, and is least for ethylic and greatest for amylic alcohol (Dujardin-Beaumetz and Audigé). For this reason the most injurious action is caused by drinks adulterated with other alcohols than ethylic, especially amylic, which is fraudulently used in the preparation of brandy, cognac, rum, absinthe, etc. But in these liquors we find other substances, such as aldehydes, ethylic or amylic ace-

and furfuryl (Lépine), which possess more decided toxic powers alcohol, and which must therefore not be forgotten in the patho- is of cirrhosis of the liver.

ıe result of experimental studies upon the mechanism of the action of alcohol has not been followed by constant or uniform s. Dahlstrom, Duchek, Dujardin-Beaumetz, and Audigé ad- tered a certain amount of alcohol daily by the mouth to various ıls, but were unable to find any connective-tissue lesion at all bling that of cirrhosis. Strauss and Blocq[46] noticed in some a-pigs an infiltration of the portal spaces with young connective- cells, and later found that some of the lobules were surrounded embryonal proliferation which did not, however, reach the scle- ıtage. Other and later researches made with the same object pier, Sabourin, Mairet, Strassmann, Laborde, and others, give ı which are anything but constant or harmonious.

›re recently still Lafitte[47] has taken up the clinical and experi- l study of the subject, and the results which he obtained do not with those of Strauss or Blocq. The lesions which appeared liver after chronic alcoholic poisoning in animals, did not in- the interstitial connective tissue but the hepatic cells, which ecame altered in shape, then underwent atrophy, and finally dis- ed. Lafitte believes that the anatomico-pathological alterations connective tissue found by Strauss and by Blocq should not be ted to the irritative action of the alcohol absorbed and carried portal vein to the liver, but rather to the gastric lesions of estive or ulcerative nature caused by the tube which these in- tors used for the administration of the alcohol, and which they the stomach to be recovered at the autopsy.

s experimental result and the criticism made upon the researches uss and Blocq, which seemed to throw light upon the mode of of alcohol in the pathogenesis of cirrhosis of the liver, appear a doubt upon the importance of alcoholic poisoning and to sus- e view held by some, that the injurious effect of alcohol is due chronic gastrointestinal catarrh produced, which affects the ›n and gives rise to abnormal products of decomposition. n their turn are absorbed and pass through the liver, where ve origin to inflammatory changes. Some even hold that in- tion of the gastrointestinal tract may be propagated to the liver he sheath of the portal vein.

recent researches of de Rechter[48] would appear to confirm f Strauss and Blocq, as he has found interstitial lesions in the dogs and of rabbits subjected to alcoholic intoxication, which, r, resembled those of hypertrophic biliary cirrhosis and fatty

cirrhosis. In the rabbit the connective-tissue proliferation was almost exclusively periportal, while in the dog it was perisuprahepatic.

However we may interpret the mode of action of alcohol upon the liver, and notwithstanding the fact that the results of experimentation do not completely illuminate the subject, clinical experience—which should guide the physician in the study of morbid processes even more than experimentation (as the observation of natural facts is more important to the biologist than their artificial reproduction, which never can be performed under perfectly natural conditions)—demonstrates conclusively that alcoholic intoxication is one of the most important causes of cirrhosis.

Alcohol of whatever nature, taken in whatsoever form, is injurious in direct relation to the amount ingested. Much controversy has arisen in regard to the intensity of its action according to the conditions under which it is taken. We may state positively that the more concentrated it is the more intense will be its action. Liquors are more injurious than wine and beer, unless the latter be drunk to excess, or the wine be rich in alcohol and the beer have undergone much fermentation. Great importance is attached to the bad effect of taking liquor upon an empty stomach, and as a fact heavy drinkers who take a large amount of food with their liquor appear to be able to resist its effect to a great extent.

From the statistics published, it would be seen that the alcoholics most subject to cirrhosis are those who lead a sedentary life, muscular action assisting greatly in the cutaneous and pulmonary elimination of the alcohol, and in its interstitial combustion, thus reducing the toxicity of the fluids and plasma.

There is also to be taken into account the varying susceptibility of different individuals to alcoholic poisoning. It is very certain that we find confirmed drinkers who never become the victims of this disease, while it may occur in patients whose history shows no marked abuse of alcoholic drinks.

It is to this congenital predisposition that de Giovanni has called the attention of pathologists and practitioners, a predisposition which may possibly explain why the other etiological factors of cirrhosis, which we shall study later, may cause the disease in some individuals while in others they have no injurious effect. According to the Paduan pathologist, the predisposition consists in an excessive morbidity of the lymph corpuscles which enter into the structure of the gland. He calls attention to the fact that in the history of a cirrhotic patient we find either that he belongs to a family suffering from scrofulous or lymphatic complaints, or that he himself at some time in his life was affected with troubles of the mucosa of the digestive

iratory tracts, or of the skin or the glands, such as are seen in phatic or scrofulous persons. He also notes that adults predis- d to cirrhosis possess a notable enlargement of the abdomen, and r from gastrointestinal troubles referable to functional changes ie liver.

Ve must not forget to include in the etiology of cirrhosis of the of toxic origin chronic lead poisoning and poisoning by phos- us and by spices. Lead, according to Potain,[46] possesses a ked sclerotic action upon the liver, and Lafitte[47] was able to de- ine a true cirrhosis in rabbits by the continued administration of of lead. Others have, as a result of chronic phosphorus poi- ng, produced typical hepatic cirrhosis in animals. In some s we find that the abuse of condiments plays a part in the pro- ion of cirrhosis, as, when absorbed in large amount by the portal they induce an irritative and inflammatory state in the connec- tissue. Thus in India, where spices are over-indulged in, inter- l hepatitis is found in persons who exhibit no other etiological r capable of causing the disease.

inally Lancereaux has noted the occurrence of cirrhosis of the in copper smelters who were absolutely temperate, and gave no ry that could account for the affection. It would seem that te particles of copper are swallowed and, penetrating into the hes of the portal vein, induce an inflammatory process in the ective tissue of the liver, such as occurs in pneumonoconiosis.

nfective processes play a great part in the causation of cirrhosis. e general part of this work we studied into the reaction of infec- processes upon the liver, so that in this place we would merely ttention to some of the processes which are more especially con- d in the pathogenesis of cirrhosis. The disease, when due to tion, is produced either by direct action of micro-organisms *in* or as the result of the action of toxins elaborated in the liver , or in other parts of the body (especially the intestines), and ed to the liver in the circulation.

rerichs noted the influence of malaria in hepatic cirrhosis, and esearches of Cantani, Tommasi, and many others have demon- d that it is one of the most frequent causes of interstitial hepa-

According to Guarnieri it is these hæmatozoa which penetrate he liver and cause inflammation of the connective tissue, while chs attaches more importance to the melanic pigment, which, uced in the blood, is in greater part retained in the hepatic cells. me places, as in certain provinces of Italy, malaria is more im- nt as an etiological factor than even alcoholism. In some cases wo causes are combined. It is a noteworthy and unexplained

fact that children, while often affected with malaria which may go on to the stage of cachexia, rarely suffer from cirrhosis. There are probably special conditions of the parenchyma and the connective tissue of the hepatic gland which evade all observation, just as we can give no reason why children escape tropical abscess.

To syphilis belongs the second place in the infective etiology of cirrhosis. In the third stage of syphilis there may be a gummatous condition of the liver, or a sclerotic condition similar to ordinary cirrhosis. This is a clinical fact which has been the subject of much discussion and is denied by some, but our experience, strengthened by the opinion of authorities of great weight, leads us unhesitatingly to admit this pathogenesis. The tendency of the syphilitic virus to invade the connective tissue of the organs and glands is an accepted fact; it would therefore be illogical to exclude the liver when clinical data show that it may be affected. It is probable that there are secondary products of the infective virus produced during metabolism which are the cause of the inflammation in the hepatic connective tissue, as Strümpell holds to be the case in tabes dorsalis.

Of late, typhoid infection has been thought to cause interstitial hepatitis. According to the observations of Liebermeister, Frerichs, Cornil and Ranvier, Siderey, Hanot, and others, the liver is profoundly affected in this disease. There is marked fatty granular degeneration of the cells, and in the portal spaces and perilobular zones, nodules are formed which are composed of round cells embedded in a substance which is often homogeneous. Early in the disease the lesions of the parenchyma are the more marked, but later the interstitial lesions become prominent. The mechanism by which they are produced is not yet understood. It is certain that in typhoid fever the liver is a veritable filter for the Eberth-Gaffky micro-organisms which invade the portal system, since, according to the researches of Maffucci and Trambusti,[30] they are found in the bile almost as in a pure culture. From this circumstance we might infer that the lesions met with are the direct result of the bacillus. Other experimenters have demonstrated the injurious action of the typhoid toxin upon the histological structure of the liver. The pathogenic mechanism therefore would appear to be a double one, relating to both the direct action of the bacillus and the indirect action of the toxin. Whatever the theoretical opinion held upon the subject, in practice we find cases of chronic interstitial hepatitis which date from a typhoid infection, and in which all other causative influence is absent.

Less frequently other infective diseases are found in the history of the cirrhotic patient (scarlatina, measles, cholera, etc.) to the influence of which the condition of the liver is attributed. It would seem

t in these cases the affection is due to the toxin of the bacteria ıer than to the direct action of the micro-organisms themselves, the relation between these diseases and cirrhosis is not yet fully blished.

Another series of facts should be taken into account in the etiology irrhosis, for although they have not as yet been perfectly demon- ted, it is very probable that they would account for the occurrence irrhosis in persons in whose history no satisfactory cause can be ıd, and in whom we should be inclined to regard the disease as ıtaneous were such a view not opposed to physiological and path- ical teachings. Starting from the theory of the protective action he liver against intestinal autointoxication, we may accept the ıry that by means of a change in the gastrointestinal chemism e arise in the intestines certain products of the decomposition utrefaction of food-stuffs which are absorbed into the portal sys- , and by their irritating action in the liver cause an inflammatory ess in the connective tissue. This would be cirrhosis of auto- c origin. Although this hypothesis has not been established, it ırves to be carefully considered, since it is founded upon a sound siological basis.

At the International Medical Congress in Rome (1894) Rovighi ributed an interesting paper which would seem to confirm this ry of the pathogenesis of cirrhosis. In the course of his re- ches concerning the action of the principal products of intestinal efaction (phenol, skatol, indol) upon the anatomical stucture of liver, he noted an infiltration of young cells around the bile ducts in the intracellular spaces. Segers reports a clinical fact of some rest in this connection; he observed that the inhabitants of Tierra Fuego, who eat large quantities of sea polypi, which have been ed by Brieger to contain poisons that have an elective affinity he liver, frequently suffer from cirrhosis.

As we have seen, the etiology of cirrhosis is based upon physio- al teaching. The intoxications, infections, and intestinal auto- xications which constitute the etiological factors are intimately ed to the protective function of the hepatic cells which is exer- l in intoxications and infections of whatsoever nature. That is ay, the cause of inflammatory processes in the liver is merely xaggeration of its normal function, excited either by an abnormal ulus or by a greatly increased physiological stimulus. This view t be borne in mind in the consideration of a rational treatment of lisease.

The predisposing causes are limited to two, which represent ly the most opportune and frequent conditions for the action of

the determining causes, namely, age and sex, cirrhosis occurring in adult life, and chiefly in males. Children are, however, not altogether exempt from this disease, for they may and do suffer from true alcoholic cirrhosis, especially in countries where drunkenness prevails.

Pathological Anatomy.

At the autopsy of a subject of advanced cirrhosis we find marked macroscopic lesions of the liver.

The organ is so diminished in volume, especially as regards the left lobe, that it may be reduced to 700 or 800 gm. (1½ to 1¾ lbs.); in color it is brown, reddish-yellow (ξιρρός, tawny), or grayish, according to the preponderance of the biliary or fatty matters. The surface is rough, lumpy, and granular, the individual protuberances being of the size of a pin's head, a bean, or a hazelnut. The border of the liver is no longer sharp but becomes blunt and thick, and presents grooves caused by fibrous bands which tend to divide it into segments. The serous covering is thickened, and at times exhibits all the signs of perihepatitis. If Glisson's capsule be removed, which is done with great difficulty because it is so adherent, the granulations will be more evident and will be seen to consist of a mass composed of a greater or lesser number of hepatic lobules, surrounded by bands of sclerosed connective tissue. They sometimes resemble the head of a nail, whence the term, "hobnail liver;" at other times, instead of being true granulations, they are more like lumps separated by depressions of greater or lesser depth, constituting the lobulated liver. In consistence the organ is much harder. Upon section the knife causes a squeaking sound, and the cut surface is seen to be covered with large or small granulations encircled by fibrous connective tissue which may or may not be resistant.

There has been much discussion as to whether in the first stage of interstitial hepatitis there is a condition of hypertrophy which subsequently passes into an atrophic stage, or whether the disease begins with atrophy. Without entering into a theoretical discussion, it is very certain that even clinically a hypertrophic period may be found in interstitial hepatitis, and its existence should be admitted, since it more clearly explains the development of the anatomico-pathological lesions. The processes are, however, not absolutely distinct. The hyperplasia passes imperceptibly into the atrophy, and in many places the two coexist, so that for a long time one may be in doubt as to the stage of the malady. Moreover, we must unreservedly accept the fact that there are forms of alcoholic cirrhosis which always remain in the hypertrophic stage; Hanot has thoroughly studied them.

patic cirrhosis consists fundamentally of a hyperplasia of the interstitial connective tissue in all the phases of its development: embryonal cells, fusiform cells, fibrillary tissue, and contractile fibrous tissue. We might compare the process to that of cicatrization by granulation, which through the emigration of leucocytes finally reaches the stage of cicatricial contraction.

The embryonal tissue arises in the portal spaces in the neighborhood of the interlobular veins, and also from the connective tissue around the suprahepatic veins, according to Sabourin, who considers to be a *bivenate cirrhosis*.

We thus see that we have a *periangiophlebitis*, which, according to Charcot, is preceded by a radicular endophlebitis. The hyperplasia of the connective tissue which follows the stage of embryonal migration in the beginning includes several lobules, so that these gradually become surrounded by a narrow fibrous ring (multilobular cirrhosis, annular cirrhosis).

The experimental and anatomico-pathological studies of Maffucci[21] are of great value. He has demonstrated (1) that atrophic cirrhosis is intimately related to a periphlebitis of the portal vein; (2) that it may be caused by toxic or virulent substances in the portal circulation, or by mechanical stimulus to the adventitia of the vein; (3) that the process greatly resembles inflammatory processes in other tissues, and that from hyperæmia it passes on to an emigration of leucocytes and organization of the exudate; and (4) that this periphlebitis should be considered as quite distinct from interstitial inflammations originating around the biliary ducts.

Having briefly described the cirrhotic process, we may give a glance at the pathological results in the various component parts of the biliary gland.

The hepatic cells lose their radiated method of disposition, and spaces are left between them; they are crowded together and degenerated, and towards the periphery of the lobule some of them are found to have undergone atrophy and others to have become infiltrated with fat or with bile pigment. The branches of the portal vein are affected by phlebitis, which, according to Charcot, is the first step in the cirrhotic process, are in the early stage seen to be dilated (pylephlebitis); but later they are thickened and hardened, and their lumen is narrowed and sometimes occluded by thrombosis. We can thus readily see how the portal circulation becomes obstructed.

The suprahepatic veins, according to Sabourin, undergo an obliterating phlebitis, and the cirrhotic process, instead of being bivenate, may originate independently either from the portal system or from the suprahepatic, and may thus be *monovenate*.

The biliary ducts take no part in the cirrhotic process of Laennec, and there is seen to be merely a slight amount of dilatation of the smaller interlobular branches. The bile is diminished in amount, and is of a pale yellow or orange color.

The formation of false bile ducts has been studied into by Kelsch and Kiener, who believe that they are the result of a retrogressive process which converts the hepatic trabeculæ into small cuboid cells. According to Ziegler, Petrone, and others, they indicate a true regenerative process, accompanied by atypical glandular hyperplasia, comparable to the physiological development of the liver in the embryo.

Accompanying interstitial hepatitis are lesions in other organs and systems. The spleen is swollen and enlarged, and presents the evidences of marked hyperæmia from stasis, or of splenitis.

In the abdominal cavity we find an ascites which varies in amount according to the case. The parietal and visceral peritoneum may show all the signs of chronic inflammation. The intestines may possess a narrowed lumen, and may be shortened, especially in the iliac portion. Many and various explanations have been given of this shortening, which may reduce the small intestines from 8 to 3½ metres (26¼ to 11½ feet) and the large from 1.65 to 1 metre (5½ to 3¼ feet). Dieulafoy believes that it is related to the periphlebitic thickening of the intestinal radicles of the portal vein. Gratia[92] thinks it is partly due to the hypertrophy of the longitudinal fibres of the muscular coat. In all probability a chronic peritonitis with thickening and retraction of the peritoneum occurs as well.

As a result of the portal stasis we find dilatations of the œsophageal veins, which may assume the proportions of veritable varices, and stasis catarrh in the stomach and intestines.

Renal, cardiac, and pulmonary lesions may be found which are sometimes merely concomitant symptoms of the cirrhosis, and in other cases probably in etiological relation to them (arteriosclerosis).

Right exudative pleurisy is frequent, but dry pleurisy occurs still more often.

Pulmonary tuberculosis may be associated with cirrhosis. Lancereaux found it in thirty-five out of ninety-five cases.

Symptoms.

The symptomatology of interstitial hepatitis which is clear and well defined, and yet at the same time very complex, is entirely dependent upon the anatomico-pathological lesions.

Hepatic cirrhosis may follow an altogether latent course; it has

n found in persons who have died from other diseases and who ing life presented no sign of a nature to direct their own or the sician's attention to the liver. This is an exceptional condition hings, but it is quite usual for the onset to be latent and obscure, hat, as a rule, we are not able to tell the date of its commence- nt.

In the hypertrophic or preatrophic period of cirrhosis, and even lier, when there is perhaps merely a circulatory disturbance, we find some morbid phenomena of such vagueness and uncertainty ause so easily referable to the gastrointestinal disorders frequent lcoholics or in those suffering from chronic malaria) that the ex- nce of a disturbed hepatic function does not occur to us unless a sical examination reveals noteworthy changes in the liver. There however, prodromal symptoms and precursory signs of cirrhosis. se usually consist in a loss of appetite, a sense of weight in the gastrium, disorders of gastric or intestinal digestion, eructations, ated vomiting, and constipation alternating with diarrhœa. In e cases, especially when it is accompanied by perihepatitis, there vere pain in the right hypochondrium.

At this stage a physical examination will show the liver to be en- ed in volume, and the increase in size may be in a downward ction, or it may extend upwards, as has been pointed out by rilli. The enlargement, as a rule, is not more than about 2 to n. ($\frac{4}{5}$ to $1\frac{1}{5}$ inch). But this condition, which in the beginning especially if the patient subjects himself to a rigorous diet, pre- s remissions and even true intermissions, later undergoes pro- sive increase, which is usually in direct relation to the persistence e morbid cause. The patient begins to show signs of a decline utrition and a progressive emaciation. The skin becomes dry roughened, the color tends towards an earthy pallor, with occa- ally purplish spots over the cheek bones and on the nostrils, due bnormal development of the capillaries, while the abdomen be- s swollen; and thus all the symptoms of the atrophic period of osis gradually develop.

he anatomico-pathological condition, consisting chiefly in reduc- in volume of the liver and in an impeded flow of the portal cir- ion, gives us the key to the most important objective symptoms ronic interstitial hepatitis, and leads directly to the diagnosis.

irst of all, a physical examination of the hepatic area will show there is atrophy. Percussion especially will reveal a decrease e area of dulness. It is diminished in all its diameters, and the of the small lobe may entirely disappear, that of the large lobe reduced both above and below; in a few exceptional cases the

absolute dulness may entirely disappear, leaving only the relative dulness.

We must, however, guard against an error into which we are very liable to fall if there is abundant ascites or notable meteorism, which by the displacement of the liver and of the abdominal walls causes a diminution in the area of liver dulness not dependent upon an atrophic condition of the organ, and therefore called *apparent* diminution. A careful examination should be made of the liver immediately after paracentesis, or when the meteorism has been reduced by therapeutic measures. At this time, especially when, as is the rule, the abdominal walls are flaccid and there is little fat present, we may on palpation feel the rough and nodulated surface of the liver, and, if there be perihepatitis, appreciate the friction which is so characteristic of this affection (Frerichs). Cardarelli[55] advises us in case the patient refuses paracentesis not to lay much stress upon the liver dulness in the median, parasternal, and mammillary lines, but to look for it along the axillary and scapular lines, since at these points the liver undergoes less displacement by meteorism and ascites from the external abdominal walls.

Besides the atrophy of the liver, we find symptoms of the impeded portal circulation which consist chiefly in ascites, stasis in the spleen and intestines, and the development of the collateral circulation, these symptoms being in harmony with anatomical teachings in regard to the radicles of the portal vein and the accessory portal veins.

As we stated in the section on pathological anatomy, the interlobular connective tissue, that is to say, the field of irrigation of the apillaries of the portal system, is the chief seat of the morbid process. If cicatricial retraction of the connective tissue has suppressed or caused stenosis of many of the capillary vessels and of the ultimate venous ramifications of the portal system, a stasis is caused in the whole field in which the portal vein has radicles, and in this way we have the direct consequences of stasis in the organs whose veins are derived from this system—the peritoneum, spleen, and intestines.

The first consequence is ascites. Its appearance is usually gradual; the patient notices a slight increase in the size of the abdomen, and, as a rule, thinks that he is gaining in *embonpoint*, while noting that the amount of nourishment taken is decreasing. He frequently consults a physician on account of this symptom. By degrees the amount of serous exudation in the peritoneal cavity may reach 18 or 20 litres (quarts), or even more.

The ascitic fluid is of a clear amber color, sometimes containing bile, and sometimes turbid and sanguineous. The specific gravity

ies from 1.010 to 1.015. The amount of solids is from two to three cent., and consists principally of albumin (0.6 to 1.5 per cent.), ar, urea, and leucin (Frerichs). A microscopical examination ws a few rare leucocytes and endothelial cells, more or less easily gnizable, derived from the investing peritoneum. The fluid is s proved to be a true serous exudation.

We may have a rapid formation of ascites, the mechanism of ch has been explained by Potain as follows: By the action of a congestion of the internal organs, especially the abdominal ns, is produced; this results in an overloading of the portal ulatory system, which although capable of carrying the normal unt of blood is unable to stand the increased volume, and a peri- al stasis is rapidly produced and is the cause of the ascites.

We believe, however, that this phenomenon may in some cases end upon a possible complicating pylephlebitis, with secondary mbosis of the portal vein, whose chief etiological factor, as we already stated, is hepatic cirrhosis.

Moreover, the mechanical theory of the pathogenesis of ascites ot always be accepted, since we do not always find an exact re- n existing between the amount of hepatic impermeability and the unt of the transudation. Thus in some cases there is pronounced phy of the liver without notable development of the collateral ulation nor a great amount of ascites; while in others the ascites occur early in interstitial hepatitis and attain large proportions re there can possibly be much obstruction in the portal system. explanation of these facts may possibly be found in a peritoneal n of the ascites, and we quite indorse the conclusions reached Hanot, which are: 1. Peritonitis is a frequent complication of osis; 2. In many cases the cirrhosis remains latent until the onitis appears; 3. Exacerbations of the peritonitis accompany evelopment of the cirrhosis; 4. Peritonitis aggravates the course e cirrhosis; 5. The physician must address his treatment chiefly e peritonitis if he desires to relieve the cirrhotic symptoms.

he ascites has also been attributed to cardiac hypokinesis; this serve during the later stages of the disease to increase the effu- but can scarcely be regarded as a causative agency.

he ascites may have periods of aggravation as well as periods of ovement, but unless rationally treated will certainly follow a pro- ive course.

s to the tumefaction of the spleen, which is another result of the ucted portal circulation, the organ may become five or six times iginal size, its anterior extremity even reaching to the umbilical n. Palpation will demonstrate its enlargement better than per-

cussion. It is naturally more swollen in cirrhosis of malarial origi[illegible] than in any other.

This splenic enlargement is a frequent and important complic[illegible] tion of ordinary cirrhosis; it may, however, sometimes be absent, a[illegible] Frerichs, Bamberger, and others have pointed out, and as we ou[illegible] selves have frequently had the opportunity of observing. It do[illegible] not always depend altogether upon the stasis, because occasionall[illegible] a true interstitial splenitis will occur, with marked proliferation [illegible] connective tissue, a process similar to that in the liver. When ther[illegible] is no swelling this is probably due to the fact that the capsule of th[illegible] spleen has become thickened and unyielding from previous inflam- matory processes, or because the interstitial proliferation has entered upon the stage of cicatricial sclerosis. Frerichs found this to be the case in one patient in whom the spleen, instead of being enlarged, was shrunken.

Stasis of the gastrointestinal radicles of the portal vein is almost always shown by a catarrh of the digestive tract, called stasis catarrh (see p. 413). Gastric and intestinal hemorrhages may occur, instead of simple catarrh. The great amount of stasis may so increase the internal pressure in the venous radicles of the intestines and the stomach as to cause their rupture, resulting in hæmatemesis or enterorrhagia, which may prove immediately fatal. These symptoms belong to the terminal period of interstitial hepatitis, when the obstruction in the liver has reached its highest point. They may, however, occur in the early stage of the disease, before there is any marked ascites or any great atrophy of the liver, or even when this organ is in the hypertrophic stage. In this case, of course, the explanation above given will not hold. It is believed that they are due to the initial lesions of the gastric and intestinal radicles of the portal vein, which, according to the researches of Dieulafoy and Gratia, are easily ruptured by the slightest pressure. In interstitial hepatitis of alcoholic origin we believe that the hemorrhages are due rather to catarrhal ulcerations of the mucosa of the gastroenteric tract, with lesions of the venous radicles, from the abuse of alcohol. It has also been thought by some authorities that in this early stage they were due to the œsophageal varices which are found in cirrhosis as a result of stasis.

Quite recently, Debove and Courtois-Suffit have reported the death of cirrhotic patients from hæmatemesis in whom at the autopsy no lesion of sufficient extent to explain the occurrence was found in the blood-vessels of the digestive tract. They believe that there is in such cases an abdominal congestion of nervous origin, which, owing to the obstruction in the liver, produces so marked a disten-

of the gastrointestinal capillaries that under the rapid increase ressure they burst; the lesions being so small and so multiple ht easily escape notice at the autopsy.

More recently still Gauthier,[4] noting the unsatisfactory nature ll the explanations so far given of gastrointestinal hemorrhages ch are not dependent upon extensive portal stasis, and having rved that it was possible to produce similar hemorrhages by ns of the various microbic toxins, has advanced the theory that are due to the accumulation of intestinal toxins in the blood insufficiency of the liver.

As an ultimate result of the obstructed portal circulation we have development of the collateral circulation through the accessory al and the subcutaneous abdominal veins, forming the *caput sæ*, of which we have already spoken in relation to the anatomy general symptomatology of diseases of the liver. This collateral lation is a true physiological compensation in cirrhosis, espey in the disorders of the abdominal circulation caused by sclero- and up to a certain point we may note an inverse ratio between legree of ascites and this development of the collateral circula-

It is also a symptom of great diagnostic and prognostic value; scites it indicates obstruction of the portal circulation and calls ition to the condition of the whole portal system. It may also, by ating the degree of ascites and gastrointestinal symptoms (which emselves often point to a fatal result), form a valuable guide to the nosis.

n addition to these direct symptoms of the anatomico-pathologlesions of cirrhosis, we may have indirect results of no less imnce in other organs and functions whose relation to the liver we already in part described.

f chief importance is a careful examination of the urine. The nt passed daily is greatly reduced, while the specific gravity is ased. The color is darker than normal, there is an appearance rbidity with a more or less abundant deposit of urates, which are leep red color from the presence of uroerythrin.

he determination of the amount of urea eliminated in the ty-four hours is interesting from every point of view. The disconsists in a destructive process of the hepatic gland, and there narked diminution of urea, which falls from 25 to 30 gm. (375 to r.) a day to less than 20 gm. (300 gr.) or even lower. In estimathe diagnostic value of this sign we must take note of the nature e food and the conditions of the gastrointestinal digestion and abion, since a part of the fall in urea might be due to the fact that upply of albuminoids was limited or that they were not properly

digested or assimilated. From a number of observations in our own experience, we are able to state that this symptom is of great significance, although the researches of Lecorché and Talamon would tend to belittle it as a means of diagnosis. In a few cases in which we were able to study the action of urea in the early stages of interstitial hepatitis we found that in the period preceding ascites, when the process was one of irritation rather than of inflammation, there was a certain amount of increase in the elimination of urea (providing there were no severe dyspeptic symptoms) which was undoubtedly related to a state of hyperactivity of the hepatic cells.

Together with the decrease of urea we find a natural increase of urobilin and the presence of uroerythrin, which, as we have already stated and as the researches of Reale prove, represent an abundant urobilinuria. Either product may demonstrate insufficiency of the liver cells to produce the normal bile pigments; in other words, the urobilinuria is of hepatic origin. Semmola, from a study of urea in its relation to urobilin, has been able to determine that there exists a constant inverse ratio between the amount of urea and of urobilin eliminated, and that when under proper treatment the patient's condition improves, the difference between this relation gradually diminishes until the normal is again reached.

The researches of Lecorché show that in cirrhosis there is an increase of uric acid, this increase being due to the fact that the amount formed in the organism cannot all be converted into urea by reason of the insufficiency of the liver cells, and is thus eliminated as uric acid. Hallervoorden and Stadelmann noted an increase in ammonia due to the primary formation of lactic acid and fatty acids. Alimentary glycosuria and an increase in the urotoxic coefficient have often been noticed in cirrhosis, but the value of these symptoms we have already discussed in the general part of this work. Finally, in the urine we may sometimes find albumin and casts, which denote a secondary affection of the kidneys, due to the elimination by this organ of toxic matters left unchanged or not eliminated by the liver.

But this albumin and the peptones sometimes found by Semmola in the urine of cirrhotic patients may be the direct expression of the disease, which, by secondarily attacking the hepatic cell, renders it incapable of causing those necessary chemical molecular changes in the peptone derived from intestinal absorption, to change it into true serum albumin.

With special symptoms of interstitial hepatitis may coexist others which relate to the general state of nutrition and represent the final results of the functional disturbances of the liver. The patient suffers from a lack of nutrition, and finally becomes visibly thinner,

immediate causes of the change being gastrointestinal catarrh stasis, a limited amount of nourishment absorbed on account ıe disturbed circulation in the portal vein, and functional disınces of the liver. As a rule, the skin is thin, dry, and in procf desquamation. In color it is of a characteristic earthy grayellow, especially when the cirrhosis is of malarial origin; at there may be seen a subicteric coloring of the conjunctivæ (uroicterus). In a few rare cases we find a true jaundice, and then ıve urobilin in the urine. The almost constant absence of jaunıs due to the fact that in atrophic cirrhosis the biliary system is ıvolved; but occasionally only, as Hanot has shown, there is hal inflammation in a limited area.

the advanced stages of cirrhosis, when the ascites is very ed, there is apt to be an œdema of the lower limbs which may considerable proportions. It depends upon the pressure exerby the peritoneal transudation upon the iliacs and the ascendna cava, resulting in a mechanical stasis in this venous area.

some cases the inferior vena cava is found at the autopsy to be ulated by bands of connective tissue, originating from the liver he sulcus on its posterior border in which the vein lies.

other cases the œdema of the lower limb appears in the early of the disease, even before the ascites, and the mechanism by it is produced cannot be the same as in the preceding ins. De Giovanni, basing his opinion upon careful researches, that the ascites is related to primary lesions of the inferior ava, which throughout its extent is dilated and thinned, or else l and thickened, and may present signs of a true phlebitis. Acg to others, however, the œdema which occurs previously to the is due to cardiac hypokinesis of toxic origin. We should ɔs not overlook the possibility of there being an hydræmic ion of the blood from the altered functions of the liver, and semeiological studies of the blood shall have determined the of the changes which it undergoes in diseases of the liver, we ɔrobably have a more satisfactory explanation of the phenome- For the present we know only that there is a lessened resist- ı the blood to external influences.

rbid phenomena and complications in the other organs, which e found in interstitial hepatitis, deserve mention. In an ad- stage of the disease the heart may exhibit a functional in- ncy, due either to mechanical causes (ascites, meteorism) or to oxication.

right, or more rarely the left, pleura may be the seat of an natory process with a serofibrinous exudation, by extension

from the hepatic peritoneum to the pleura through the lymphatics. More rarely we find right hydrothorax in ordinary cirrhosis. Piazza-Martini, who has given more attention than any one else to this complication, explains it by the repletion of the azygos vein, due to the increased flow of blood from the œsophageal veins into which empties the coronary vein of the stomach, whose flow into the portal vein is obstructed by the interstitial sclerosis of the liver.

The kidneys may be the seat of inflammatory lesions due to the increased elimination of toxic products. Renal lesions in cirrhosis are a grave symptom, for, the protective function of the liver being diminished, this important compensatory organ is practically abolished, and grave autointoxications may arise and lead to a fatal issue.

We have already spoken of the peritoneal lesions in Laennec's cirrhosis, and will in this connection merely recall the fact that tuberculous peritonitis is often found in cirrhotic patients (Lancereaux, Rendu, Strümpell). Up to the present time no satisfactory explanation has been given of this phenomenon. It is, however, probable that the disturbed condition of the peritoneal circulation forms a predisposing cause for the lodgment of the tubercle bacillus.

During the course of cirrhosis, hemorrhages may occur in various parts of the body (skin, mucosa, retina, etc.), caused very probably by nutritive disturbances in the walls of the blood-vessels, and by a blood dyscrasia.

Course, Duration, Results.

The course of the disease is chronic and there may be remissions or even intermissions of the morbid symptoms.

For a long time the patient may be able to get up from his bed and attend to his various occupations, but later ascites supervenes, the respiratory difficulties increase, the appetite diminishes, diarrhœa appears; and unless appropriate treatment is inaugurated, the patient falls into a cachectic condition which terminates in death.

The duration of the disease varies. As a rule it lasts twelve to fifteen months, but it may, with alternating periods of improvement and aggravation last ten to fifteen years (Carral). There are rapid cases of cirrhosis, true acute forms of the disease, which, according to the researches of Hanot, are caused by the occurrence of acute yellow atrophy in a liver already affected with cirrhosis.

The onset may be slow and insidious, or it may be acute; the initial symptoms may consist in severe hæmatemesis or even in those of peritonitis, followed by all the usual symptoms of the disease.

In the rapid forms the morbid phenomena usually occur in the following order: a slight and atypical febrile movement, pain in the

hypochondrium, early ascites and œdema of the lower limbs, ient gastrointestinal hemorrhages, progressive cachexia, and n. The usual termination of the disease, if untreated, or if the vention be too late, is death, which frequently occurs from ca- a, or from some complicating cause such as pulmonitis, pleurisy, ntery, gangrenous erysipelas, acute peritonitis, etc. Profuse ointestinal hemorrhages may induce a grave condition of anæmia ng to a fatal issue, or, if there are renal lesions, a true toxæmia arise from the accumulation in the blood of toxic products, which ot be eliminated or neutralized because of the lesions in the liver. chiefly characterized by nervous phenomena. The patient is at troubled with unusual restlessness, insomnia which may alter- with coma, muscular twitchings, delirium, and convulsions to an extent that we are forcibly reminded of meningitis. Death next step.

the last stage of cirrhosis Riva has noticed an hepatic fever of mittent type, quotidian, tertian, or quartan, preceded by chills llowed by profuse sweating, due, he thinks, to autointoxication intestinal ptomains which can neither be eliminated nor neu- d.

Diagnosis.

e diagnosis of hepatic cirrhosis is difficult. When the clinical e is complete, when, in other words, a sufficient cause is found l the symptoms of the disease are present, especially ascites, y of the liver, enlargement of the spleen, and development of llateral circulation, the diagnosis can be promptly made. But l one of these data be absent, more especially the etiological or the collateral circulation, the disease will be easily con- d with some others.

is of first importance for the diagnosis that the abdomen be ied *after* paracentesis, or when meteorism has been reduced by ssible means, since both conditions may cause an apparent re- n in the size of the liver when there is no real diminution. ve pylephlebitis presents symptoms very like interstitial hepa- nd is followed by atrophy of the liver which reduces its area ness. A mistake in diagnosis is the more easily made in that osis of the portal vein is often caused by cirrhosis. To avoid he cause must be carefully sought, the degree of ascites noted, so the rapidity with which the effusion is reproduced after ntesis, because in cases of occlusion of the portal vein the assumes enormous proportions, and is reproduced much more than in interstitial hepatitis.

The diagnosis of cirrhosis may often be confounded with that of chronic peritonitis, especially when the peritoneal irritative symptoms are not marked, and when the history of the case would seem to suggest cirrhosis (alcoholism, infections). It is of great importance to the diagnosis that we ascertain whether there is pain in the abdominal region (either spontaneous or induced), whether after puncture and evacuation of a large amount of fluid the liver appears to be diminished, and whether there is enlargement of the spleen. The fluid will be apt in chronic peritonitis to be somewhat turbid, holding flakes of fibrin in suspension, and somewhat charged with albumin; the determination of the amount of urea, and to a certain extent of the urotoxic coefficient, will be of value in the diagnosis.

Cirrhosis of the liver, especially when œdema of the lower limbs appears in the early stages of the disease, may be confounded with a disease of the cardiovascular system in the hyposystolic period, more especially when lesions of the right heart complicate the hepatic affection. (See page 435.)

Cirrhosis of the liver might in these cases be considered as a cardiac cirrhosis dependent upon the circulatory disturbance. Only an accurate study of the history, and especially of the successive steps in the morbid process, will aid us to a proper interpretation of the clinical facts.

Many physicians endeavor to make a differential diagnosis between syphilitic cirrhosis and the other cirrhoses. We have already seen that syphilis is one of the causes of cirrhosis, but the disease so arising presents no anatomico-pathological differences from cirrhoses of different origin, and therefore we believe that no clinical distinction can be made between them. Pain in the hepatic region, referable to the peritoneal capsule of the liver, is frequent in syphilitic disease and might incline us to think the disease was of that nature, especially if there were anything in the history to bear out this view. An examination of the blood and of the effect upon its constitution of specific treatment is of the greatest value in the diagnosis, as Semmola was the first to point out. We shall enter more at length into the question in the section upon hepatic syphilis. The differential diagnosis from amyloid degeneration and from cancer of the liver is less difficult; in the first we have the etiology and amyloid degeneration of the other organs (kidneys, spleen, intestines) to assist us; in the second we must take into consideration the age of the patient, the rarity of coexistent splenic enlargement, the occurrence of a characteristic cachexia, the diminution of red blood corpuscles pointed out by Lépine, and the presence of leucocytes found by Hayem and by Alexander in all cases of cancer.

Prognosis.

For a long time hepatic cirrhosis was regarded as an incurable ase, it being supposed that if inflammation of the interstitial ective tissue had once set in, it must necessarily go on to the ess of sclerosis, which cannot be arrested by any known the- utic measures. One of us (Semmola) was the first to cast a doubt this prognostic conception of the disease, by reporting to the national Congress at Amsterdam in 1879 several cases of perfect ery under his treatment, and this doubt has since been abun- y confirmed by others.

nterstitial hepatitis consists of a chronic inflammatory process e connective tissue, which, as we have seen in the section on ological anatomy, passes through two periods—that of prolifera- represented by the development of the cirrhosis, and that of icial retraction represented by the involution of the organ. e two periods are not coexistent in the same tissue, nor do they r each other with absolute regularity, for one part of the liver be found in the sclerotic stage while another is in the stage of eration.

e must also bear in mind that the symptoms of cirrhosis, which r lead to a fatal termination, do not always represent the scle- period, but may all appear in the pre-atrophic stage (ascites, rrhagia, hepatic insufficiency).

om the foregoing considerations which, for the sake of brevity, not give more in detail, it is evident that we cannot judge of natomico-pathological state of the organ from the symptoms, when there is a marked increase in its size; and that during eriod of its development a rational treatment may, as in any c inflammatory process elsewhere, lead to resolution instead of rosis.

e curability of cirrhosis will depend, however, upon the ana- -pathological conditions; there is of course more hope of re- in the early stages of the disease. We have always laid stress this fact, ignored by so many practitioners, that there is the st probability of a cure when the hepatic area is enlarged, eing a sign that the atrophic stage is certainly not far ad- . We may sum up the matter as follows: 1. Interstitial he- , like all other chronic inflammatory processes, may undergo ion and end in recovery; 2. The earlier the stage of the dis- e better the prognosis; 3. Even should the liver be found in ition of atrophy, a prognosis of death should not be given, but ent should be commenced and faithfully adhered to.

Treatment.

The treatment of hepatic cirrhosis is above all of an etiological nature. To remove the morbid causes, such as the abuse of alcohol or residence in a malarial district, and to cure existing syphilis, are the chief indications to be met. More is needed, however. In an irritative and inflammatory condition of an organ, even physiological conditions may act as morbid causes. In a meningitis, for example, the slightest sensory impression may act to produce a convulsive attack. In a cutaneous inflammation the merest touch may cause pain. In gastric catarrh the mildest diet, and one adapted to the physical needs of the patient, may increase the trouble. Could a diseased organ be given complete rest, nearly every malady might be cured. Could the heart in endocarditis and the lungs in bronchopneumonia be given repose, the disease would soon disappear. As it is, such a state of rest as is compatible with life must be obtained for them. In the case of the liver, therefore, knowing how important is the action of this organ in digestion, the first indication is to reduce as far as possible the activity of the digestive organs.

Upon the foregoing fact we base our method of treatment in cirrhosis. A milk diet alone will secure for the liver a condition of rest compatible with life. When considering the subject of the general treatment of hepatic diseases, we demonstrated the value of milk in rational therapy; in the case under discussion its most important action consists in its securing for the liver a condition of repose, since the demands upon its activity are reduced to a minimum. A milk diet constitutes, so to speak, a subphysiological or almost negative stimulus to the liver, so that its functions are exercised without the occurrence of the nervous or vascular stimulation which accompanies its ordinary physiological activity, producing the *febris digestiva*. Moreover, the peptones formed by the digestion of milk are, by reason of their physico-chemical composition, more assimilable than other peptones and need no further transformation by the hepatic cells.

The treatment of cirrhosis, therefore, is based upon an exclusive milk diet, which must be long continued in order to produce a good effect. Too much attention should not be paid to the protestations of patients that they are unable to take milk, for experience has shown that when an hepatic cirrhosis exists this idiosyncrasy can be overcome. Every means should be tried to make it palatable, and so great is its value that not until there is absolute proof that it cannot be tolerated should it be given up.

The milk should be prescribed in frequent divided doses during day, and according to circumstances and to the tolerance or taste the patient, it may be cow's, goat's, or ass's milk. To improve its te we may add lime water or a small amount of some aromatic ffee, tea, etc.). By using these means, we shall find that very few ients are really unable to take the milk, and in these few the in- erance will be manifested by diarrhœa or by gastric autointoxica- ns. In such an event, as light a diet as possible must be adminis- ed, consisting of broths, boiled meat, eggs, weak soups, purées of etables, etc., but even this will scarcely accomplish the desired ult, as it causes a somewhat complex chemical process in the diges- tract.

The second therapeutic indication in chronic interstitial cirrhosis sists in the iodine treatment. The solvent powers of iodine are l known, especially when it is given in the form of an alkaline ide, and this action is directed especially to the liver, through ch nearly the whole amount of the iodine absorbed has to pass, where it is delayed for a certain time, as shown by numerous eriments, upon the action of the liver. In combination with the k diet, therefore, the iodide of potassium or of sodium (according hich is the better tolerated) will be prescribed; the salt may even administered in the milk, which is the best of excipients. The e should be from 1 to 3 gm. (gr. xv. to xlv.) a day, taken in ll amounts with each drink of milk. In syphilitic cirrhosis er doses should be given, 8 to 10 gm. (℥ij. to ℥iiss.) a day a due regard to the gastrointestinal tolerance of the drug.

The basis of the treatment of cirrhosis consists in the milk diet the iodine, and if persisted in faithfully for a long enough time method and the only method from which we can expect good re- s. Nevertheless it is of importance that the various symptoms h arise should receive treatment, for it is our duty to remove thing which aggravates the disease and may contribute to a fatal lt.

Ascites is the most important of these phenomena, and may be ted by either intestinal derivation or abdominal paracentesis. To e by salines an abundant intestinal serous transudation is to inish the pressure in the venous radicles and arterial capillaries, thus to place the peritoneal circulatory system in a condition to sorb the ascitic fluid. At the same time the circulation in the al vein is improved and the hyperæmic condition in the liver minished, because there is caused a depletion of the portal blood h is the functional blood of the liver. The saline purgatives h have given the best results are calcined magnesia, sulphate of

sodium, and even the various purgative mineral waters. Calomel may also be given, and has been highly recommended by some, in daily divided doses of 1 to 2 cgm. (gr. $\frac{1}{6}$ to $\frac{1}{3}$), and is thought to act not only as a mild purgative and diuretic, but also to modify the functions and nutrition of the liver and perhaps even of the sclerosed tissue. It appears to us, however, that the good results obtained from this treatment, in which calomel displays the usual action of mercurials in being easily absorbed, are not so much due to its local effect as to the fact that the hepatitis in these cases was probably of syphilitic origin.

Treatment by intestinal derivation may be successfully applied in conjunction with a milk diet and the iodine treatment unless the ascites be excessive, in which case paracentesis will have to be resorted to, being performed with all due antiseptic precautions.

The question has been much discussed as to whether paracentesis is indicated in the early stages or should be kept for the extreme degree of ascites. Without entering upon a long scientific controversy, we would merely say that in our opinion, when undertaking the cure of cirrhosis, we should endeavor by all known means to remove the ascites or reduce it to a minimum; therefore the saline purgatives should be given several trials, and if they do not give the desired result, even though the ascites be not marked, paracentesis should be performed.

The advantages obtained by removal of the fluid are immense, and relate to the condition of the hepatic circulation and the greater facility with which a collateral circulation may be established, no less than to the good effects obtained through diuresis upon the congested condition of the intestinal walls.

In any case, we should never wait for the ascites to reach large proportions before operating, as some authorities advise, because the too great distention of the abdominal walls causes such weakness that by their relaxation there might be produced a notable diminution of the endoabdominal pressure, which would tend to a rapid reproduction of the ascites (hyperæmia and ascites ex vacuo).

Drainage by means of Southey's trocar has been advised for the prevention of a return of the ascites, and Professor Difautona was among the first to adopt the measure; the results which it has given in conjunction with a milk diet and iodine have been very satisfactory.

Various diuretics have also been recommended for the cure of the ascites (diuretin, acetate of potassium, juniper) but have little action, because the circulatory disturbance is local not general. They may be tried, however, when there is not much distention of the abdominal walls.

In conclusion we would draw attention to the importance of select- a suitable climate in the treatment of cirrhosis and especially of ascites. The patient should be advised to live in a temperate, climate, of medium altitude, where there are no great or sudden mometric and barometric changes. In Italy these conditions are id at Torre del Greco, Pugliano, etc., in the neighborhood of les, the Ligurian coast, etc. Other symptoms which deserve at- ion are constipation or diarrhœa, intestinal hemorrhages, cardiac kness, hepatic fever, nervous phenomena due to autointoxication, h should be variously combated by laxatives or astringents, hemo- cs (perchloride of iron, acetate of lead, opium, tannin), heart ulants (digitalis, caffeine), antipyretics (quinine, antipyrin), and tive or stimulating nervines, according to the indications.

f grave nervous symptoms are present, Professor Cardarelli ad- that we ascertain whether they are due to the hepatic insuffi- y or to an incidental intestinal infection. If to the first, then functions of the kidneys should be stimulated, and should that e successful, we must endeavor by the means of drastic cathar- o produce a copious diarrhœa. If to the second, we must see there is careful intestinal antisepsis.

Venous Hypertrophic Cirrhosis.

his is not a well-defined form, for numerous grades exist between rophic and a hypertrophic cirrhosis. According to Hanot and rt, who have given especial attention to this variety of hepatic se, it is not to be considered as the first stage of an ordinary osis, but as a form by itself, the liver remaining enlarged even e stage of sclerosis.

s anatomical characteristics are the following: the weight may from 2 to 3 kgm. ($4\frac{2}{5}$ to $6\frac{3}{5}$ lbs.), the borders are blunt, the color sh-yellow, the surface roughened but less irregular than in the hic form. Histologically, we have to do, as in atrophy, with *nular* or *periphlebitic sclerosis*. The increased size of the liver is bly due to the extensive capillary dilation found around the s rings, or to an hypertrophy of the concentrically disposed ic cells seen in nodular hepatitis (Dieulafoy).

ne symptomatology does not differ materially from that already ibed under atrophic cirrhosis. A physical examination of the will show an increase instead of a decrease in size of the organ.

ne prognosis of hypertrophic cirrhosis is favorable, for this is rm which can be most benefited and cured by the milk and e treatment.

The cause of this form of cirrhosis is the abuse of alcohol, according to Hanot and Gilbert, who call it *alcoholic hypertrophic cirrhosis*. We have, however, met with an absolutely identical form of cirrhosis due to malaria, and have recently discharged from the Hospital for Incurables several such cases perfectly cured.

Biliary Hypertrophic Cirrhosis.

Although Raquin and Ollivier both wrote about this disease, it is only recently that it has been carefully studied and described; it has received special attention from the French school of medicine (Hayem, Cornil, Hanot). The importance of its position among diseases of the liver dates in fact from the time of Charcot's minute description of its lesions.

Etiology and Pathogenesis.

Much obscurity surrounds the origin of the disease, and our knowledge of its etiology is quite insufficient to satisfy the requirements of either practice or pathology. This form of hepatic affection occurs more frequently in men between the ages of twenty and twenty-five, and has only exceptionally been noticed in children and in the newly born.

We sometimes find a history of alcoholism, malaria, or syphilis, but this scarcely throws light upon the diagnosis, since the disease often occurs when no such cause is present.

The nature of the anatomical process does, however, somewhat elucidate the pathogenesis. The disease consists fundamentally in a primary affection of the biliary reticulum, more especially in an angiocholitis and periangiocholitis of the bile ducts of small and medium calibre. There must therefore be some pathogenic agent which causes primary inflammation in the biliary system.

The first one to advance a theory in regard to the causation of this form of hepatic cirrhosis was Schachmann. He believes that the process is not a primary one of the biliary ducts, but that it is due to functional hyperactivity of the hepatic cells, which by producing an increased amount of bile, causes a true polycholia. There is increased pressure in the small biliary ducts which first became distended and then, owing to stagnation of their contents, become the seat of an angiocholitis and a periangiocholitis, which are the direct causes of the anatomico-pathological lesions of the disease.

This hypothesis is scarcely tenable. In the first place, why should there be hyperactivity of the hepatic cells? Even admitting

from some unknown reason this may occur, it is not possible to erstand why it should cause an anatomical lesion of the biliary s and yet the cells themselves be uninjured. Moreover, when any n enters into a condition of hyperactivity, by means of those ner- influences and circulatory and nutritive compensations which as yet not understood, everything becomes modified and adapted e increased activity, for which reason it is difficult to understand the biliary ducts do not adapt themselves to the augmented sure and allow free passage to the greater amount of bile.

The pathogenesis of biliary cirrhosis must be sought for in some e which would be more liable to affect the walls of the bile ducts. theories founded upon this conception are not yet fully estab- d, but they harmonize with the laws of pathology better than Schachmann's theory.

has been thought that the immediate cause of radicular angio- tis was an upward extension of an infective process through the y system, an opinion sustained by Charcot, Goubault, and ffard, but which has not been absolutely proved. The presence e *karyophagus hominis* found by Poduyssowski has not been usively demontrated in a cirrhotic liver, nor can the infection be dered pyogenous, as some writers hold. The theory derives some support from comparative pathology. Balbiani describes a of coccidium found in the rabbit which invades the biliary m and causes a true angiocholitis. Cazin has seen a true ic cirrhosis in a rat, from the migration of the ova of nematoid s into the biliary tract.

theory which seems to us to be more in keeping with the teach- f pathology is the toxic. Bile representing, as it does, the final of all the biochemical processes of the hepatic cell, its composi- ust necessarily depend upon the complex functions of the cell pon the substances brought by the portal vein, so that its com- on will vary according to the chemical composition of the por- od. Thus the quality of the bile depends upon the substances ed from the intestines. Under conditions the nature of which as yet been impossible to ascertain, toxic products may arise he alimentary substances which, when absorbed by the portal n and eliminated by the hepatic gland, may cause irritation flammation in the small branches of the biliary ducts, resulting oangiocholitis and then in periangiocholitis. The only objec- this theory is the difficulty of understanding why the toxic cts absorbed do not cause a primary lesion of the hepatic cell. the cell exercises a protective action against intestinal autoin- tion, it probably possesses a certain amount of resistance to

the irritative agents, which therefore, instead of causing inflammation, merely stimulate the cell to hyperactivity, which helps in the elimination of the toxic agent. This is what occurs in ordinary cirrhosis due to alcoholism; the primary lesion is developed in the branches of the portal system, and not in the cell, which is attacked only when the intoxication is of long standing, or is rapidly developed. We believe therefore that the etiology and pathogenesis of hypertrophic cirrhosis can be clearly understood only when the relation shall have been determined between the products of intestinal absorption and the composition of the bile, and when it is known what irritative agents the bile may contain.

Another series of causes must be considered in biliary cirrhosis, which have a more evident pathogenic influence, although they produce very different anatomico-pathological lesions than the above-mentioned cause. We think it best to mention them here, because the clinical necessities of the case which are our guide seem to demand that they should not constitute a separate morbid form. We refer to the biliary cirrhoses due to prolonged stasis of the bile, a condition which for the most part occurs with biliary calculi. Various attempts have been made to produce this form of cirrhosis by ligating the ductus choledochus, and the results have proved that biliary stasis may act as a causative agent of inflammation. If produced under absolutely aseptic conditions (Maffucci, Dupré), it causes sclerosis; if operatory infection is produced (Charcot, Gombault), a true suppurative periangiocholitis will follow. In the first case, all the lesions of biliary cirrhosis from stasis are produced.

Pathological Anatomy.

The liver is enormously increased in size and weight, sometimes being heavier than 3 kgm. ($6\frac{3}{5}$ lbs.). It is not altered in shape; the borders remain sharp, the surface is smooth, although it may sometimes have a few granulations. In color it is a greenish-gray or olive, especially in the neighborhood of the granulations. It is hard and resistant to the knife, but does not give the creaking sound heard in an atrophic liver. The cut surface, which is of a greenish-brown color, shows large bands of connective tissue and many granulations which project only slightly and are enucleated with difficulty. Inflammatory lesions of Glisson's capsule are sometimes found. In none of the lesions are there large venous, arterial, or biliary vessels, neither can any abnormality be found in the bile duct, unless it be a calculus in the cirrhosis due to that cause.

The first thing seen in a histological examination is that the lob-

are separated from each other by connective tissue which even
trates within them in the form of more or less thickened bands.
constitutes a *perilobular cirrhosis* (Charcot), which however differs
bly from the annular form characteristic of venous cirrhosis, in
causing compression or strangulation of the lobule, but in giving
to irregular and sinuous bands (insular sclerosis) which may in-
the lobules (intralobular sclerosis). In an advanced stage of the
se the sclerosis may even invade the connective tissue surround-
he suprahepatic venous system, but this is an absolutely secon-
condition, and the fact that it is such constitutes the chief differ-
between venous and biliary cirrhosis (Sabourin).

minute examination of the hepatic lobule will in an advanced
show that the peripheric cells are separated, flattened, and de-
ed by the connective tissue which from the periphery of the
es invades the intralobular spaces, and that they are rarely
ed by the granular atrophy so frequent in atrophic cirrhosis.
epatic cells in the centre remain intact, as do those of the per-
y if the sclerosis has not become intralobular, and preserve
normal shape and size, some even being enlarged.

ypertrophic biliary cirrhosis may thus be defined as *chronic*
r or perilobular interstitial hepatitis of biliary origin.

ne most important and characteristic lesion of the disease is that
ng to the biliary ducts. The medium-sized extralobular biliary
are found to be larger than usual, tortuous and numerous, form-
complex system; their epithelial layer is fairly well preserved;
ternal coat is thickened and infiltrated with embryonal con-
e tissue which subsequently undergoes sclerotic degeneration,
ives origin to perilobular sclerosis. In other words, we have a
s periangiocholitis, which represents the first stage of the ana-
o-pathological lesion.

addition to these important changes in the biliary system, we
n abundant formation of pseudo-bile ducts, as in venous cirrho-
In the connective tissue may be seen under the microscope a great
er of small sinuous ducts which anastomose and branch dicho-
sly; their wall is composed of a single layer of cubical, almost
onal epithelial cells with large nuclei. These pseudo-ducts are
ct communication with the interlobular ducts. Their origin is
uted point, but being constant they are very characteristic of
cirrhosis. Recently Letulle[55] has stated that he does not be-
heir origin to be due to a process of proliferation of the bile
nor to development of the lobule, but that their frequent oc-
ce in hepatic affections of whatever nature, and their direct
n to the biliary ducts of the portal spaces and also to the intra-

lobular ducts would prove them to be preëxistent ducts brought into prominence either by compression of the hepatic lobules or by inflammation of the connective tissue in the midst of which they are situated.

The circulatory system in biliary cirrhosis undergoes no noteworthy change. This integrity of the portal system secures unaltered functional powers to the hepatic cells which last for a considerable period. The lesions in biliary cirrhosis due to stasis of the bile are not quite similar, since in an advanced stage of this disease there may be atrophy of a part or of the whole of the liver.

Histologically we find in these cases the evidences of a portal-biliary cirrhosis; the bile ducts are dilated and thickened, the hepatic lobules are enormously dilated, the intercellular spaces and the parenchyma cells are atrophic and degenerated. The branches of the portal vein are constricted, atrophied, and rich in elastic fibrous tissue. Chauffard thus synthetically describes the successive steps in the pathological anatomy: dilatation of the bile ducts, chronic hypertrophic angiocholitis, atrophy of the hepatic parenchyma from compression, transformation into elastic fibrous tissue of the venous system, and all this without acute or subacute inflammatory reaction, without the embryonal infiltration of sclerotic tissue or the formation of pseudo-bile ducts. As a final result we have atrophy of the liver from disappearance of the hepatic acini; the capsule of Glisson, the spaces and branches of the portal vein, and an intermediate elastic tissue being the only portions remaining (Brissaud, Sabourin).

Experiments with a view to producing the lesions of biliary cirrhosis from stasis by ligature of the ductus choledochus have been made by Meyer, Legg, Charcot, Gombault, Foà and Salvioli.

Maffucci,[58] who ligated the duct in such a way as most nearly to approach the morbid conditions of the disease, was the first to demonstrate that cirrhosis from bile stasis has quite a different origin and significance from hypertrophic cirrhosis with jaundice, the chief difference consisting in the action of the hepatic parenchyma.

In cirrhosis from stasis, we have in the first place destruction of the hepatic cells, which is in a measure compensated for by proliferation of the connective tissue. In hypertrophic cirrhosis, on the other hand, the hepatic lobules remain unaltered, their trabeculæ are sometimes transformed into embryonal vessels, and the connective tissue follows the various changes of the hepatic parenchyma. In other words, while in the first form there is a tendency to atrophy, in the second there is persistence of the hypertrophic period.

Symptoms and Course.

biliary cirrhosis there is usually a long latent period, during
there are no symptoms suggestive of so grave a lesion as that
ocess of development in the liver. The patient complains of
stion, and has all the symptoms of dyspepsia—oppression in
igastrium, flatulence, acid eructations, loss of appetite, a sense
ight and sometimes of tension in the right hypochondrium, and
of strength. Jaundice of more or less intensity follows, occur-
ither gradually or acutely, and is easily mistaken for catarrhal
ce subsequent to gastric catarrh. Slight fever may accompany
undice, and there is a little enlargement of the hepatic area,
itable to biliary stasis. As a rule, however, this stage disap-
because, the patient being subjected to appropriate dietetic
ent, the digestive condition is improved; the jaundice is greatly
shed and may even disappear; the liver may become reduced
but never again attains normal limits. This remission or com-
ntermission of symptoms lasts for a longer or shorter period;
ave reported cases in which for from two to three years there
omplete cessation of morbid symptoms. As a rule, however,
s neither so long an interval nor so complete an intermission,
tead, the improvement of the jaundice and of the general con-
is followed by rapid and grave retrogression, often accom-
by a more or less intense febrile movement. The icterus
s permanent, the liver is increasingly enlarged, the spleen
fied, and we finally reach the crisis of the disease.

other rarer cases the affection is ushered in by the jaundice,
is readily attributed to catarrh, but which persists and be-
gradually more intense without remission until the other
ms of the disease appear.

lly, there may be an acute onset in cases of cirrhosis from
sis, caused by the impaction of a calculus in the hepatic duct,
tus choledochus, or in a large bile duct.

teachings of pathological anatomy in regard to this disease
the symptoms which we find in it, and which consist mainly
lice and hypertrophy of the liver. Hypertrophic biliary cir-
onsists chiefly in a process of periangiocholitis with prolifera-
l sclerosis of the connective tissue that surrounds the bile
d by pressure partially strangulates them. This causes an
d pressure of bile in the smaller bile ducts with its subse-
bsorption by the lymphatics and veins, producing a true
rom stasis.

The jaundice, which is a constant symptom in biliary cirrhosis, has a chronic course which may be interrupted by decrease or by aggravations of its intensity. Its color varies in depth in different cases; it may be a light yellow, saffron colored, or a greenish-yellow, but as a rule is very intense. The icteric disorders of the skin, mucous membrane, and vision, which we have studied in a previous section, are of frequent occurrence.

Bradycardia rarely occurs, probably because the inhibitory ganglia of the heart have acquired a tolerance to the stimulus of the biliary acids and pigments.

The chemical changes in the urine are in direct relation to the amount of jaundice; as a rule we do not find altered bile pigment, or urobilin, or alimentary glycosuria, because the liver cells are still in working condition. The toxicity of the urine is increased, but this is due to the amount of biliary substances present. Some investigators have found the urea to be diminished in amount; in our experience, however, the quantity found depends upon the stage of the disease. It is quite unaltered until the process reaches the point where there is extensive atrophy of the hepatic cells, but becomes somewhat diminished in the last stages of the disease. The compression of the biliary ducts is neither general nor complete, nor is there total obstruction of the biliary circulation, hence there are variations in the intestinal acholia.

The slight hypertrophy of the liver cells sometimes met with would appear to be due to their increased activity, and the hypercholia which follows seems to be a method of compensation for the impeded flow of bile. The fæces are usually of about normal color, but in biliary cirrhosis due to calculi, especially if the ductus choledochus be obstructed, the intestinal acholia is complete, and the effect upon the alvine dejections is marked.

The enlargement of the liver is of the greatest importance in the diagnosis. It usually occurs very gradually, and a certain ratio may be observed between the jaundice and this hepatic enlargement. It may at other times, like the icterus, be subject to periods of diminution and of increase. It is always persistent, however, and grows progressively, never showing any tendency to atrophy. The hepatomegaly may become enormous, the liver extending three or four fingers' breadth across the costal arch, and as high as the fifth rib. Upon palpation the surface appears to be smooth, but it may be finely granular and sometimes the friction sound of perihepatitis is appreciable. The liver is harder in consistence, but never reaches a degree of stony hardness. It is of importance in the diagnosis to remember that owing to the uniform enlargement of the liver, its general shape

eserved and the anterior border is as sharp and smooth as in ormal condition. The right hypochondrium and the epigas- are very prominent, as is the left hypochondrium from enlarge- of the spleen; the abdomen becomes ovoid in shape, with its end towards the thorax, and its smaller extremity towards the

The lower ribs undergo upward rotation, the intercostal s are distended from internal pressure; the increased tension in epatic region may, by suggesting echinococcus cysts, lead to an of diagnosis. This persistent hepatomegaly may undergo fur- ncrease during attacks of aggravation of the jaundice which may ompanied by a febrile movement.

in may be caused by examination of the liver, or may, when is hepatitis, be spontaneous. It is deep and diffuse in charac- ot confined to the region of the bile ducts. If found in this sit- alone it would point to the existence of a calculus. It would r that in the final stages of the disease, when a cachectic condi- as been induced, the hypertrophy of the liver becomes some- educed (Hanot).

the case of biliary cirrhosis due to stasis from a calculus, the rophy, which is constant in the first stage, may at an advanced of the disease, when there is extensive destruction of the lob- ive way to atrophy; this is sometimes limited to the left lobe. e splenic tumor constitutes the third diagnostic symptom of ance. The spleen is always enlarged and may even extend to the median line, and inferiorly to the umbilicus or to the f the ilium. Its surface is smooth; and if there be para- is, palpation will give rise to pain. In consistence it is some- ardened. In some cases auscultation will reveal a soft, deep c murmur (splenic murmur).

do not yet know the reason for this hypertrophy of the spleen. authorities attribute it to the infection which is the cause of atic disease, but the enormous proportions which it attains and ure of its anatomical lesions would seem to be the morbid ex- n of a constant involvement of both organs in the morbid proc- n spite of its pathogenesis not being understood, it is a symp- great importance in the differential diagnosis of this disease hers which resemble it in their objective symptoms.

hepatic and splenic tumors and the jaundice are the positive symptoms by which we diagnose the disease, but it is also of ance to note the absence of certain symptoms which form a demarcation between venous and biliary cirrhosis.

hese forms of interstitial hepatitis we do not have the results al stasis, ascites, gastrointestinal catarrh, and dilatation of

the subcutaneous veins of the abdomen. There being no impediment to the portal circulation, the veins of the portal system undergo no special anatomico-histological change, the process consisting in inflammation and proliferation of the connective tissue around the bile vessels.

Yet in some cases, towards the last stages of the disease, there may be a more or less abundant peritoneal transudation, which may, as Hayes has shown, be entirely independent of any inflammation of the peritoneum. This might be assumed from the fact that the ascites may disappear while the course of the disease is unaffected. In some cases the ascites occurs in the last stage of the disease, and would seem to denote that the morbid process has invaded the periportal connective tissue and caused a disturbance in the intrahepatic portal circulation (Hanot).

As the disease progresses the general nutrition becomes profoundly affected; the skin becomes thin and roughened, and can be taken up in large folds as the subcutaneous adipose tissue is reduced in amount; the enormous size of the abdomen forms such a marked contrast to the general emaciation of the patient that it almost seems as though it were enlarged at the expense of the rest of the body.

The digestive disturbances in this form of cirrhosis are nearly always marked; bulimia has occasionally been noticed (Jaccoud, Chauffard).

We may have hemorrhages from the mucous membranes, and more rarely from the skin. Epistaxis is the most frequent form in which they occur, and may be severe enough to demand tamponing. More rarely we have gastrorrhagia, enterorrhagia, hemorrhage from the gums, and bronchorrhagia. Purpura hæmorrhagica is very exceptional. The cause of these hemorrhages is not, as in ordinary cirrhosis, found in a disturbed local circulation, but depends upon a hemorrhagic diathesis which may be induced by the deleterious action of the biliary pigments and acids upon the blood-vessels.

Cardiovascular disturbances may arise and cause grave symptoms or even fatal ascites. They sometimes consist of cardiac adynamia with its sequelæ, and sometimes of valvular lesions, especially insufficiency of the tricuspid with marked dilatation of the right ventricle. The first may be caused by paralysis of the innervation of the heart as a result of cholæmia, the second may be due to the causes mentioned by Potain of which we spoke under the head of general pathology of the liver.

At an advanced stage of cachexia from hypertrophic cirrhosis, complications such as affections of the peritoneum, pleura, and lungs,

ntly repeated attacks of facial erysipelas, endopericarditis, etc., asily occur.

uring the course of the disease certain crises occur which are racteristic and so frequent as to be of material aid in the diag-

Following the abuse of alcohol, or digestive disturbances, or ctive diet, we have marked and rapid increase of the jaundice, rease in the size of the liver with pain which may be very , vomiting and bilious diarrhœa, a lessened amount of urine is rich in pigment and poor in urea, and cutaneous and mucous rhages; febrile movements occur, preceded by chills and fol- by profuse sweating, possessing all the characteristics of inter- t hepatic fever. The general condition is weak, the nervous depressed; occasionally there will be a typhoid state with m and coma. The patient may die in one of these crises, but, le, after fifteen to thirty days there is rapid improvement ac- nied by polyuria and azoturia; the urine is greatly increased unt, and the urea which was so notably diminished increases r 50 gm. (600 to 750 gr.); the general condition improves, the sappears, the jaundice becomes less intense, and there is a dim- of pain in the hepatic region. These crises, however, leave the weaker than before, and tend to recur more and more fre- with increasing severity; owing to the diminished powers of ice of the organism they become more dangerous, and acquir- the characteristics of grave secondary icterus, may cause

se crises are the result of hepatic insufficiency, which may be anied by functional insufficiency of the kidneys, due perhaps dition of overwork caused by the abnormal products of intes- emism; this view is in part sustained by clinical facts, which e crises to be dependent upon disturbances of gastric and in- digestion. They might also arise from secondary infection iliary passages. The grave febrile attacks should certainly be red to be due to this cause.

Results, Duration, and Prognosis.

ertrophic cirrhosis is a chronic disease of long duration, last- n three to ten years. A few cases have been known to last ger.

result is almost invariably fatal, and the prognosis therefore is ily grave. Death occurs either from an intercurrent disease he cachectic period, or with the above-described phenomena s gravis.

Improvement and even remissions of the disease may occur, but not recovery.

Diagnosis.

The diagnosis is attended with difficulty, especially in the first stages of the disease when all the symptoms so greatly resemble those of catarrhal icterus. Only a careful examination and observation of the patient will enable us to form a positive opinion, which must be based upon the persistence or frequent recrudescence of the jaundice without any known cause for its appearance, upon the absence of gastroduodenal catarrhal affections, and upon the persistent flow of bile into the intestines. If painful phenomena occur with sudden intensity, we would naturally think of biliary lithiasis, from which, however, we may distinguish the affection by the absence of a true paroxysmal colic, by the inability to find calculi in the fæces, and by the previous history of the patient.

The further course of the disease will clear up the diagnosis. At an advanced stage the diagnostic signs are the uniform enlargement of the liver and the splenic tumor. These will serve to distinguish hypertrophic cirrhosis from other hepatic diseases, such as primary or secondary cancer, hepatic abscess, or echinococcus cysts; the latter, however, possess positive signs of their own which are absent in cirrhosis.

Chronic malaria with enlarged liver and spleen may be accompanied by jaundice, and might therefore be diagnosed as hypertrophic cirrhosis, but the icterus is less marked, there is always a history of malaria, and quinine has a curative influence which it does not possess in cirrhosis.

In amyloid degeneration the liver may attain the size which it does in hypertrophic cirrhosis, but there is no jaundice, there are always important etiological data (syphilis, malaria, suppuration), and there is amyloid degeneration of other organs.

In biliary cirrhosis from calculi, there is intestinal acholia, the enlargement of the liver is not enormous, and towards the end of the disease the hepatic area becomes somewhat diminished.

Treatment.

The indications are the same as in venous cirrhosis. A milk diet above all, as it diminishes the amount of intestinal toxins, allows a condition of rest to the liver, and brings non-irritating and easily assimilable substances to the hepatic cells; and iodine to induce resolution of the chronic inflammatory process in the pericholitic connec-

tissue. In a few cases, especially when there is a marked in-
ıe of appetite, an exclusively milk diet is not tolerated, and a
, nourishing diet will have to be added (eggs, vegetable purées,
meats, broths, etc.).

esides this direct treatment of the morbid cause, we must en-
ɔr to improve the intestinal digestion and circulation, to increase
aning strength of the patient, and to overcome any complications
ı may arise. To these ends we must administer bitters, strych-
purgative mineral waters, or calomel, tonics (cinchona, quinine),
ɔstatics for the hemorrhages (perchloride of iron, opium, and
n in enterorrhagia, possibly tamponing of the nares in epistaxis,
acid, ergotin in bronchorrhagia, etc.), cardiac stimulants in
lic weakness, etc. In the active crises the patient must be kept
d and fed upon an exclusive milk diet. Intestinal antisepsis
be attempted by means of calomel in small doses, and alkaline
ulphites which are also slightly purgative; in the febrile attacks
ıst give injections of quinine, and resort to other curative agents
ling to the indications.

lief may by these means be given to the patient and life may
ps be prolonged, but a complete cure is not to be expected.

Mixed Cirrhosis.

e have seen how great is the difference existing between venous
iliary cirrhosis as to their pathological anatomy and sympto-
ɔgy, there being in fact so little resemblance between them that
ıssion of the differential diagnosis is not necessary.

t in speaking of venous cirrhosis we noted the fact that jaun-
ıight appear when the process of periangiophlebitis invaded
ıvolved the connective tissue around the bile ducts, and when
bing the symptoms of biliary cirrhosis we stated that in an
ed stage of the disease ascites might occur from the invasion
connective tissue around the portal veins by an inflammatory
s which ultimately so compressed them as to cause a stasis in
rtal circulation. This leads us to a study of mixed cirrhoses,
hose forms of cirrhosis whose anatomico-pathological and clin-
ıaracteristics are as much those of biliary as of venous cir-

We may in practice meet with a case in which there are
ɔms belonging to both of these forms, or one in which the no-
is that of hypertrophic biliary cirrhosis, with some added
ɔm of ordinary cirrhosis.

the autopsy of such patients, histological lesions are found
belong to both forms of interstitial hepatitis. Thus we may

have to do with a liver which has undergone slight atrophy, and under the microscope find a sclerotic process which is both extra- and intralobular, and a highly developed network of newly formed bile ducts.

In another case we may find a larger sclerotic liver with all the signs of a hypertrophic biliary cirrhosis, presenting at the same time abundant and well-developed bands of connective tissue in the interlobular spaces.

These are the forms of cirrhosis especially studied by Dieulafoy, who named them mixed or intermediate, because they seemed almost to form a connecting link between the various forms of cirrhosis.

Of more frequent occurrence are the cases in which to all the symptoms of hypertrophic biliary cirrhosis is superadded a more or less extensive ascites, or those in which jaundice occurs in the course of a typical form of Laennec's cirrhosis. A knowledge of the existence of mixed cirrhoses will alone save us from serious diagnostic error.

The *prognosis* is grave, even more so than in the other forms of interstitial hepatitis.

The *treatment* is based upon the hygienic and pharmaceutical rules which we have already sufficiently emphasized.

INFECTIOUS DISEASES OF THE LIVER.

The liver is essentially an organ of purification and protection for the body, so that in infectious diseases due to the invasion of micro-organisms which are either pathogenic or become so under special conditions, and produce their various toxins, the liver cells enter upon a state of hyperactivity, and in the vital struggle which ensues may become the seat of varied functional and anatomical lesions.

The advance in knowledge due to researches into the etiology and pathogenesis of infectious diseases, which is so great that one may almost daily chronicle a new triumph for science, is gradually teaching us more and more about the lesions met with in the liver in the course of these affections.

A systematic study of these lesions is not within the scope of this work, because they do not possess a special symptomatology of their own nor represent a complete morbid entity. There are, however, some structural alterations of the liver of infectious origin which, either because of their being limited to the hepatic gland or because of their invading its tissues to a greater extent than that of any other organ, constitute important and frequent nosological forms and merit

cial description. These forms are the malarial, syphilitic, and culous.

Malarial Liver.

Etiology.

hen discussing the etiology of atrophic cirrhosis we spoke of nportance of malaria in that connection, especially in certain ries, and described the last stages of the infectious lesion of the n that disease.

malarial liver we do not mean the cirrhosis which accom- malaria; we should clinically designate a liver which has be- irrhotic from malaria as one affected with "malarial cirrhosis," ing the term "malarial liver" for those various anatomical and nal alterations which are caused by the micro-organism of a, and which precede atrophy.

e noxious influence excited upon the liver by malarial infection en recognized since the days of the ancients, and continues to confirmation. In Italy especially the researches of Tommasi, i, Cardarelli, and Guarnieri have called the attention of prac- rs to this morbid form which is so important from an etiologi- l therapeutical point of view.

laria in general causes well-known hepatic changes, but certain ons of situation, climate, and age influence and modify the ncy and character of the lesions.

areful study of facts shows that malaria possesses certain which are directly related to locality; in some places the acute revails, in others the chronic with cachexia and hepatic lenic enlargement. Without going so far as to attribute the n in form to the presence of Laveran's hæmatozoa, we may s hold that it depends upon the mode of introduction of the l organisms. Thus Senige,[57] in a locality where malaria could imputed to climatic conditions, found it to be due to the drink- infected water, the disease being introduced through the h.

h temperature also possesses an injurious influence. We ready spoken of the relationship existing between high tem- e, hyperæmia, and suppurative hepatitis, and the same causes to make the occurrence of hepatic lesions more frequent in l infection in hot climates or during the heated term.

ough the malarial liver and malarial cirrhosis are common in in children with even grave cachexia no hepatic lesion can be this cannot be due to the absence of alcoholism, as malarial

liver and malarial cirrhosis occur even in the temperate and abstemious, but is probably owing to some special resistance of the hepatic cell which has so far escaped the minutest observation.

However clear the etiology may be, the pathogenesis of malarial liver is as obscure as it is of interest. Some authorities attach much importance to the temperature during acute attacks of malaria, knowing how great and injurious an influence a high temperature exerts upon the nutrition and functional activity of the hepatic cell. Rendu is the chief one to sustain this theory. But although the fever may be a causative influence in the acute forms of malarial liver, this cannot be the case in the chronic forms which are the most frequent and the most important, for they often become established without there having been any marked rise of temperature.

Another cause has by some been found in the pigment derived from the hæmoglobin by the action of the hæmatozoa which, carried in great amount to the liver (the essential organ of destruction to the blood corpuscles and of transformation of blood pigment into bilirubin), there cause an irritative action and a special process of inflammation.

However important this pathogenetic condition may be, certainly of not less importance is the action of the special organism of malaria, which, according to Guarnieri, finds in the liver the best field for conflict with the phagocytes, on account of the abundance of blood and slowness of the portal circulation.

The malarial lesions of the liver are very similar in their pathogenesis to those of the spleen; that is to say, the inflammatory process occurs in a parenchyma peculiarly susceptible to invasion by the infective agent.

Morbid Anatomy.

The structural changes are to be studied in both acute and chronic forms of the disease.

In the acute form the liver is enlarged, hyperæmic, softened in consistence, and of a dark red color from the presence of a large amount of pigment. Under the microscope we find, besides the hyperæmia, that there is a great accumulation of leucocytes charged with pigment, and that the endothelial cells of the interlobular capillaries, which are undergoing cloudy swelling, contain a large amount of the pigment and of leucocytes, which in their turn enclose residua of dead hæmatozoa. In the red blood corpuscles of the capillaries are found many of Laveran's hæmatozoa in their various forms.

The chronic variety of the disease may occur in three forms, as pointed out by Kelsch and Kiener:

nflammatory malarial hyperæmia, in which there is intense hyper-
, proliferation of the endothelium of the blood-vessels which
in pigmented leucocytes, and hyperplasia and hypertrophy of
ells.

liary parenchymatous hepatitis, characterized macroscopically
large and granular liver, microscopically by a proliferation of
epatic cells, and by a network of capillaries rich in leucocytes
n proliferating endothelial cells.

hronic nodular parenchymatous hepatitis, formed by a process,
, arising in epithelial cells, ends in epithelial hyperplasia in the
of nodules surrounded by embryonal connective tissue.

Symptoms and Course.

ıe course of malarial liver may be acute or chronic, both forms
of great clinical importance.

the acute variety the patient, who has had no prodromal symp-
but has had attacks of fever of an intermittent type, is suddenly
ed with pain in the right hypochondrium, with high fever which
ɔe either continuous, remittent, or intermittent, and gastro-
inal symptoms perhaps of sufficient gravity to lead to profuse
rrhagias. The skin and mucous membranes become of a sub-
color, the urine is diminished in amount and becomes intensely
nd charged with urates; the liver enlarges, and may attain pro-
ns much greater than in hyperæmia from any other cause.
plenic enlargement is always marked, meteorism often occurs,
is sometimes a slight degree of ascites, and unless there be
t intervention, death will supervene from general infection.

e symptoms in the chronic form are less fierce in their onset
semble those of catarrhal hyperæmia, while presenting some
oms of interstitial hepatitis.

connection with a more or less pronounced degree of malarial
ia and an enlarged spleen, we find a somewhat enlarged
subicteric coloration often accompanied by urobilinuria and
thrinuria, a more or less marked development of the collateral
tion, and ascites of varying degree. These symptoms indicate
atic lesion connected with malaria, but they do not indicate
ıtomico-pathological condition.

diagnosis of this chronic form is easy, if we bear in mind the
y and the characteristic malarial lesions. The diagnosis of
ıte form is, however, attended with great difficulties; it will
ed by the type of fever which if not absolutely intermittent
ts characteristic exacerbations and remissions; by the peri-

odicity of the morbid symptoms, especially those connected with th gastrointestinal tract, as enterorrhagia; the etiological data, the en largement of the spleen, and the effect of medication.

Treatment.

The treatment consists in the administration of quinine, fro which we can expect the best results. In the acute forms especiall we may resort to the use of hypodermatic injections, and should th case demand it, even to intravenous injections of quinine bichlorid (Baccelli's method), as there frequently are conditions more unfavo able to internal medication. In addition, treatment should be ad dressed to the symptoms, and will consist principally of astringent and sedatives.

In the chronic forms also an antimalarial treatment should be in stituted, consisting of quinine and arsenic, and all known method for combating the cachectic condition of the patient.

In all cases a rigorous milk diet should be prescribed; in general the same indications are present as those considered in the therap of cirrhosis.

Syphilitic Liver.

Etiology.

Syphilis is a very frequent cause of disease of the liver. Of th parenchymatous organs the liver comes directly after the testicles an the brain in its susceptibility to specific infection. Syphilis may un der all conditions and at any period of its development attack th hepatic gland, and does so in either the secondary or tertiary perio and even in the hereditary form. We do not here refer to the atro phic cirrhosis of syphilitic origin, of which we have already spoken but of those special hepatic lesions which have no connection wit the simple process of interstitial hepatitis, but are true specifi lesions.

Hepatic lesions occur most frequently in the tertiary stage; gum matous hepatitis may even arise at an early period of the infectiv process, and this in a measure explains the fact that in Ricord' classification the divisions are not more clear and precise.

There has been, and still is, much discussion as to whethe syphilitic infection can be long latent without causing secondar phenomena, and can then later attack the liver. Our clinical experi ence is in harmony with the ideas expressed by Cardarelli, tha whether there be an actual absence of symptoms of general infec tion, or whether they be so light in character as to pass unnoticed

act remains, and is of great diagnostic importance, that some nts have a syphilitic liver without giving any history of sec- y phenomena of the disease.

here are several conditions which influence the occurrence of lis in the liver, the chief among these being the age of the ts, and the nature of their alimentation. Syphilitic hepatitis e common in infants and in the newly born than in adults, and re frequent in males than in females, they being more often d to the contagion.

are not to understand, however, that among the tertiary oms of syphilis those relating to the liver are of very frequent ence. In Fournier's statistics, which are the most complete we as yet possess, we find that in 3,429 cases of tertiary syph- ere were 9 of hepatic syphilis. We must, however, bear in hat syphilis of the liver may run a latent course, or may be nded with other hepatic affections.

e most important etiological factors are those which act spe- y upon the liver, thus setting up a *locus minoris resistentiæ* in gan; in this way malaria and alcoholism act, and they are so ntly found in the history of sufferers from syphilitic liver n to lead to errors of diagnosis.

nical experience teaches us that the syphilitic virus affects the st as it does the other organs which it attacks, especially when e there has been induced a condition of hyperactivity through ditions of life, habits, and surroundings.

editary syphilis of the liver is caused by the various condi- f hereditary syphilis, a subject upon which we cannot enter in ce. As a rule it manifests itself in the fœtus, in the newly r a few months after birth; very many fœtal deaths are due ntal syphilis. Although of rarer occurrence, there is a form y hereditary syphilis which is manifested in childhood or youth. The fact is emphatically denied by some, and strongly ned by others; for our part, we must uphold it, since the most trative clinical cases have shown us the possibility of its e.

Morbid Anatomy.

anatomico-pathological lesions of hepatic syphilis observed in nd in infants as hereditary syphilis assume, as a rule, the ous form. In the secondary forms we have no clear knowl- the lesions, for the result being usually favorable an autopsy dingly rare, and it may even happen that the affection is mis- r catarrhal icterus.

The anatomico-pathological and histological changes and the pathogenesis of secondary hepatic syphilis are therefore entirely unknown. It is probable that there is catarrh of the bile ducts, analogous to the syphilitic catarrh so often found in the laryngeal, pharyngeal, and bronchial mucous membranes in the secondary stage of the disease.

In tertiary syphilis we have the classical lesions of the liver which were first investigated by Dietrich and Ricord, and have been greatly elucidated by the further researches of Rayer, Ricord, Virchow, Wilks, and others.

The typical form is *gummatous nodular hepatitis*. The liver is enlarged and deformed. Its surface is usually of a whitish color from chronic diffuse perihepatitis, which may attain such proportions as to enclose the whole liver in a thick and adherent capsule. Protuberances of various forms and sizes are seen, and depressions and sulci of varying depth which may divide the liver into lobes (lobulated liver). The sharp border of the liver is also deformed, presenting elevations and grooves of various extent and depth. The liver is hardened in consistence throughout. Upon section it is of increased resistance, especially in certain spots when we may hear a creaking sound as the knife cuts through it. The surface of the section shows the presence of gummata and inflammation of the connective tissue surrounding them. The gummata are the shape of millet seeds, as large or larger than an egg, of variable numbers in different cases, and in various stagesof development. They are easily recognized by their yellowish-white color, by their roundness, and by their being sharply defined from the homogeneous parenchyma.

Surrounding these gummatous tumors we find fibrous connective tissue, which when young is of a milky white, succulent appearance, but when old becomes dry and tendinous and forms true cicatricial bridles which traverse the parenchyma as far as the surface, causing the characteristic depressions and sulci.

Gummata may be found in any stage of their development, from the period of endothelial proliferation to that of fusion and to the retraction caused by cicatrization, which when on the surface produces the most marked hepatic deformity and characteristic stellate cicatrices. In some cases the liver is gummatous without there being any inflammatory process of the connective tissue, and consequently there is no cicatricial contraction.

Histologically there are some points which deserve consideration. The proliferating connective tissue penetrates into the interior of the lobules and separates the cells which are often undergoing granular fatty degeneration or true atrophy. According to Malassez, there

e found in the centre of the gummata small refractive bodies, cted by carmine but stained by purpurin, which are characteris- these granulomata. The external fibrous zone is infiltrated with drops of fat at its internal portion, while the connective-tissue reserve their nuclei.

patic syphilis may, as we have seen, assume the form of an in- ial hepatitis very similar to cirrhosis, but differing from it in respects. Syphilitic cirrhosis is irregular, tortuous, and, as a loes but little injury to the portal system. According to Lan- x, the process originates in the lymphatic vessels. We, how- elieve that as the arterial system is the one most affected in is, it is highly probable that in the liver, as in other organs, ocess begins in the arterial system, the hepatic artery and its ations in this case. There may possibly be an arterial cir-

some cases of syphilitic liver the connective-tissue lesions are re prominent, in others the development of the gummata. dvanced syphilis accompanied by cachexia, we sometimes find d degeneration of the liver.

pathological anatomy of syphilitic liver in the adult differs at of early, but resembles that of tardy hereditary syphilis. in relation to the mode of infection. In tertiary hepatic s of adults of hereditary or acquired origin the characteristic are in foci disseminated throughout the parenchyma; in early ary infection the whole organ is uniformly involved, and this nearly all of the placental blood containing the infective asses through the liver. Hereditary hepatic syphilis when nally developed in adults, differs in no wise from the ac- but the early development of the disease is characterized by diffuse, interstitial, parenchymatous hepatitis. The liver is l, smooth, hard, elastic, of a yellowish-gray color, and upon exhibits small seed-like gummata, which are found chiefly in lobe and upon the anterior margin. Histologically we find is of a periportal interstitial inflammation with the produc- gummata; the connective tissue is, however, in the embryonal ot in the sclerotic. In the early period of the disease, before mata are well defined, there are no special characteristics, liver might easily be mistaken for a tuberculous or infected

ffard had the opportunity of examining microscopically the ic liver of a new-born infant, which under specific treatment d of the hepatic syphilis but not of the general disease; he at while the acute interstitial and parenchymatous lesions

had undergone resolution the portal and biliary sclerosis and the suprahepatic endophlebitis remained, probably as permanent stigmata of the infection, and might perhaps have been the starting-point for future hepatic disturbances.

Symptoms.

A separate description must be given of the symptoms of secondary, tertiary, and hereditary syphilis.

Syphilitic Jaundice.—In the secondary period of a generalized syphilis there may be more or less jaundice, accompanied or not by intestinal acholia, and characterized by all the attendant symptoms of cholæmia. More or less severe general phenomena also occur, such as fever, general weakness, and headache, which are due to the primary disease.

It is of importance as regards the diagnosis that syphilitic icterus be diagnosed from catarrhal icterus or from other biliary disorders. To this end, making a careful examination of the patient, we must note the following points: the existence of a specific infection; the complete absence of gastrointestinal disturbances, for these are the cause of catarrhal jaundice; a vitiated general condition, not usual in catarrhal jaundice; the short duration of the disease, and, according to Queirolo, enlargement of the spleen, which is a constant symptom in the secondary period. The liver is enlarged in both syphilitic and catarrhal jaundice, and usually projects two or three fingers' breadth beyond the costal arch, but the enlargement is more marked in the specific disease.

The examination of the urine and the severity of intestinal disorders will further aid in the diagnosis. In syphilitic jaundice albumin is often found in the urine as the kidneys are frequently invaded. Cardarelli attaches much importance to the obstinate diarrhœa which sometimes accompanies syphilitic jaundice, constipation being the rule in the catarrhal disease. Treatment constitutes an important diagnostic sign, the jaundice disappearing under antisyphilitic remedies if of specific origin, while catarrhal jaundice, which may besides occur during the course of syphilis, remains unaffected by these therapeutic measures.

The prognosis is good, as in the case of other secondary lesions, when energetic and appropriate treatment is instituted, unless the syphilis be of a rapid type. The disease may last from a few weeks to several months. The syphilitic lesions of the liver in the secondary stage should, however, serve as a reminder to both patient and physician that in the hepatic gland the infective agent finds the best

ble conditions for its development, and that therefore the process
s organ is very likely to go on to the grave anatomical lesions
tertiary period.

rtiary Syphilitic Hepatitis.—The symptoms of the cirrhotic form
tiary hepatitis being the same as those of ordinary cirrhosis,
ed not dwell upon them further than to state that in some forms
disease the painful phenomena referring to the liver predom-
because perihepatitis is frequently present. Hepatic friction
n to be found.

ue syphilis of the liver, characterized by the presence of gum-
is that which we wish to describe. Its course may be alto-
latent; severe syphilitic lesions have been found in the liver
sons whose death was due to other causes, and who during life
ted no symptoms of hepatic disease. As a rule, the onset of
ection is insidious and obscure, which makes it difficult to give
nion as to when the hepatic symptoms first made their appear-
The gummata may develop slowly upon the hepatic paren-
, without causing any serious disturbance in the circulation of
od or the bile, and without injuring the parenchyma, which
its normal functional powers.

the case, however, of syphilitic liver with inflammation of the
itial connective tissue and coexisting lesions of the capsule,
re apt to be painful symptoms or morbid phenomena due to
culatory and functional disturbances which arouse the patient's
and serve as a guide to diagnosis.

the majority of cases the first symptoms consist of progres-
aciation, with a characteristic malaise, digestive disturbances
ing severity, and pains in the hepatic region which may be
nd dragging or severe and lancinating.

he same time a more or less pronounced tumefaction appears
right hypochondrium, ascites begins to form, and jaundice
tes the morbid picture. A physical examination of the liver
eat value, and the best results are obtained from palpation.
ht hypochondrium is seen to be enlarged, the lower intercostal
projecting, the epigastrium swollen. Upon palpation we may
e friction of perihepatitis, and may, in addition, note that
r is irregularly enlarged and changed in shape. The up-
face is characterized by scattered globular nodules of varying
d by depressions and sulci of various depths. In con-
it is so hardened as sometimes to be of woody induration.
terior border is distorted and covered with eminences and
the latter sometimes so deep as almost to give the impres-
a notch. The size of the liver varies; it may attain large pro-

portions, reaching beyond the transverse umbilical line and descending into the hypogastric region. Percussion will show that the organ is also enlarged superiorly, extending into the thoracic cavity by upward displacement of the diaphragm.

In addition to these valuable diagnostic signs, we shall find the results of obstructed portal circulation from compression of the intrahepatic branches of the portal vein, consisting of ascites, development of the subcutaneous collateral circulation, and enlargement of the spleen. Jaundice is somewhat rare, and never severe.

In one case which came to our notice the disturbances of the portal and biliary circulation were so great as to lead us to diagnose a large gumma at the hilum of the liver which was exercising pressure upon the portal vein and the hepatic duct; our opinion was completely confirmed by the autopsy.

When the gummatous type of the disease predominates there may be no deformation of the liver, which may be simply enlarged, no disturbances of the portal circulation and no jaundice.

When there is icterus the urine shows all the signs of cholæmia; when it is absent the urine is charged with urobilin, poor in urea, and in some cases will give the sugar reaction to the test for alimentary glycosuria, all signs of hepatic insufficiency.

The course of the disease is a protracted one, and the result fatal unless we promptly intervene with appropriate treatment. The patients succumb to a well-defined cachexia or to the results of syphilitic lesions in other organs (kidneys, brain, etc.).

The *prognosis* should always be reserved, but is not necessarily bad; it will depend upon the condition of the liver, the general condition and age of the lesion. Gummata become modified and disappear under appropriate treatment—not so the consequences of interstitial hepatitis, consisting of cicatricial contraction. The more prompt the intervention the better the results.

The *diagnosis* of tertiary syphilitic liver may be easy or extremely difficult; it is easy when to the signs of a progressive syphilis are added the objective symptoms of the sclerogummatous form of hepatic disease; difficult when the history of the case is not clear and the form of the affection is atypical. It may in this case be confounded with cancer of the liver, cirrhosis, etc.

The etiology and the result of treatment are of the utmost importance to the diagnosis. Whenever there is a suspicion of syphilis, the personal and family history of the case must be carefully inquired into, and the patient must be thoroughly examined. Hutchinson's triad of symptoms must also be sought for (ocular, aural, and dental lesions), and the arrest of physical development (infantilism) and

ular atrophy are also important symptoms of hereditary lis.

ıe therapeutic test may be tried in doubtful cases, but should be d by an examination of the blood for a determination of the num- red corpuscles and for the estimation of the amount of hæmo- ı. We have established the fact that the administration of mer- s to syphilitics causes an increase of red blood corpuscles and ally of hæmoglobin, and that therefore in order to determine er specific treatment is adapted to a given case accurate com- ve examinations should be made to see whether the number of ood corpuscles and the amount of hæmoglobin is increased; if ıercury is contraindicated, because the subject is not syphilitic. s a most important test, and of more value than any other, nables us to judge in the very earliest stages whether a speci- e is indicated.

reditary Syphilis of the Liver.—In hepatic syphilis in the fœtus ost marked symptom is hydramnion, due to the hepatic ob- on of the portal circulation. This causes a notable enlarge- of the abdomen of the pregnant woman, and the phenomena of tion may be felt in the distended uterus. The fœtal move- are indistinct. Death of the fœtus frequently occurs, and with tion.

ıuffard notes that serious consequences of compression may to the mother, such as dyspnœa, vomiting, cyanosis, and ıbdominal pains.

he newly born, as a rule, hepatic syphilis is a continuation of yphilis. Sometimes there are characteristic symptoms from ment of birth; in other cases these are developed in the first of life, and are always accompanied by other symptoms of ary syphilis, such as coryza, mucous and cutaneous syphi- ssures of the umbilicus, the anus, the lips, etc. Cachexia is marked. The liver is uniformly increased in size and occupies part of the abdomen, is painful to the touch, smooth and with- minences. There is almost always enlargement of the spleen hepatic form). The abdomen is voluminous from meteorism ites, the superficial venous circulation is greatly developed, emaciation and cachexia proceed to a fatal termination unless nt be prompt and efficacious.

diagnosis is easy because, in addition to the history, we always symptoms of hereditary syphilis.

hereditary syphilis is exactly similar to the acquired form, hich head a description of its most characteristic symptoms found.

Treatment.

In no disease of the liver do we obtain better results, because th treatment may be administered to the cause of the affection.

Mercury and iodine are the antisyphilitic remedies *par excellenc* The first may be given by means of friction with mercurial oint ment or by injections of the bichloride preparations, which we prefe to all others, no matter how much vaunted they may be (calomel oleum cinereum, mercury succinimide, sozoiodol of mercury). Inunc tions are to be preferred in the tertiary stage when the symptoms ar not very severe, as their effects (although slower) are more durable but injections are preferable in the secondary stage when the symp toms are so grave as to require rapid and efficacious intervention On account of the gastric conditions the remedy is not to be admin istered internally.

Chlorate of potassium may be used as a mouth wash or given in ternally to prevent or cure stomatitis.

Iodine, in the form of iodide of potassium, which is more activ than iodide of sodium (which is, however, to be given should the forme not be tolerated), should be prescribed in cases of gummatous an sclerogummatous hepatitis, in progressive doses until as much as to 5 gm. (60 to 75 gr.) daily is taken; it is to be largely diluted i water, and taken a little at a time during the day.

In hepatic syphilis of the new-born mercurial inunctions shoul be given, and iodine may be administered indirectly by giving iodid of potassium or sodium to the nursing mother or wet-nurse.

The points considered under general therapy are to be borne i mind in the treatment of hepatic syphilis, especially those relating t the physiological treatment.

Tuberculous Liver.

Tuberculous infection of the liver is more interesting from a patl ological and experimental than from a clinical point of view. Th hepatic lesion being almost invariably secondary to some other grav morbid process, the symptomatology is not a clear and definite one but is complicated with that of the primary disease. In a few cases however, tuberculous liver may be considered as an idiopathic di ease, from a clinical standpoint at least, as the origin of the infectio from other organs or tissues is so obscure as to pass unperceived an to allow the liver symptoms to predominate.

The tubercle bacillus may make its entrance into the liver i

ways, all of which may be demonstrated practically and in the tory.

berculosis of the liver is most frequently found in connection ıberculosis of the peritoneum and of the digestive organs; and xperimentally inject a very small amount of the culture of the e bacillus into the peritoneum or the inferior mesenteric vein, disseminated tubercles will be found in the liver post mortem. fection may, however, be carried through the general circula- s in acute miliary tuberculosis in which the liver is always l. If a rabbit be injected with a tuberculous culture in a eral vein, some tubercles will always be found in the liver r with lesions of the other organs.

intrauterine life infection may reach the fœtus through the al vein. Sabouraud reports a case in which the mother was l with pulmonary tuberculosis, and at the autopsy of the in- eleven days disseminated tubercles were found in its liver and

few rare cases the hepatic tuberculosis may be primary. has shown that bacilli introduced into the intestines through lous meat may penetrate directly into the liver through the ıd lymphatics without causing any apparent lesion of the in-

Pathological Anatomy.

rculous infection may in the liver cause lesions which differ t and in their nature. Three special forms may be distin- which are not absolutely distinct types, but bound together mediate and temporary forms.

most frequent is miliary tuberculosis, which, however, never an absolutely individual type. The liver is hyperæmic and , and upon its surface, beneath the peritoneal covering and in ior, may be seen a large number of round, gray, and semi- ent tubercles, some of which are undergoing caseation. Ac- o Brissot and Toupet their distribution is subject to definite us, the tuberculous granulations become systematized into ons similar to those of Glisson's capsule, and the sublobular aces are the most frequent seat of these lesions.

ıistogenesis of hepatic tuberculosis has thrown much light general origin of tubercles. The bacilli, it would seem, are in the capillaries of the liver, causing a fibrinous clot which mptly invade and in which they multiply up to the fifth or ıth day without causing any local reaction or cellular emi- Around this fibrinous and bacillary thrombus there follows

an emigration of leucocytes which by the action of the bacilli ar converted into epithelioid and giant cells (Cornil, Yersin).

Metschnikoff believes that the endothelial cells of the blood-vessel and the large mononucleated leucocytes are the chief agents in th formation of the granulations and the giant cells. Baumgarten, o the other hand, holds that the hepatic cells themselves play th chief rôle in the formation of the tubercle.

In exceptional cases the hepatic tubercles undergo fusion and for a cavern. When this exists it is usually around the bile ducts, t result of a tuberculous angiocholitis. On account of this locati Chauffard believes that in these cases suppurative germs from t intestines unite their action to that of the Koch bacillus. The caver rarely attain dimensions larger than a pea or a cherry, and ma in case the patient does not quickly succumb, go on to a process cicatrization, thus causing biliary cysts, as they eventually co municate with a bile duct, and their contents become turgid wi bile.

Experimental researches have shown that the tubercle bacillus do not produce tuberculosis alone; on the contrary, it possesses sclero and steatosis-forming properties which it exercises upon the conn tive tissue and parenchyma of the liver. Martin and Ledoux-Leb found that tuberculosis induced in the rabbit settled in the por biliary spaces and caused an abnormal proliferation of interstit connective tissue. Hanot and Gilbert produced a true tubercul perilobular cirrhosis in a rabbit; they believe that this result is d to a slight degree of virulence upon the part of the bacillus. Its tion in producing fatty degeneration is shown by the zone of steato almost constantly found surrounding the tubercles. Additional pr of these facts is derived from the special class of alterations in t liver which follow tuberculosis, namely, tuberculous cirrhosis, a fatty hypertrophic cirrhosis.

In tuberculous cirrhosis, which has been especially described Hanot and Lauth,[58] the liver is lobulated and furrowed by fibr bands which almost convert it into a lobated liver. The cirrhosis portobiliary, with marked formation of a biliary network. There also periportal fatty degeneration, with fine radiating fibres of int cellular connective tissue, and numerous tuberculous granulatio are disseminated throughout the hepatic parenchyma.

Fatty hypertrophic cirrhosis has been studied chiefly by Hutine Sabourin,[60] and Bonygues.[61] According to some authorities it is d to alcoholism combined with tuberculous infection. But the searches of Hanot in especial would go to prove that it may ex without any alcoholic history; the latter, by causing a certain amou

tic insufficiency, may predispose to the causation of the lesion, s not an essential condition.

liver in fatty tuberculous cirrhosis is so greatly enlarged that weigh 3 to 4 kgm. (6½ to 9 lbs.), is of an ochre color, and the face is smooth and fatty. In the portobiliary spaces there id young connective-tissue formations. Under the microscope that there is a portobiliary cirrhosis which may be isolated se. The perivascular and perilobular connective tissue does me sclerosed, but contains small scattered tuberculous gran- which may even invade the lobule, and sends out fine pro- ns which penetrate between and separate the cells. The ls are swollen and deformed, and either throughout the lobule times (in the initial stages) in the median portion of the t an equal distance from the portal and the suprahepatic hibit all the signs of fatty degeneration.

lly, there is a form of hepatic tuberculosis which has been d by Kelsch and Kiener, characterized by a nodular hepatitis o that found in the malarial liver. Upon section the paren- s seen to be mottled like marble, which is due to whitish ions that may be miliary or pyriform or rounded, and which t upon a red or yellowish background.

r the microscope we find trabecular hypertrophy around a ary centre, a notable growth and centrifugal expansion of beculæ, a double or triple increase in size, and pressure of the peripheral trabeculæ with a tendency to the formation fibrous capsule.

athogenesis is still obscure; the process appears to be due to matory reaction of the trabecular epithelium from irritating rought by the biliary ducts, rather than to Koch's ba-

nmediate cause of the sclerotic and fat-forming influence of us infection has been widely discussed, and opinions still on the subject. Hanot and Lauth believe that the toxins by the bacilli have a sclerosing action upon the connective d a fat-forming action upon the cells. But Chauffard e observation that experiments with Koch's tuberculin do e least support this theory. On the contrary, the bacillus pear to induce these alterations according to some special of the hepatic tissue. It is very certain that for the forma- y morbid lesion not only is the etiological factor to be con- ut also the condition of the fluids and tissues, which so eluded all clinical and experimental research. It is there- ible that the liver in tuberculous infections reacts in a dif-

ferent way according to the virulence of the bacillus and the conditions of nutrition and resistance of its parenchyma.

Clinical Forms.

When during the course of a tuberculous affection of the othe organs there occur hepatic lesions, these, as a rule, exhibit no es pecial symptoms, because the symptoms of the primary affection pre dominate. But if particular attention be given to the anatomical an functional condition of the liver, symptoms will be perceived whic point to an invasion of the liver by the tuberculous infection. Palp tion will produce a certain amount of pain in the epigastric regi and right hypochondrium; the liver is found to be slightly enlarge extending about two fingers' breadth beyond the costal arch. T skin is slightly yellowish, and we find urobilinuria and glycosuria.

These symptoms are also found in the fatty or amyloid li which is often due to tuberculosis, and we cannot therefore make differential diagnosis without the data furnished by the history the case.

In some cases the liver symptoms will predominate, either cause the primary affection is unperceived or, more rarely, becau the primary tuberculous lesion is in the liver.

The sclerotic and fatty forms of tuberculous cirrhosis are th which present the most characteristic symptoms.

The Hanot-Lauth type of tuberculous cirrhosis develops clinica with the symptoms of ordinary cirrhosis, which it greatly resemb in its anatomico-pathological constitution. The presence of abdo nal pains, sensitiveness to pressure, a somewhat marked jaundice rapid cachexia, and advanced or beginning tuberculosis will be chief guides in the differentiation of tuberculous from atrophic rhosis of the Laennec type. During this form of cirrhosis the pe toneum is apt to be involved, and the patient is likely to die of tub culous peritonitis. The course of the disease is somewhat rap and the prognosis is absolutely fatal.

In fatty hypertrophic tuberculous liver there are clinically t periods: the premonitory, which relates to the abuse of alcohol spirituous liquors, and is marked by gastrointestinal disorders, sli hyperæmia of the liver, and contemporaneous signs of tuberculos such as cough, evening fever, and night sweats; and secondly, p nounced hepatic disease.

Chauffard notes that, as a rule, the first period passes into t second abruptly, and perhaps as the result of alcoholic excess cold, or aggravation of the pulmonary condition. The digesti

rbances then become marked, there is total anorexia, the skin nucous membranes are subicteric, the urine is diminished in nt, and we have hypoazoturia, the presence of biliary pigments, lin, and even albumin; the fæces are pale, there may be gastric, inal, and nasal hemorrhages, the general condition is depressed, is progressive cachexia, and perhaps grave symptoms occur of ic autointoxication.

ne following is the classification adopted by Gilbert and Hanot various forms of hepatic tuberculosis:

ent.

ite—Fatty, hypertrophic tuberculous hepatitis or fatty hypertrophic cirrhosis.

acute { 1. Fatty tuberculous hepatitis, atrophic or without hypertrophy.
{ 2. Parenchymatous nodular tuberculous hepatitis.

ronic { 1. Tuberculous cirrhosis.
{ 2. Fatty degeneration.

e may add that it is not possible clinically to distinguish the -described forms from fatty degeneration of tuberculous origin, ch we shall speak in the section upon degeneration.

Diagnosis.

e diagnosis can be made only by means of a careful physical nation of the liver, which should never be omitted in tuberculous ts. The liver is found to be enlarged, passing the costal arch o 5 cm. (1½ to 2 in.). Its surface is smooth, its consistence hat harder, the anterior border is a little blunted; pressure y causes pain. The spleen is enlarged, ascites is not marked, ere is but a slight development of the collateral circulation.

ien the hepatic insufficiency has fully declared itself a veritable d condition, characterized by the classical nervous symptoms um, convulsions, coma), may arise and lead to a fatal issue. er cases the progressive emaciation, general vitiated condition, eat loss of strength are the immediate causes of death. Death vever, sometimes due to the pulmonary condition.

ring the course of this morbid form, the first period, charac- especially by disturbances of alcoholic origin, may be en- absent, this not being an essential pathogenic condition of ulous cirrhosis. Clinical facts observed by Laure, Honorat, utinel in non-alcoholic patients and in children conclusively his to be a fact.

Prognosis and Treatment.

The prognosis of this affection is altogether bad.

The treatment is merely symptomatic and should be followed ac cording to the rules already given.

PARASITES OF THE LIVER.

A variety of animal parasites may pass from the general circul tion, the portal circulation, and the biliary system into the liver an there find a soil adapted to their development. The greater numb invade that organ through the portal and biliary systems, comin from the food in the gastroenteric tract. Some of these organisn are accidentally present in the liver, their natural territory being th intestinal tract, while others are always and only developed in th liver; a third group of parasites may be found in any tissue, but a more frequently found in the hepatic tissues for which they have certain preference.

In the first class we may place the ascaris lumbricoides, in th second the distoma hepaticum, in the third the cysticercus cellulos and especially the tænia echinococcus.

The *ascaris lumbricoides* which is so frequently developed in th small intestines may pass into the ductus choledochus, the hepat duct, the gall-bladder, or even the large bile ducts, causing an ob structed flow of bile with icterus from stasis, and giving rise t catarrhal or purulent inflammation of the bile ducts, and even t hepatic abscess of cholic origin. The diagnosis of these morbi forms, which may vary in intensity from a simple catarrhal icteru to a grave biliary infection, is very difficult, and only possible in fa when lumbricoids are found in the intestinal tract and there is seen t be a certain relation between them and the symptoms of the hepat disease.

The *distoma hepaticum* and the *distoma lanceolatum* are frequentl found in the livers of sheep, where they may cause grave lesion (gangrene of the liver). They have but rarely been found in man.

The hepatic distoma is a trematode, flat, laminar, of a long ova shape, and when fully grown from 2½ to 4 cm. (1 to 1½ in.) in length the head has a sucker 4.5 mm. (⅛ in.) long. The ova of this dis toma are easily recognizable. The distoma lanceolatum is smalle and more slender, and its ova are of a different shape.

It is most probable that these parasites enter the intestines in th ovum stage, and migrate into the biliary passages. They remai

bile passages where they cause stagnation of the bile and
ıal and suppurative processes.
e most commonly observed symptoms are jaundice, enlarge-
›f the liver, diarrhœa, œdema, cachexia, hemorrhages, etc. A
ısis is almost impossible, and errors are frequent, the symptoms
by the parasite being attributed to specific or other inflamma-
fections of the bile ducts. The ova or the parasites themselves
›metimes, by the aid of the microscope, be found in the fæces
omited matters.
› *cysticercus cellulosæ* of the liver is more often found at the
y than diagnosed in practice. When it gives rise to an abscess
clude that there is a purulent hepatitis, but it rarely occurs to
it is due to so exceptional a cause as the cysticercus.
ew rare cases of *actinomycosis* have been reported. Objective
›ms are caused which greatly resemble those of abscess or of
r cysts, but they do not in practice suggest such a pathological
ıs actinomycosis.
›ng the parasites which may primarily invade the liver, the
chinococcus merits our chief attention, for while invading all
it is in the liver that it finds the most favorable conditions for
ılopment.

Echinococcus of the Liver.

(Hydatid Cysts; Echinococcus Cysts.)

ETIOLOGY, PATHOGENESIS, STRUCTURAL CHANGES.

a complete understanding of the causes which operate in the
ment of the echinococcus in the human liver, it will be neces-
efly to describe the natural history of the parasite.
fully developed echinococcus is found in the dog and in other
of the same family (wolf and jackal). The ova are found in
s of these animals, and by reason of their capsule offer a long
ce even to the process of putrefaction. They may invade
waters or be deposited upon grass and vegetables and be in-
›y human beings. More frequently still the ova of the tænia
an directly from the dog. From the dog's habit of licking
he may introduce the ova into his mouth, and the unclean
›f permitting dogs to lick their master's lips may suffice
luce the ova into the human mouth, when they are swallowed
saliva. For these reasons echinococcus cysts are often
ı countrymen and in shepherds, especially those who are
imate friends with their dogs. In countries such as Ice-

land, where the dogs live in the constant companionship of thei masters, echinococcus cysts are the most frequent. According t Budd, sailors never have echinococcus cysts, because they so rarel have anything to do with dogs.

It is easy to understand why echinococcus cysts are more frequen among the poor than among the rich, in adults rather than in childre or old persons, and in men rather than in women.

The process of invasion of the liver by the tænia echinococcus as follows: In whatever manner introduced, the capsule of the ovu in the stomach is dissolved by the action of the gastric juice and t protoscolex of the tænia armed with six hooks is set at liberty. T exact path taken by the embryo to reach the liver is not perfect understood. It is probable that by means of its own active mov ments aided by its hooks, it penetrates the intestinal walls, and e tering into some branch of the portal vein is carried by the circ lation to the liver. It is not impossible that it may travel along the walls of the portal vessel, or that it may perforate the duodenu near the mouth of the ductus choledochus, or travel through the bi ducts. Once in the liver it loses its hooks, which are no longer ne essary to its existence, and passes into the so-called vesicular stag that in which the hydatid cysts are formed. From its caudal extre ity it puts out a serous vesicle which develops until it complete surrounds the tænia, which is then free in the cystic cavity, the vi ceral walls of which, so to speak, invaginate and surround it. T endocystic fluid increases and accumulates, and constitutes a tr echinococcus cyst. This cyst therefore is composed of a membra which is the product of the dwarf tænia, of fluid, and of the prot scolex which is attached to some one point of the internal surface the membrane, and floats freely in the fluid. The cyst may be 2 to mm. ($\frac{1}{12}$ to $\frac{1}{8}$ in.) thick, is of gelatinous appearance and elastic co sistency, and is easily detached from the hepatic parenchyma, which it is not very intimately connected. The hydatid fluid is clear as distilled water; its reaction is neutral or slightly alkaline, t specific gravity 1.008 to 1.015; it contains sodium chloride, traces succinic acid or calcium succinate, inosit, and sometimes leuci tyrosin, glucose, and hæmatoidin. As Gubler was the first to poi out, it never contains albumin, which he thinks due to the fa that the parasite uses all the albumin derived from the blood for i own nourishment. When the tænia dies, albumin is now no long needed by the parasite, and appears in the fluid. The amou of liquid contained in a cyst varies from a few grams to sever litres. In some cases a microscopical examination of the flu will reveal the presence of the hooks of the echinococcus; this

ıst important diagnostic sign. It is sometimes colored by bile ɔod.

esearches have recently been made with the view of ascertain- hether the fluid contained any toxic substance. Maurton and ıgdenhauffen found special ptomains in the cystic fluid of the). Viron[62] found an albuminoid substance similar in its chemi- ıd biological properties to toxalbumin.

he development of a cyst is not without its effect upon the liver, as a foreign body it provokes marked inflammatory reaction of ɔnnective tissue, which by proliferation forms a connective-tissue le that later undergoes fibrous degeneration. A fibrous mem- is thus formed around the hydatid membrane, and, enclosing 'st as in a capsule, acts to prevent its further extension. This le, which is of a more or less thick fibrous structure, is richly lar, and from it the cyst derives its nourishment. Around this le the hepatic substance undergoes atrophy to a greater or less ; and if the cyst reach to the peritoneal covering it will cause a epatitis.

ıe variety of cyst just described is the *sterile*, as there is no re- ction of the tænia. A more common form is the vegetative or hydatid, characterized by the reproduction of other tæniæ and ıghter cysts within the mother cyst. As a rule, from the in- surface of the hydatid membrane, which may in this case be a germinal membrane, there arise small sessile eminences about ze of a millet seed which later become pedunculated; each of represents a daughter tænia furnished with hooks and suckers, vhose extremity is formed the vesicle which fills with fluid and surrounds the tænia. These small daughter cysts may become ed from the internal surface of the germinal membrane and n a free state in the mother cyst. But the same cyst formation ıke place in their interior and so on to the third or fourth tion. The parent cyst thus becomes filled with a large number cles which by rupturing give exit to younger cysts, until, as in are cases, thousands are formed. This fertile cyst is the more ıt form and the more deserving of attention from a therapeutic oint.

e process of involution of these cysts, due to death of the e whether it occurs naturally or artificially, is as follows: the l fluid is largely reabsorbed, the cystic membrane becomes and wrinkled, the contents become dense and syrupy or caseous, ous capsule retracts, and the cyst little by little disappears, mes leaving in its place a calcareous infiltration surrounded by ial connective tissue. An important form of cystic involution

from a clinical point of view is the suppurative; this will be treated of among the results of the disease.

Hydatid cysts of the liver may be single or multiple; Murchison has found as many as one hundred in one case. They may be situated in any part of the liver, but are usually found in the right lobe and upon the convex surface. At the autopsy cysts of the same nature may be found in other organs.

Biological researches have been made as to the development of the tænia echinococcus, as tænia nana (of Van Beneden) in the intestine of the dog family, and tænia expansa in herbivorous animals. The latter are frequently subject to cysts caused by taking in the ova of the echinococcus with the grass which they eat, upon which they have been deposited directly with the excrement of dogs, or indirectly in water used for irrigation. They develop in the tissues of these animals and give origin to cysts; ingested later as meat by dogs and kindred animals, and finding in their intestines conditions suitable for their development they fasten themselves to the walls, and develop as adult tænia.

Van Beneden in 1857 demonstrated this fact experimentally by taking a number of fertile cysts from the hog, and making two puppies ten days old who were still suckling, and therefore not infected with tænia, swallow a little of the cystic fluid filled with echinococci. A post-mortem examination showed an enormous development of adult tænia in the intestines.

Symptoms.

The presence of hydatids in the liver may cause no morbid symptoms whatsoever and be entirely unperceived. Central cysts of small size, and postero-inferior cysts may run through all the stages of their evolution without causing more than perhaps a sensation of pressure or of heaviness in the right hypochondrium, or a dull pain which may extend to the shoulder, but nothing of sufficient importance to suggest the correct diagnosis. Disturbances of any severity are caused only when the cysts are of a considerable size, when they are superficially situated, or when by compression of the bile or portal vessels they induce marked functional disorders.

Some authorities hold that there is an initial period in echinococcus cysts characterized by a sense of weight in the right hypochondrium, epistaxis, and often a continuous paroxysmal pain in the right shoulder. Dieulafoy[13] has pointed out that in this stage there may be attacks of urticaria due to reabsorption of the cystic fluid which by reason of the toxins contained acts upon the vasomotor

ous system. The same writer noticed in this latent period a what strange phenomenon, consisting in the patient's aversion l fatty substances. In some cases the initial manifestation is y pleurisy of the right side. In our opinion the existence of precursory stage is anything but well established; the symp- are only discovered when minute questioning is followed by a ul physical examination, and if a cyst is then diagnosed, the osed initial symptoms may as well be attributed to its presence. are certainly of no use in the diagnosis of a latent cyst.

he diagnosis of a cyst is made possible only by an objective ination of the liver. As a rule, this examination should be anteriorly, cysts being commonly situated upon the anterior on of the convex surface of the liver. A tumor will be seen in the hypochondrium and in the epigastrium which is globular in and which pushes the ribs outwards. This tension is one of ost valuable signs of hydatid tumor. Upon palpation the tumor to be smooth and very elastic; when tension is great, the sense ctuation is clearly perceptible, and when only moderate, by ap- iate manipulation we may appreciate the most prominent part tumor, and undulation as well, if the cyst is large. Fremitus a frequent or constant symptom in echinococcus cysts; its im- nce and its pathogenic conditions have already been touched

Unless the tension be very great, we may by palpation feel arp inferior margin of the liver, which, when involved in the formation, will have a prominent nodule at some one point is continuous with the main tumor.

rcussion will define the limits of the tumor and its continuity he liver dulness, which is of great importance in the diagnosis situation. The outline of dulness will show us the change in of the liver, which is not evenly enlarged; this is owing to t that the tumor is autochthonous and involves only a portion hepatic tissues.

e foregoing are the cases of most frequent occurrence and easy sis, but there are other cases whose mode of development is re complicated and leads to frequent errors in the diagnosis, instance, when the cysts attain enormous proportions and push ls into the thoracic, or downwards into the abdominal cavity. the cysts are situated upon the postero-inferior surface of the ney are apt to develop downwards in the direction of least re- e and fill the whole right side, sometimes even invading the ssa. They begin as sessile cysts, but may later become pe- ated so that a deep depression may exist between the tumor e liver which is liable to mislead in the diagnosis. The cystic

tumor in these cases moves with the respiratory movements, and deep palpation will enable one to appreciate the fact that it is continuous with the liver.

Cysts which develop anteriorly cause no such functional phenomena as ascites, jaundice, splenic enlargement, development of the collateral circulation, etc. When, however, they extend in a downward direction we have all the above symptoms of pressure of the hilum of the liver. The most frequent diagnostic error is that of mistaking these cysts for right hydronephrosis. We have, however, the following important differential symptoms: 1. The cystic tumor responds to the respiratory movements whenever it is of sufficient size to reach the iliac fossa and thus immobilize the diaphragm; this movement is absent in the renal tumor; 2. The renal tumor is longer than it is broad; the cyst is largest in its transverse diameter; 3. In the case of cysts the left lobe of the liver is usually somewhat hypertrophied; 4. In the case of hepatic cysts the intestines are usually displaced downwards; in hydronephrosis they are pushed forwards; this may be appreciated by means of percussion and palpation, which give a sense of crepitus when the intestines are compressed; 5. An echinococcus cyst is not in such direct contact with the posterior abdominal wall as the tumor of hydronephrosis, and when the patient is placed on the hands and knees, it falls forwards and leaves the posterior wall.

Carderelli points out these further diagnostic signs: 1. The cystic tumor gives no ballottement such as may be felt in the renal tumor; 2. In hydronephrosis an impetus may be transmitted from the anterior wall of the tumor to the posterior abdominal wall, but this is not the case with cysts because they are not closely connected with it.

Echinococcus cysts, as we have said, may develop upwards, pushing up the diaphragm and invading the thoracic cavity; this occurs when they are situated upon the convex surface of the liver. In this case they may easily be mistaken for a pleuritic exudation. It is of importance to the diagnosis to determine the superior outline of the dulness. This is not possible when the cyst is of such a size as to ascend as far as the second rib. In this case we shall have to be guided by the following differential signs, given by Cardarelli: 1. In pleuritic exudations the right side of the thorax is more or less deformed, but not uplifted as a whole; the ribs are divaricated and the intercostal spaces are protuberant; in cysts the thoracic walls are pushed upwards and outwards *en masse*, and the intercostal spaces are unchanged; 2. In pleuritic effusions the heart is displaced downwards and to the left, in cysts it is either pushed upwards or is not displaced at all; 3. In cysts there is complete silence upon ausculta-

the subclavicular fossa, while in even extensive pleuritic effu-
here will always be some respiratory murmur audible. If these
re carefully sought for, it will not be possible to fall into any
rror.

Course, Duration, and Results.

is not, as a rule, possible to estimate the duration and course
st, since the onset is so insidious and since, owing to its latent
it may acquire a considerable volume without causing any
lar disturbance to the patient, and without being perceived by
ysician unless he make a thorough physical examination of
r. It may also occur abruptly with the causation of symp-
ifficult to diagnose. Variability in course and duration is a
eristic of this disease. One important diagnostic sign it pos-
which is easily comprehensible from a knowledge of its patho-
anatomy. The cyst is developed at some one point of the
parenchyma, without either directly or indirectly causing any
tial or parenchymatous morbid lesion in the rest of the gland,
e exception of the production of a membrane of sclerotic tissue
ne of atrophied parenchyma directly around the tumor. The
erefore, unless its situation be such as to cause compression of
ulatory or biliary vessels, may attain great proportions without
g nutritive or functional disturbances of the liver substance.
s reason the functional hepatic disturbances which variously
e digestion, absorption, urogenesis, protection against toxins
tions, etc., are entirely absent. The general condition of the
is unaffected, nutrition is normal, and we have none of the
ns of hepatic insufficiency, namely, urobilinuria, increase of
oxic coefficient, and alimentary glycosuria. This fact is the
portant because it is in contrast with the marked objective
the organ, and the direct opposite of the conditions usu-
d in lesions involving the parenchyma.

possible terminations of hydatid cysts should be borne in mind
he course of the disease. A cyst may, as we have already
ndergo spontaneous resolution, which is proved by the fact
s of former hydatids are often found in the liver at autop-
e cure results from death of the echinococcus, especially in the
terile cysts, or the acephalocyst of Laennec. The death of
ite is probably due to an exudation of bile or of blood into
c cavity which would cause chemical alterations injurious to
a in the cystic fluid. Such a result is, however, altogether
al. The cyst, as a rule, ruptures; its eccentric development
a pressure atrophy in the hepatic tissue, so that while the

the fever of a pyæmic type and the vitiated general condition are especially to be noted. As Cardarelli has pointed out, a valuable diagnostic symptom consists in the marked contrast between the rapidity in appearance of the suppurative symptoms and the great change in the size and shape of the liver.

As to the conditions determining suppuration of the cysts, Chauffard and Widal by their valuable experimental researches have demonstrated that although the cystic fluid is itself aseptic, it is an excellent culture medium for pyogenic micro-organisms, but that the hydatid membrane filters out bacteria while allowing free passage to chemical substances and to toxins. It would appear, therefore, that the pus microbes reach the cystic cavity either through a pericystic inflammation involving the hydatid membrane, or through the entrance of bile or blood from rupture of a vessel. According to Raffi the biliary passage is the more frequent, according to Dupré ascending angiocholitis. That entrance through the blood-vessels is possible has been shown by Letulle, who reports a case in which suppuration followed a phlegmonous tonsillitis, and by Mori, who reports one in which it followed typhoid infection.

In our opinion the portal system as a means of carrying infection to the cysts deserves attention and study, because the intestines are constantly traversed by the most varied forms of pyogenic organisms. In some cases, now fortunately rare, suppuration has occurred after a badly conducted aspiratory puncture. The pus of suppurating cysts is, as a rule, sterile, for the reasons given when treating of abscess.

The symptoms and results of suppuration of cysts and of their rupture into various organs and cavities are very similar to those of hepatic abscess. When a cyst has undergone suppuration it is more liable to rupture, the process being essentially aggressive and inflammatory in relation to neighboring tissues and organs.

Diagnosis.

We have already considered the differential diagnosis between cysts and some other forms of disease; it remains to note the symptoms which enable us to distinguish cysts not only from hepatic diseases but from affections of other organs as well. The important diagnostic signs in cysts are: 1. The general physical condition; 2. Enlargement of the liver with deformity of the organ; 3. Aspiratory puncture, which should always be performed before a diagnosis is made, and should be attended by all antiseptic precautions; 4. Attacks of urticaria.

An examination of the cystic fluid will, on account of its special

cteristics which have already been described, materially aid in iagnosis. The hooks of the tænia may be absent, especially in ase of sterile cysts. The presence or absence of albumin will ate the death or life of the echinococcus.

Prognosis.

ognosis should be reserved, because of the possible conse- es of rupture into the thorax, peritoneal cavity, biliary ducts, nd of suppuration, which is a dangerous complication. Sterile end in recovery more often than fertile cysts.

Treatment.

thing can be hoped for by internal medication. Brilliant re- ollow surgical procedures alone, and these may range from aspiratory puncture to laparotomy.

diagnosis of cyst once established, it should be entirely evac- f its fluid by Potain's or Dieulafoy's aspirator, the operation f course done under all antiseptic precautions. A good result llows. If the echinococcus is dead, the fluid will naturally reproduced; if alive, puncture may cause its death. Absolute st be enforced after the operation. Urticaria may occur from rance of a small amount of fluid into the peritoneal cavity and rption in that situation, but it is of short duration.

uld simple aspiration not suffice to a cure, we must resort to ction of antiseptic solutions. Baccelli, in 1887, suggested the ichloride of mercury, 30 c.c. (7½ dr.) of the cystic fluid being d and 20 c.c. (5 dr.) of bichloride solution (1:1,000) injected. ocedure may be repeated, if necessary, in from fifteen to days. Many cases of complete cure by this method have orted.

ve modified Baccelli's method by removing all the cystic fluid cting 500 c.c. (16 fl. oz.) of Van Swieten's liquor, which was lace for from five to ten minutes and then completely re- he cavity, as a precautionary measure, being then washed sterilized salt water.

elli's method is to be preferred for the reason that in the case cysts, which are not infrequent, a simple washing of the ill not kill the daughter cysts, protected as they are by their but as this capsule admits of the passage of the antiseptic is well, as Chauffard and Widal have shown, to leave them time in contact with it. Baccelli's method overcomes sup-

puration to a certain extent, because, as these investigators have shown, 36 gm. of a 1:1,000 bichloride solution will prevent the development of pyogenic micro-organisms in a hydatid cyst containing 2 litres of fluid.

Piazza Martini in 1887 suggested endocystic injections of 20 gm. of a 1:100 solution of acetic acid, preceded by the removal of an equal amount of the cystic fluid, and he obtained good results.

At the last International Medical Congress held in Rome in 1894, Berucco, of Madrid, reported excellent results obtained by extracting a third of the cystic fluid and injecting an amount equal to a third of that extracted, of a 1: 500 solution of silver nitrate. He claims that this agent by precipitating the chlorides so changes the vital conditions of the parasite that it dies. Finally, before resorting to radical surgical procedures we should try the effect of treatment by electrolysis combined with iodide of potassium, whose value in hydatid cysts was first demonstrated by Semmola.

Surgical intervention will vary according to the case and to the situation of the tumors. The best and most certain results are obtained by laparotomy, performed with all the adjuncts of modern surgery, and according to methods varying with the situation of the cyst. In the case of anterior cysts, lateral or median laparotomy would be preferred; for postero-inferior cysts, the lumbar incision; for antero-superior cysts Lannelongue's method, and for postero-superior the transpleural incision of Ségond (see treatment of hepatic abscess).

Of course all physiological and symptomatological indications will be met as required.

Multilocular Echinococcus Cysts.

(Alveolar Hydatid Cysts.)

Virchow was the first to establish the parasitic nature of this tumor and to differentiate it from colloid cancer of the liver.

It has not as yet been decided whether the parasite is the same as in the case of unilocular cysts; Bollinger and Klemm believe that it is. They claim to have caused the development of the dwarf tænia in a dog by the administration of substances taken from a multilocular cyst. According to Müller, however, the echinococcus of multilocular cysts differs from the other in having smaller hooks, and in the possession of an ovary filled with ova in the terminal segment of even old tæniæ. Blanchard holds that a different mode of development,

genous in the case of unilocular echinococci and exogenous in ase of multilocular echinococci, accounts for the fact that the er are found in the hepatic parenchyma, the latter in some passage dy formed. According to Virchow, this would be the lymph- according to Leuckart the blood-vessels, and according to lreich the biliary passages.

he *anatomical changes* are characteristic. The greatest develop- occurs on the right side in the form of a tumor composed of a resistant stroma, sponge-like in its arrangement and enclosing l or elongated cells, varying in form and number, but always rous. Each one of these cells is composed of a fibrous mem-, a gelatinous hydatid membrane, and a central cavity contain- ie tænia attached to its wall and a yellowish caseous fluid in may be found the characteristic hooks, biliary pigment, ls of cholesterin, a few special mononucleated cells (Rendu). The aggressively propagating nature of this form of echino- is very distinctive, and causes it clinically to resemble a can- tumor; it tends to invade the biliary ducts, the lymphatics, ranches of the portal or suprahepatic veins, and the inferior ava itself, and thus gives rise to grave morbid symptoms. unilocular cysts, multilocular cysts may often remain latent. ver undergoes important changes. It becomes enlarged, and palpation we find the surface to be covered with hard excres- resembling cancerous nodules. The spleen is enlarged, and and jaundice are liable to occur from obstruction of the por- biliary vessels.

e disease is chronic in its course and may have periods of ration or exacerbation. The duration is long (six to nine The result is eventually death.

e *diagnosis* is difficult because of the rarity of the affection. It n confused with hypertrophic cirrhosis and with cancer. In mer, however, the enlargement of the liver is uniform, and in ond we have the special cachexia, the more rapid course, and e of splenic enlargement. Exploratory puncture is of great nce in the diagnosis, but the cystic fluid is difficult to remove unt of its density.

treatment of this form of cyst is very unsatisfactory, even urgical measures are adopted, on account of the large number communicating chambers which prevent opening all the cyst. on of a portion of the liver, an operation first performed by of Bologna, is the only one which holds out any prospect of result in cases in which the lesion is diffused but can be re- *en masse*. The measure is a heroic one, and may itself re-

sult fatally. In the majority of cases we are obliged to be satisfi with addressing treatment to the symptoms.

NEOPLASMS OF THE LIVER.

New growths may arise within the hepatic parenchyma, whi nearly always belong to the class clinically defined as malignar Carcinoma and adenoma are the most frequent. More rarely we fi cavernous angioma, sarcoma, and fibroma, which are, therefore, mo important from the standpoint of pathological anatomy than fro that of practice.

Cancer of the Liver.

Etiology.

Without entering into the immediate causation of carcinomata, must yet distinguish two clinical forms, the primary and the secon ary, the latter of which is the more frequent. It usually arises metastasis from those organs in which the portal system originat —the stomach, rectum, intestines, pancreas, etc.—but it may also due to arterial metastasis when there exists cancer of the breas uterus, testicles, or other organs.

Primary carcinoma of the liver occurs for the most part at advanced period of life (between the ages of forty and sixty), and men rather than in women. A few well-authenticated cases have c curred in childhood. It would seem that the causes already enume ated as influencing the functional powers and nutrition of the hepat cells, such as alcoholism, malaria, etc., may play a part in determi ing this situation for a cancerous process.

Pathological Anatomy.

Primary cancer of the liver occurs in three different forms.

The first consists of those cases in which a cancer with few nodul is found, usually upon the right lobe. When single, it may atta such a size as markedly to enlarge the lobe and finally to transfor it into neoplastic tissue (scirrhous cancer).

Cancerous tumors are usually round in shape, and formed of ti sue which may be hard or soft, and white or pinkish-white in colc The nodules are sometimes sharply defined from the hepatic tissu but sometimes the passage from healthy to neoplastic tissue gradual.

occasionally find necrotic and hemorrhagic foci in the midst of ned centre. Nodules situated near the periphery often appear mbilicated from temporary absorption of a part of the softened crosed tissue. Metastases subsequently form in the interior liver, and the cancer then assumes its true nodular form.

second type is characterized by diffuse cancerous degenera- the liver, which becomes enlarged and the surface of which is ed as in cirrhosis. Upon section the hepatic parenchyma is be divided by more or less fibrous bands of connective tissue olated masses of soft whitish or reddish tissue protruding from t surface, and of a cancerous structure. This is cancer with is.

the third variety the seat of the cancer is in the periportal tive tissue. Around the branches of the portal vein we find nodules swollen and crowded together, and even confluent, in size from a millet seed to an egg. The liver is en-

histological structure of hepatic cancers is the same as that rcinomata. Cancers with cylindrical epithelium are rare, and ey occur, so far as we have been able to ascertain, the prolif- of epithelium proceeds from the hepatic cells or from the um of the bile ducts.

econdary cancer the nodules are developed from cancerous in the blood-vessels. In their later stages they partly replace atic tissues and in part infiltrate it along the course of the essels, causing atrophy of the parenchyma while the connec- sue proliferates. It is not known whether the hepatic cells liferate.

metastatic process may be more or less extended; cases have own in which the liver was completely studded with nodules us size. The subserous nodules appear like small whitish tions upon the surface, and when they attain a certain size umbilicated. The liver is enlarged and may attain enor- oportions, and its surface is covered with tuberosities. Its s altered in very much the same way as in nodular cancer. dary cancer the carcinomatous nodules will also be found in her organ.

lly, cancer of the liver may exhibit a deposit of pigment, nparts a color ranging from gray to intense black, the tumor en called *melanotic cancer*.

Symptoms and Course.

Cancer of the liver may pursue a latent course, and is occasional found in its initial stages at the autopsy of one who has died fr some intercurrent disease.

In the case of secondary cancer the symptoms of the hepa neoplasm may completely overshadow those of cancer in other gans (stomach, rectum, etc.), but in other cases the very oppos may obtain.

Occasionally a progressive marasmus leading to a fatal res may arouse suspicions of the existence of a malignant new growt but the autopsy will fail to reveal any lesion worthy of attention.

The objective symptoms are the most important. The liver m become huge, and is always somewhat enlarged. This increase size is very rapid, especially in the case of metastatic cancer, so th an increase may be noticed almost from day to day. Palpation w reveal still more important data relating to the form, the surfac and the consistence of the liver. This organ may be uniformly e larged, or it may be more or less deformed, according to whether have to do with a diffuse cancerous process or with nodules. T umbilicated tuberosities found in nodular cancer have a great dia nostic value. Frerichs recommends a careful palpation of the a terior margin, which is often the seat of these nodules, and whi may sometimes be even appreciated by inspection if there is ascites or meteorism. The response of the organ to the respirato movements is an important characteristic, but may be entirely wa ing if the cancer has attained a sufficient size to wedge the liver i movably in the abdomen. In any case it is hardened in consistenc and we may sometimes get a sense of fluctuation resulting from t breaking down of the tissues. Occasionally there will be peritone friction from secondary perihepatitis. Palpation usually caus pain, which may also occur spontaneously, and which is of a lan nating or piercing character, frequently radiating to the rig shoulder and arm. In some cases there is a vague sense of pressu and tension in the right hypochondrium rather than a definite pain

A pulsation propagated from the aorta, and quite different fr the diffuse pulsation of aneurysm may sometimes be felt. Function derangements may occasionally be noticed; icterus is frequent a depends upon the compression and occlusion of the hepatic or so large bile duct which induces stasis of the bile. It is progressive its nature, and may reach a point where the coloring is of a greenis yellow or even a bronze tint. Ascites is less frequent than jaundi

ın scarcely be called a rare phenomenon, for Gilbert and Hanot
shown by their statistics that it occurs three times out of five.
)ends, not upon stenosis of the portal system, but rather upon
neal irritation due to the presence of the peripheric carcinoma-
odules. Development of the collateral circulation is rare, either
se of the rapid production of the disease, or because of the viti-
ondition of the blood. The spleen is not enlarged, which is a
diagnostic value.

addition to the objective and functional symptoms which at-
ttention to the hepatic gland, there is a condition which may
he chief factor of the diagnosis. We allude to the cancerous
ia. The patients gradually become emaciated, the skin be-
thin, is easily taken up in folds, and is of a characteristic straw
the physical strength constantly diminishes, œdema of the
appears, and the general appearance is one of great suffering.
ce assumes an expression of hebetude. Stretched upon his
ımovable, the patient becomes apathetic to every one and to
ıing; without lamentation or expressions of discouragement he
ly endures the destruction of his own organism as though he
o be an irrevocable decree of fate.

he last stages it is not unusual to have fever, which is usually
ımittent type, with evening exacerbations, more rarely of a
an intermittent type. It is due to absorption of the cancerous
ts derived from the breaking down of the neoplasm.

ome cases the fever occurs in the early stages of the disease,
:h case it is caused by suppuration or by acute hepatitis
: cancer) and leads quickly to a fatal issue. Nervous symp-
ue to the fever or to autointoxication, may be present.

tro-intestinal symptoms are frequently prominent, and are
e first to occur. Loss of appetite, a sense of weight in the epi-
n during digestion, and nausea and vomiting are common oc-
es during the course of hepatic cancer, and may in some cases
of marked intensity. There is usually constipation, and the
ıre of a grayish-white color. The urine is diminished in
and dark in color. Hypoazoturia, as we have ascertained
own researches, is constant, and the amount of urea may
ced to a few grams in the twenty-four hours. Not only
insufficiency, but general inanition due to the anorexia and
e disturbances contribute to this result. Only a small
of nitrogenous matter is absorbed, only a small amount con-
he hepatic cells are injured, and therefore complete oxidation
ubstances resulting from organic combustion is interfered
Urobilin is found in the urine and sometimes albumin, with

all the signs of nephritis, and uræmic symptoms may lead to a fa issue.

A morphological examination of the blood will show all the sym toms of progressive anæmia with notable diminution of the red blo corpuscles, and a reduction in the hæmoglobin which is sometimes extraordinary degree. Marked leucocythæmia has been noted fr the early stages of the disease, and can therefore not be a result the general vitiation of the system; a diagnostic importance has some been attributed to this symptom which, in our opinion, it d not deserve.

Phlegmasia alba dolens, with painful œdema of the lower limb may occur in the last stages of the affection.

The course of the disease may be acute, subacute, or chronic. would appear, however, that an acute course is not only depend upon a rapid development of the carcinoma but upon the fact t there may be a latent period, and that the duration of the course is timated from the appearance of the severe symptoms. In other cas the course may be protracted through months and even years. would seem that the development of hepatic cancer occurs more ra idly in the young than in the old. As a rule, the duration is fr three to five months. Death, the inevitable result of this affecti may be due to the profound and progressive cachexia; but it m also occur from other determining causes, such as rupture of a brok down nodule into the peritoneal cavity causing septic peritonitis; enormous amount of ascites inducing grave respiratory disturbanc intoxication from reabsorption of matters produced by the breaki down of the nodules and the deficient functional powers of the liv intracancerous hemorrhages with rupture of the liver; dissolution the blood, which is of frequent occurrence and causes parenchymato and subcutaneous hemorrhages, or some intercurrent disease (diffu bronchitis, pneumonia, dysentery).

Eichhorst observed a case in which a cancerous nodule whi was adherent to the anterior abdominal wall finally eroded it a appeared externally.

There are a few details in regard to the symptoms of the vario forms of primary cancer which deserve mention.

The cancer occurring in the form of a diffuse infiltration is rapid development, and at an advanced stage there may be so decid a deficiency in the secretion of bile that it would seem that there we no hepatic cells left which were capable of exercising their function powers. The fæces then become colorless, although there is no jau dice; the amount of urea may be reduced to 50 cgm. (7½ gr.) in twent four hours. The peritoneum is not involved in this form of canc

is no ascites, nor does the patient complain of any pain. The ic tumor is distinctly felt on palpation, its surface appears to ooth, hard, and not appreciably deformed. The cirrhotic form ilar in its development to the nodular, but differs from it in here is no marked enlargement of the liver, while there is abun- scites.

Diagnosis.

e chief diagnostic signs are rapidly progressive hypertrophy liver and the characteristic cachexia. From these two symp- lone we should be able to judge of the nature of the disease. etimes happens, however, that although the diagnosis of can- y be correctly made, we may confound hepatic cancer with ne disease in other organs (pylorus, omentum, colon, pancreas,

order to make a correct differential diagnosis it should be n mind that the hepatic tumor gives complete dulness on per- and is displaced by the respiratory movements, while tumors other organs give a dull tympanitic resonance, and are very affected by the respiratory movements. The functional dis- es will serve as a guide in localizing the cancer; for instance, ill be digestive disturbances in cancer of the intestines, vomit- ancer of the stomach, etc. Cancers of the omentum are char- d by their great passive mobility.

affection may often be confounded with other hepatic dis- especially such as are accompanied by grave nutritive dis- es similar to the cancerous cachexia. It may be mistaken for *ic liver*, with which it has many characteristics in common ted tumor, deformed liver, icterus, ascites, œdema, cachex- ides to the differential diagnosis are: (1) The cancerous nod- e harder and smaller than gummata; (2) the course is rapid er, slow in syphilis; (3) the etiological data; (4) the age of ent; and (5) the effect of antisyphilitic treatment, which is per- e most important of all.

loid degeneration may be mistaken for the diffuse infiltrated cancer, unless we bear in mind the splenic enlargement, the uria, the pathogenic conditions, and the course of the former.

ertrophic cirrhosis may be diagnosed from cancer by the slow ment of the tumor, by its complete indolence, by the absence e phenomena for a long period of time, by the characteristic ons, and by the marked splenic enlargement.

nooth, rounded carcinoma, with a larger antero-lateral devel-

opment, may in form and consistence resemble an *echinococcus cyst.* The general condition of the patient should serve as a guide, as it is good in cysts, rapidly deteriorating in cancer. Exploratory puncture will decide the doubtful cases, as in cancer the most that can be obtained is a bloody fluid sometimes laden with cells.

Hepatic abscess of rapid development may often be taken for cancer, especially when there are fever and much pain. The etiology, the form of the tumor, and exploratory puncture should decide the case. Sometimes, however, these do not give sufficient assistance; the etiological data are absent, puncture does not reveal pus because the abscess is centrally situated, the enlargement simulates carcinoma, and yet the disease will be an abscess. We must, on the other hand, bear in mind that a cancerous tumor may be surrounded by a suppurative process, in which case the diagnosis becomes almost impossible. Only by noting every detail of the history and symptoms can we arrive at a correct diagnosis. In doubtful cases, when there is a possibility of abscess, exploratory laparotomy should be resorted to.

Prognosis.

As in all cases of cancer, death is the inevitable result. In this variety the course of the disease is particularly rapid.

Treatment.

The recent efforts to cure cancer by orrhotherapy, undertaken especially by Henricourt in France, and by the toxin of erysipelas as recommended by Coley, Emmerich, and Scholl, have not been substantiated, and therefore hold out no hope of a cure for hepatic cancer.

When it was demonstrated that resection of a part of the liver was practicable (Loreta), the endeavor was made to treat cancer by excision. But the bold efforts of Lücke in this direction were unsuccessful, because cancer of the liver is usually either diffuse or secondary.

There is therefore no therapeutic resource of value in these cases; and rational treatment will have to be purely symptomatic, consisting chiefly in a nourishing diet, and the administration of remedies for the gastrointestinal disorders which are so apt to supervene and to aggravate the patient's discomfort. For relief of the pain morphine in large doses may be given, for there is unhappily, by reason of the rapidly fatal course of the disease, no fear of causing a drug habit.

Adenoma of the Liver.

lenoma must also be considered among the epithelial neoplasms. net with in the liver in the form of multiple nodules, varying in om a millet seed to a hazelnut, the cut surface of which is of a ish-white or reddish color. The smallest ones appear to be liately surrounded by the hepatic tissue, the largest have a con- e-tissue capsule; these may undergo softening in the centre. nodules be many in number, the liver is enlarged and covered ounded excrescences which resemble those of nodular cancer. asis, however, rarely ocurs.

ıdfleisch and Eberth, who have given more attention to this f neoplasm than other investigators, found that each nodule is ult of the agglomeration and anastomosis of glandular alveoli, are developed from the trabeculæ of the hepatic lobules, the which proliferate and group themselves together in the form olar glands. These branch out and infiltrate and supplant the nding hepatic tissues. According to Bonome these alveolar may also arise from the biliary ducts and thus form true tu- ıdenomata. An active proliferation of connective tissue with uent sclerotic degeneration may occur around these adenomata. in[64] considered adenoma to be a complication of cirrhosis than a neoplasm; the nodule he thought was in the beginning cular hyperplasia, which, when in a more advanced stage of ıment, penetrated into the blood-vessels and diffused itself ı the parenchyma, thus acquiring the characteristics of a ma- neoplasm.

clinical symptoms of adenoma are similar to, and confound ves with, those of nodular cancer. Their course is, however, sometimes covering several years. The result is always fatal, h when the tumor is circumscribed modern surgical methods t some hope of a cure.

Bergmann reported to the German Surgical Society (1893) a extirpation, with resection of a portion of the liver, of a tumor as as large as the head of a child, and which under micro- examination was seen to possess the characteristics of an a. The result was excellent.

ɔmi, in the surgical section of the Eleventh International Med- ıgress held in Rome in 1894, presented a patient, a man of even years, from whom he had removed the left lobe of the ich was affected with tubular adenoma, developed from the ts.

Connective-Tissue Tumors of the Liver.

The connective-tissue tumors of the liver, while of interest from the standpoint of pathological anatomy, are not of great practical importance.

Sarcoma.

Primary sarcomata of the liver are of extremely rare occurrence. According to Arnold they may arise from the interstitial connective tissue of the gland, or from the connective tissue surrounding the blood-vessels and bile ducts. They occur in the form of nodules of varying size which penetrate throughout the liver and frequently cause metastases in other organs. As a rule they occur in young persons.

Secondary sarcomata are found more frequently, especially when the primary sarcoma is situated in the region of the portal vein.

Primary melanotic sarcomata have been found in the liver, but even these may be secondary to cutaneous melanosarcoma or to melanotic sarcoma of the choroid, the anus, or the rectum. The liver in these cases is so studded with brown or black nodules of various sizes that upon section it may have the appearance of granite. The lymphatic glands of the hilum are engorged, and the liver may attain an enormous size—7 to 9 kgm. (15 to 20 lbs.). Lancereaux states that there may be true melanosis of the liver without the production of nodular neoplasms, as a result of the infiltration of melanotic pigment from tumors in other organs, which invades the hepatic cells and the endothelium of the blood-vessels in the form of minute black granules.

The *symptoms* consist in the local phenomena of a malignant tumor of the liver, and a gradual systemic deterioration, so that they are absolutely similar to those of cancer. The diagnosis, therefore, in secondary sarcoma, when we do not know the seat of the primary disease, can be made only by means of the resources given us by pathological anatomy and histology, and even then is difficult. The fact that it occurs with some frequency in youth is something of a guide. The course of the disease is rapid and ends in death.

Some importance has been attached to the examination of the urine in melanotic sarcoma, as in melanotic cancer. When treated with active oxidizing agents, it is said to become brown or even black, and upon cooling there forms a black precipitate.

From the result of v. Jaksch's experiments, however, who obtained this reaction in healthy persons (from the presence of chromogens)

id not obtain it in patients affected with the disease under dis-
n, it would seem that it is not of great importance.
eatment is directed to a relief of the suffering, as nothing can
ıe to cure the disease unless the tumor be sufficiently circum-
d to be removed by surgical skill.

Cavernous Angioma.

mors of this nature may be found in the liver, and may be
or multiple, the size of a hemp seed or of the closed hand.
section we shall find them to be of a dark red color, and under
icroscope we shall be able to distinguish the true cavernous
ıre.
e largest angiomata are surrounded by a connective-tissue cap-
hich is entirely absent in the case of the small ones. The
s in the liver begins with a cavernous degeneration of the
ries, which dilate, proliferate, and cause compression atrophy
cells of the parenchyma. The occurrence of thrombi with
lasia of the connective tissue sometimes forms hardened masses
the angiomata. These tumors being absolutely exceptional
t causing serious disturbances, are of slight clinical interest.
ough errors in diagnosis they have occasionally been the sub-
surgical intervention; it is certainly unwise to perform so
n operation as resection of the liver except for a grave disease.
erg[95] has reported a case in which the hepatic cavernous angi-
ighed 470 gm.; resection of the liver resulted in a cure.
omata, lipomata, gliomata, etc., of the liver belong to the class
cal curiosities and are of no importance clinically.

DEGENERATIONS OF THE LIVER.

hepatic cells may from various causes undergo degeneration,
nay be secondary to circulatory or inflammatory changes, or
ı the other hand, be primary. The latter class is the one of
ve treat in this chapter, and more especially the fatty and
l degenerations which often constitute true morbid affections.

Fatty Degeneration.

is the form of degeneration of most common occurrence in
ıtic cells.
fat-forming function of the liver is, with the glycogenic, a
function. Even in health, the liver cells often contain small
ules in the protoplasm and towards the periphery, which are

easily demonstrable by treating with osmic acid. During digestion the fat in the cells increases in amount, for which reason it is probable that its origin is found in the absorption of the fatty matters resulting from intestinal chemism.

This increase in fat is also noticeable during pregnancy and lactation, probably because it is needed for the nourishment of the fœtus and for the formation of the milk. The nature of the food taken has a marked influence upon the amount of fat found in the cells. Frerichs has shown that when dogs are fed exclusively upon fatty substances the liver cells in a few days become the seat of a true fatty infiltration. According to Virchow's researches, confirmed by Rosenberg, the hepatic cell not only seizes upon the fat which comes to it in the state of soap in the portal circulation, but acts as an excretory organ as well, as it eliminates it through the bile, the fat being in part reabsorbed in the gall-bladder (intrahepatic circulation of fats). In addition to seizing and storing up the fats derived from intestinal absorption, the hepatic cell possesses the power of manufacturing fat from nitrogenous matters; this is proved by the fact that in dogs fed exclusively upon a diet of lean meat the characteristic fat granules are found in the liver cell, although in small numbers (Frerichs). It follows that fat may accumulate in the liver in one of two ways. There may be simply an increase of the fatty matters derived from digestion and deposited in the cells without alteration or destruction; or the fat accumulated in the hepatic cells may be due to direct degeneration of the nitrogenous matters of which they are composed.

Etiology and Pathogenesis.

From what was stated above, it is clear that there are two distinct processes, namely, fatty infiltration and fatty degeneration. They are also of clinical interest.

Fatty infiltration results largely from dietetic errors. Persons who eat an over-supply of fats and carbohydrates, and at the same time lead sedentary lives, not only have fatty infiltration of the liver, but also have a subcutaneous accumulation of fat which possesses all the characteristics of polysarcia. Voit and his followers have demonstrated that the formation of fat may occur directly from nitrogenous matters, but it is doubtful whether it is ever derived from carbohydrates. It would seem rather that these aid in the accumulation of fat by virtue of their being easily oxidizable nutritive products, and therefore reserve products which limit the combustion of fatty substances which as a result accumulate in the tissues.

While fatty infiltration is largely dependent upon an increased

nt of fat ingested and consumed, it may also be due to incom-
or slow oxidation of the tissues, and this not because of an over-
y of the substances to be oxidized, but because of a deficiency
ygen. This diminution in the amount of oxygen absorbed by
lood occurs especially in anoxæmia. When, from some dis-
d condition of the circulatory or respiratory functions, there is
ed oxidation of the tissues, the hepatic cells beyond all others
e laden with fat, which in the beginning is deposited as fat
ts but which finally invades and fills all of the protoplasm.
h cases, however, it would seem that while the process is at
erely a fatty infiltration, it changes later into a true fatty de-
tion of the cellular protoplasm; this may perhaps be due to
oxication, which so readily occurs during the nutritive inter-
s in anoxæmia. The increase in fat due to anoxæmia has been
nentally demonstrated by Velpeau and Dechambre, who suc-
in fattening dogs by subjecting them to slight but frequent
g.

his manner may be explained the fatty liver of progressive
ous anæmia, of grave chlorosis, leukæmia, the cachexias of
malaria, scrofula, and syphilis, of serious gastrointestinal
rs, etc. Among the diseases which cause a diminished ab-
n of oxygen through local pulmonary conditions, and conse-
hepatic steatosis, phthisis pulmonalis deserves the first place.
ver is very frequently met with in tuberculosis of the lungs;
s found it in sixty-eight per cent. of his cases, Louis in thirty
t. Many have been the discussions as to the cause of this
nce, and many the theories advanced. It has not as yet been
y determined whether the process consists in a fatty infil-
or a fatty degeneration, and the determination of this point
n truth be the first step taken towards a solution of the prob-
noxæmia, a surplus of fat in the blood due to the progressive
on constant in this disease, a diet rich in fats, the gastroin-
disturbances which usually accompany phthisis, medication
s of cod-liver oil or arsenic, the diminished interstitial com-
of fats, and finally a special action of the bacillus tubercu-
its toxin, are the various causes to which fatty degeneration
attributed.

most important of these in our opinion are anoxæmia and
ific action of the tubercle bacillus or its toxin, which, as
nd Lauth have shown, is endowed with fat-forming proper-
causes diminution in oxidation of the fats and at the same
ltration of the cells and degeneration of the cellular proto-

The etiological study of true degeneration of the hepatic cells is of the greatest interest. Two causes may act to produce it, namely, intoxications and infective diseases. Phosphorus and alcohol are the chief poisons which induce fatty degeneration of the liver. In the case of acute phosphorus poisoning the action is so rapid that in a few days the whole liver may be completely involved. There is in the first place a direct and destructive action upon the cellular protoplasm, and in the second place abstraction of oxygen from the blood. The action of alcohol is very similar, as is often noted in practice, and as Strassmann and Sabourin have proved by experimental researches. Arsenic, antimony, chloroform, carbonic oxide, and many other chemical substances also cause fatty degeneration of the liver.

Investigations in regard to the precise manner in which infective diseases act upon the liver have of late been numerous, and we may consider the fact to be proved that important histological alterations, if not visible lesions, are produced, and consist of more or less advanced fatty degeneration of the cellular protoplasm. Important hepatic lesions leading to this degeneration have been found by Siredey and Legry in typhoid fever, by Hanot and Gilbert in cholera, and by Widal and Pilliet in fevers and in puerperal eclampsia. Is this occurrence due to the action of the micro-organism or of its toxin? As the latter is constantly being credited with a more important rôle in the production of morbid lesions and symptoms, it would appear likely that fatty degeneration might well be attributed to their influence. The hepatic lesions met with in diphtheria, in which disease, as we know, the pathogenic bacterium does not enter into the circulation and therefore cannot act directly upon the liver, but whose toxin does circulate in the blood, show that microbic toxins have an elective action upon the protoplasm and the nutrition of the hepatic cells. Further and more thorough research is needed to fully elucidate the matter.

We have ourselves studied into the protective action of the liver in infections and intoxications, and hold that precisely because of this function the liver is liable to become the seat of the degenerative process. The protective power is not due solely to a separation or neutralizing of chemical or bacterial toxins, but also to the presence of phagocytes which are especially active in the liver. This double mode of action would almost seem to sustain the theory that fatty degeneration of the cells is due to direct action of the bacteria rather than of their toxins.

We must not omit the fatty degeneration of the liver consecutive to other morbid processes in the organ itself, already alluded to when treating of these diseases. More or less extensive degenerative

s may be observed in stasis of any of the hepatic veins, in the
titial inflammations of cirrhosis, in suppurative hepatitis, tu-
echinococcus, etc. The changes in the circulation and the sub-
nt check to the respiratory exchanges in the tissues must cer-
have some effect on its causation, but other factors also take a
such as autointoxications and secondary infections. Secondary
ic steatosis is an important anatomico-pathological fact and one
le of aggravating the primary disease.

Pathological Anatomy.

cording to Chauffard, there are three chief varieties of this dis-

Large fatty liver with total steatosis, with increase in size and
of the organ, but decreased specific gravity which may be less
hat of water. The color is a pale yellow or a reddish-yel-
esembling suet. The borders are thickened and rounded.
nsistence is softened, and digital pressure leaves a more or
ep impression. The investing peritoneum is smooth and
and may have dilated blood-vessels of stellate arrangement.
er tissue is only slightly resistant, and the cut surface is seen
ly and exsanguinated, it being possible to collect an oily fluid
ie knife blade. If the liver be cut with a heated knife a layer
eous matter will be found upon the latter when it cools. The
dder contains little bile, and that little is thin and pale in
reason of the small amount of bile pigment, and, according
r, of bile salts.
er the microscope the cells are seen to be filled each with a
ining fat droplet, which reduces the protoplasmic contents to
um, and pushes the nucleus against the cell wall. The pro-
is scarcely altered in character, however, for if we tease a
of the tissue we shall find that it is easily stained by the
ents employed. Fatty degeneration begins at the periphery
obules and gradually penetrates towards the centre. The
usually commences as an increase in the number of fatty
ions of the protoplasm, small droplets of fat are then formed
lly run together and form the one large drop which occupies
ie whole cell. Some have considered that the presence of
l granulations indicated fatty degeneration and the single
ty infiltration; but as may easily be proved, these are but
stages of the same process, and it is not possible micro-
y to distinguish between the two forms which are pathogen-
parate.

A clinical examination of fatty liver shows the enormous amount of fat present, which may increase from 3 or 5 to 78 per cent. (Frerichs). The amount of water is diminished, that of lecithin is increased (Dastre and Moret). Frerichs believes that it is possible to judge by the amount of fat in relation to the diminution of water whether the case is one of infiltration or of degeneration. In the former the fat is increased and the water diminished; in the latter the water remains about the same in amount, and the fat is greatly increased.

2. *In fatty liver with partial steatosis* the lesions described above are found only here and there in the liver. This is well shown in the nodular partial fatty hepatitis of Sabourin, in which the process follows the course of the periportal system, while the suprahepatic zone remains normal.

3. *Granulo-fatty degeneration* is the form especially seen in infectious diseases. The lesions are microscopic, scarcely any change being perceptible even under the microscope. The process begins as a cloudy swelling; the hepatic cells swell and become opaque and turbid, and this is followed by the appearance of numerous fat granules, which are clearly seen as black spots when treated with osmic acid.

Symptoms and Course.

Fatty degeneration of the liver is initiated and runs its course without causing any special phenomena, so that often its existence is to be inferred from the morbid causes which may produce it rather than to be demonstrated.

As it may be the result of grave general or local affections, the functional disturbances of the liver are usually completely masked by the symptoms of the primary disease, the more so that in conditions of extreme cachexia or anæmia but few demands are made upon the liver, owing to the small amount of food ingested or to the digestible and assimilable nature of such as is taken. The processes of organic oxidation are thus so slow and limited in extent that the part of the liver which has remained unaltered is usually sufficient for all needs. At an advanced stage of the disease, however, there may be symptoms of hepatic insufficiency which are of enough severity to attract attention. The first symptoms often relate to the enlargement of the liver, the patient complaining of a sense of tension and pressure in the right hypochondrium, or there may even be sharp pain, due in all probability to excessive tension of the capsule from the increased size of the liver.

In persons suffering from polysarcia, the fatty infiltration of the

may be entirely masked by the enormous development of the taneous abdominal fat, while the hepatic protoplasm is as yet ently normal to prevent the occurrence of any marked functional oms.

e hepatic steatosis which accompanies grave infectious diseases nterest at the autopsy rather than during life. Yet there are a mptoms which relate to the anatomical conditions in the gland, hich may assist in the diagnosis. The liver is uniformly en-, and upon palpation will be found to extend three to four ' breadth beyond the costal arch, the superior line of dulness tending beyond the normal. If the abdominal walls are thin to allow of it the surface will be felt to be smooth and some-softened. The inferior border is not especially changed, but s to be more rounded than usual, except when the fatty degen- is the result of hepatic disease, such as cirrhosis. There is ites or development of the veins upon the surface of the ab-, no jaundice or splenic enlargement; the fæces may be dis- from intestinal acholia due to a deficient bile production by enerated hepatic cells. The other organs, especially the heart lneys, may be involved in the process of fatty degeneration.

examination of the urine will show the insufficiency of the he-ells; hypoazoturia may be marked, owing to the general ca- condition; urobilinuria and glycosuria are frequently met nd Lépine considers the presence of phosphoglyceric acid to iognomonic of the disease. The patient's general condition ly deteriorates; the skin, especially that of the face, becomes uliar pallor, which Addison compared to alabaster. A state hy and hebetude gradually comes on and the patient goes d to worse. Frerichs has noted that the skin of these pa- s oily from excessive development of the sebaceous glands. form of autointoxication may cause death, but as a rule this o the primary disease.

attention of surgeons has been called to the danger of opera- diseases of the liver in general, and of fatty degeneration in ar. According to our views, this is because the liver is a g agent, and is usually in a measure able to compensate for complications following operation; because of its impaired in the cases under discussion, even a slight surgical infection rove fatal. Under the present *régime* of asepsis and of anti- however, no skilful operator need fear such a result.

Diagnosis and Prognosis.

The diagnosis, which for reasons above stated is obscure, may be made by a careful study into the determining causes of the disease.

The prognosis depends absolutely upon the nature of the cause and the degree of injury to the liver.

Treatment.

The physician is rarely called upon to treat fatty degeneration of the liver, the primary disease claiming his full attention. Treatment must always be addressed to the cause. A reducing treatment in polysarcia, the prohibition of alcohol, and the prescribing of remedies appropriate to the condition of anæmia and cachexia will indirectly meet the indications for a rational therapy.

Fatty substances must be eliminated from the diet, as they serve to increase the fatty infiltration of the hepatic cells; food consisting chiefly of nitrogenous matter will be compatible with the functional powers of the intestines. Consumptives should not be given cod-liver oil or arsenic when there is fatty degeneration; as we stated above, some authorities consider that these drugs may cause the disease. A rational symptomatic treatment should of course be instituted.

Amyloid Degeneration.

Amyloid degeneration is a process which may invade all the parenchymatous organs and tissues, and occurs with especial frequency in the liver. It was indeed first described in relation to the liver by Virchow, although Meckel and Rokitansky had already given a fairly complete macroscopic description of the morbid process.

Etiology.

Amyloid liver never occurs as a primary disease, but is always secondary to morbid lesions of other organs and tissues. Generally speaking, it may be attributed to the profound exhaustion consequent upon various cachexias. It is chiefly found following suppurative processes, those of the bones in especial, which are of long duration and induce a marasmic condition. It also occurs after suppuration of the lymphatic glands, after empyema, suppurative psoitis, paranephritis, pyonephritis, etc.

Less frequently amyloid degeneration of the liver and of other organs occurs as the final result of chronic infective processes accom-

ɔd by profound cachexia. Tuberculosis of the lungs, bones, uri- tract, and other systems, syphilis, and malaria are the most ent causes of amyloid degeneration. It may also occur during ourse of chronic constitutional affections, such as rachitis, os- alacia, leukæmia, pseudoleukæmia, Bright's disease, malignant rs (carcinoma, sarcoma, lymphosarcoma, etc.), and digestive ders which affect nutrition. Finally, amyloid degeneration of ver may occur to a greater or less extent with other hepatic dis- , such as tuberculosis and syphilis of the liver.

ıe condition is found more frequently in men than in women, ıbly because they are more subject to the morbid causes, and often in youth and adult life than in childhood or old age. The se is always acquired, with the exception only of that of syphi- rigin, which may be congenital.

Pathological Anatomy and Pathogenesis.

the condition be in a sufficiently advanced stage the macro- signs are usually well marked. The liver as a whole is so en- l as to be sometimes even more than double its normal size, but goes no change in form, with the exception that the anterior r is rounded and thickened. The weight is increased. Its sur- smooth and shining, its consistence harder, and in color it is haracteristic yellowish-gray tinge. The substance is more re- t than usual to the knife, and the cut surface has a special lar- ıs appearance, which has caused the condition to be called *lar- s liver*. Thin sections are somewhat transparent in the light. be treated with a drop of Lugol's solution (iodine-iodide solu- he degenerated portions will become of a deep red color, while ılthy parts are a light yellow. Methyl violet colors the affected ıs a purplish red, the normal parts becoming an intense blue l). Saffron stains the amyloid portions yellow and the re- er a reddish pink. These and other reactions will serve to dis- ıh the lesion when not at a very advanced stage, and the two ıentioned are the methods of staining to be preferred in histo- researches.

ıen amyloid degeneration of the liver is far advanced, the spleen, s, and intestinal walls are also involved in the process. By of microchemical reactions we have been able to follow the s from the beginning to the end. It commences in the ar- the primary lesion appearing in the median zone of the he- obule, in the connective-tissue walls of the branching capil- when it appears in the form of a thin vitreous layer, the

endothelial cells remaining normal. The amyloid substance next appears as small parietal masses in the interlobular arterioles, the portal branches, suprahepatic radicles, and even the hepatic cells becoming progressively involved. The latter when affected are swollen and vitreous in appearance, while the nucleus and the normal pigment and fatty granules disappear. They may, on the other hand, undergo atrophy from pressure by the amyloid substance which infiltrates the intralobular capillaries.

The clinical conditions of amyloid liver have been the subject of much research. Frerichs has never succeeded in finding either sugar or glycogen, which demonstrates how completely the hepatic cells have lost their functional powers. Dickinson found a marked diminution in alkalies, for which reason he believes that the pathogenesis of the affection is in relation with the great loss of alkaline matter sustained by the organism in suppurative diseases. The chemical nature of the amyloid substance has been the subject of much discussion. Virchow, who gave it its name, considers it to be a tertiary compound, similar to starch and vegetable cellulose. But the latest researches of Friedreich and Kékulé, Schmidt, etc., have demonstrated that it is a nitrogenous substance differing from albuminoids, in that it is unaffected by pepsin and resists putrefaction.

It has been, and still is a subject of discussion whether amyloid liver is due to degeneration or to infiltration of amyloid matter preformed in the blood. The second view would seem to be sustained by the fact that the first degenerative lesions are found as a deposit upon the arterial walls, while the cellular lesions are the last to appear, and even, according to some authorities (Wagner), never do occur. This would presuppose biochemical changes in the albuminoids, as a result of the nutritional disturbances due to cachexia, sufficient to convert them into amyloid substances which would infiltrate the various organs, and finally be deposited within the cells.

The amyloid substance has as yet not been found in the blood; its presence there would be a complete proof of this theory, which we incline to believe because it seems to throw light upon the etiology and the histogenesis. The absence of the amyloid substance does not, however, prove the theory to be unsound. It might be deposited as soon as formed, in the liver and other parenchymatous organs, and therefore exist in the blood in amounts too small to be detected by microchemical researches, which, moreover, have not as yet reached any considerable degree of accuracy.

Quite recently Bouchard and Cornil have suggested that the pathogenesis of amyloid degeneration may be dependent upon the specific action of bacteria or of their toxins in the cells; in some

indeed, these investigators were able to produce true amyloid
itis in rabbits poisoned by pyocyanin or inoculated with tuber-
s. Although this result may and does in a measure elucidate
thogenesis of amyloid degeneration, further researches will be
ary before we can unhesitatingly accept the conclusions reached
se experimenters.

li[66] found sulphochondroitic acid in the amyloid liver, which
pposed to have been formed in the cartilages and set free by the
nd cachectic condition. He experimented upon dogs and rab-
th the sodium salt of this acid, but was not able to produce a
nyloid degeneration. In one case he observed nuclear degen-
of some of the cells.

Symptoms.

yloid degeneration of the liver, as a rule, runs its course with-
sing any morbid symptoms, a fact which is explained by the
ico-pathological conditions to which we have already called
n. The hepatic cell not being primarily attacked, but re-
g normal for a considerable space of time, and there being no
ance in the portal or biliary circulation, the functions of the
e but slightly interfered with.

symptoms are almost entirely objective, and relate to the in-
area of liver dulness; they simulate those of hepatic tumor,
sess certain characteristics important to the diagnosis. An
d prominence of the right hypochondrium and the epigas-
sense of tension and oppression especially after eating, will
e attention of both patient and physician to the liver. This
ill be found to be enlarged and to extend beyond the costal
ts form is unaltered, its surface smooth, its consistence hard-
l even stony; the antero-inferior border may be perceived by
on if the abdominal walls are thin, and is found to be hard
nded, presenting the two normal notches. Percussion will
es demonstrate that the liver is enlarged in an upward di-

The organ may become enormous; in a young girl suffer-
suppuration, of tuberculous origin, of the left foot and of
hatic glands of the neck, we found an amyloid liver of such
as to fill nearly the whole of the abdominal cavity. The
also enlarged from degeneration of the same nature. There
ndice, nor ascites, nor development of collateral circulation.
is found in the urine, and there are signs of amyloid degen-
of the kidneys, if they are involved in the process, as they
are.

functional condition of the liver remains for a long time un-

impaired, wherefore there is little hypoazoturia or urobilinuria, nor is alimentary glycosuria produced. The urotoxic coefficient is not increased, as we have been able to demonstrate in cases in which there was neither suppuration nor infectious process; in some cases it would appear to be diminished, a fact explained by the lowered general nutrition accompanying profound cachexia.

Intestinal disorders are of frequent occurrence, especially in the later stages of the disease, and are manifested by profuse mucous diarrhœa, whitish in color, which oftentimes is the immediate cause of death. These disorders appear to be due to amyloid degeneration of the intestines.

The general condition grows progressively worse, the coloring becomes of an earthy pallor, which, according to some authorities, is characteristic, the blood becomes poor in hæmatocytes and hæmoglobin, and the patient falls into a condition of marasmus which, as a rule, leads to a fatal result.

The duration of the disease is from one to several years; the prognosis is bad, although several authorities claim that a cure is possible if the process is in the initial stages and the cause can be treated and overcome.

Diagnosis.

We have already given the differential diagnosis of amyloid liver. Important and fixed points in the diagnosis are:

1. The etiological factors, which are always present;
2. Coexistent degeneration of the kidneys, spleen, and intestines;
3. The objective signs of hepatic enlargement, especially the hardness, the roughness of the surface, and the condition of the border already described.

Should the etiological factors be such as to permit of a diagnosis of fatty liver, the last two points will serve to distinguish between the two diseases.

Treatment.

There is no curative method known which is capable of acting directly upon amyloid liver. Treatment should be largely prophylactic, and we should endeavor with all means known to surgery and medicine to combat the causes of the degeneration. Old and profuse suppurations should be checked, tuberculous bones should be removed, chronic malaria and syphilis should be treated hygienically and medicinally, etc. Even if degeneration have begun, energetic treatment should still be directed to the cause, as thus alone can any good results be hoped for.

nic and reconstituent medicines such as quinine, arsenic, and nd a rational treatment addressed to the symptoms, will com- he therapeutic measures possible in this disease.

DISEASES OF THE BILIARY PASSAGES.

hough they differ widely in their nature, we deem it best to e all the diseases of the biliary ducts in this section. We shall all treat of jaundice as a morbid entity, having in the general this work considered it as a symptom and having treated of hogenesis.

Classification of Jaundice.

to within a few years catarrhal jaundice was the only variety rus known, but recent studies into infective processes of the passages have extended the boundaries of diseases character- iefly by the symptom of jaundice and by primary or secondary of the biliary ducts, sometimes accompanied by functional or ical alterations of the hepatic cells.

he first place the discovery was made that jaundice could exist preëxistent intestinal catarrh and without any special ascer- cause. It was also noted that some cases of icterus were in nature, resulting with more or less rapidity in a cure, while ses resisted all treatment and led to a fatal issue. A careful clinical data also showed that a benign form of icterus might the character of a grave and fatal jaundice. These facts drew ntion of pathologists and practitioners to the forms of icterus tiology was obscure; and when bacteriological investigators riumphantly to invade the field of the pathogenesis of many , attention was directed to this hepatic disorder from the bac- ical point of view. At the same time, practitioners began to at icterus sometimes occurred in epidemic or endemic form, g to certain special hygienic conditions, and this fact gave support to the experimental researches. As we pointed out neral pathology, these researches established the fact that patic diseases may find their determining cause in infectious l primary lesions of the bile ducts. Thus acute yellow atro- long considered to be the most characteristic manifestation nary lesion of the hepatic cells, is now known to be due to s or local causes brought to the cells and the excretory bile the biliary or the portal circulation.

and various classifications have been made of jaundice, those é and Chauffard[3] deserving chief consideration, although

based simply upon prognostic and symptomatic data, and therefore deficient in clearness. Between each variety is found a whole intermediate series which cannot be placed in any one group, and one group also may pass by stages into another. The course of the disease forms the basis of one classification, Dupré having divided icterus into the acute and the chronic forms. No such division exists clinically; an icterus which begins acutely may in a later stage become subacute or even chronic.

The prognostic form of classification divides icterus into benign jaundice of medium intensity and severe jaundice. Although from a clinical point of view this is an important distinction, it is open to the same objections as the classification based upon the course of the disease. Icterus beginning with symptoms so benign as to justify a good prognosis often assumes a malignant type and becomes fatal.

These classifications are uncertain and unsatisfactory. A classification based upon the etiology of the disease, or an anatomico-pathological one giving the morbid lesions characteristic of each kind of icterus would be valuable, but neither is feasible in the present state of our knowledge of the disease.

Research directed to a discovery of the exact bacteriological condition of the bile passages in the various forms of icterus is met by obstacles which are always serious and often insuperable; and insuperable they will remain until it becomes possible to reproduce the morbid forms in animals. At present bacteriological research is possible only upon the cadaver, and upon passages in direct communication with the intestines, in which, as we knew, the most varied micro-organisms are found. A few important experiments would seem to show that after death the bile ducts may be invaded by intestinal bacteria; Wurtz and Hermann in a number of autopsies performed twenty-four to thirty-six hours after death, found the bacterium coli commune in the liver, spleen, and kidneys. This completely upsets the theory that some forms of icterus may be accounted for by the action of this bacterium. The same reasoning holds good in the case of other micro-organisms to whose influence the jaundice is often attributed because they are found in the bile passages after death. They may yet be found to be a secondary occurrence rather than a cause, the more so that many of the bacteria found under these conditions are normally present in the intestinal tract.

An exact determination of the infective pathogenesis of icterus is not yet possible. It is true that of late experimental research in this direction has greatly increased, and that from the bacteriological study of the lighter forms of icterus we have advanced to that of the more severe forms, such as acute yellow atrophy of the liver; but a

and exact demonstration that in any given case the cause is to
und in any special infective agent is absolutely impossible.
the study of infectious diseases proved that many anatomical
unctional lesions were in direct relation to the secretory prod-
nd transformation products of the morbid agent rather than to
gent itself, thus giving a toxic interpretation to the subject, the
genesis of icterus was also studied from this point of view.
d of seeking for a primary infection of the bile passages, the
d was taken that the primary affection might be in the intes-
tract, the toxins produced being absorbed into the portal circu-
and through the protective function of the hepatic cell elimi-
with the bile, perhaps causing in their exit some anatomical
of the mucous membrane of the bile ducts capable of account-
r the morbid symptoms of the affection. This theory was fur-
upported by the fact that in some forms of icterus the hepatic cell
olved and may oftentimes enter into a condition of hyperactivity,
by an increase in the quantity or quality of the bile (poly-
or pleiochromia). In the graver forms of icterus an early par-
ion in the morbid process by the hepatic cell is evidenced by
ofound atrophy which it undergoes, as we shall have occasion
cribe later.

us the theory of the infective origin of icterus has been in great
re discredited, and the attempt has been made to substitute
the theory of a toxic or toxico-infectious origin of the disease.
nal autointoxication from altered chemism or from extensive
tion of toxins due to intestinal infection might be the cause of
forms of icterus held by some to be of infectious origin.

is view of the pathogenesis of jaundice is not incompatible with
nical aspect of these cases, that is to say, with the fact of their
accompanied by a febrile movement. Nearly all pathologists
eed that fever is due to the action of special toxins of bacterial
and researches have been made into fever of purely toxic ori-
ch as that which occurs in uræmia (Richardière and Thérèse).
ot improbable that the icterus which forms the chief symp-
the disease may be related to exogenous or endogenous intox-
, like the icterus due to poisoning of which we have already
.

anatomical lesions of the bile ducts are such as could be ac-
l for by this hypothesis. In the early part of this article we
of the grave lesions produced in the smallest bile ducts by
norus and sulphuretted hydrogen poisoning, and we are there-
epared to admit the toxic or ptomain origin of this variety of
.

Pathological anatomy is unable to furnish a clear and unchanging classification. In the first place, there has not been a sufficient number of autopsies in cases of primary icterus, especially those of a benign type, of which the patients seldom die, to enable us to distinguish between them. Nor has a study of the anatomical lesions in cases of more or less serious febrile icterus shed much light upon the subject. In acute yellow atrophy of the liver alone have the data obtained post-mortem been of a nature sufficiently clear to differentiate this from other morbid forms.

From what we have already said, the diversity in the course taken by the various forms of icterus and the fact that it is not always strictly in accordance with the initial symptoms, lead to the possible conclusion that the anatomico-pathological lesions may, like the symptoms, be progressive in their nature and without limit in either degree or characters. The classification proposed by Chauffard for angiocholitis, excluding icterus of infective origin, in which he believes that there is always a lesion of the hepatic cell, is based upon the persistence or non-persistence of some obstacle to the flow of bile. He divides angiocholitis into two groups; in the first there is no previous retention of the bile, in the second there is precedent retention, or retention secondary to other processes external to or within the liver. He describes two forms of icterus, which he calls benign infectious and grave icterus.

In our opinion such a classification leads to great confusion. To separate infectious angiocholitis from infectious icterus would imply a boundary line between the two diseases, whereas it is known that the anatomico-pathological basis of infectious icterus is found in catarrh of the smaller bile ducts. Neither can the classification be supposed to concern suppurative angiocholitis, for this properly belongs to suppurative hepatitis.

The classification of icterus is therefore seen to be far from exact. Much remains to be done in the line of demonstrating the pathogenic agent, as well as the anatomical lesion which it produces. Both will be greatly elucidated by bacteriological research, and even more by chemical research into the digestive processes, to determine the composition of the normal and pathogenic substances which may arise in the intestines during digestion or from exogenous infections, and to enlarge our knowledge of their nature and biological action, the manner of their absorption into the portal blood, the functional, nutritional, and histological modifications which they cause in the hepatic cells, the condition in which they are eliminated from the bile, and their action upon the mucosa of the bile ducts. These are multiple and difficult problems awaiting a solution, from which alone

hoped a rational classification of icterus and an explanation of thogenesis of hepatic diseases, since, as we have frequently in the changes in the composition of the bile will probably nd the determining cause of many affections of the liver.

r practical purposes it is absolutely necessary to follow some ication of icterus, and the classification which we give, while tisfactory from the present standpoint of pathology, is based linical facts. To begin with, we do not believe it practicable ove the catarrhal icterus of the older writers from its place in thology of the bile passages, as some modern pathologists have us do, for it is very usual to find it in practice in relation ial morbid causes, and it is therefore deserving of the atten- the practitioner.

ndice of a benign nature following gastrointestinal catarrh, mpanied by fever or symptoms which would indicate infection intoxication, is of common occurrence and deserves special n. Neither the complete absence nor the rarity of anatomico- gical data (which do exist, as we shall see presently) can the omission of this form of jaundice, since clinical considera- e above those of the autopsy; in other words, we cannot ex- disease simply because a definite anatomical base for it is n.

valuable fact distinguishes catarrhal icterus, namely, that it ys accompanied by intestinal acholia, due to the fact that the l condition of its pathogenesis is the swelling of the mucosa accumulation of mucus in the large bile ducts, while in other f benign icterus the intestinal contents are not discolored, but ontrary often loaded with bile.

tiological classification being impossible, since we are unable hether the infection or intoxication, autochthonous or second- astric infection, is the chief cause of the icterus, we shall base sification upon the nature of the disease, although even this atisfactory. Benignity and malignity in icterus are to a cer- ent a matter of contingency, as we cannot predict the succes- os in the morbid process. "In icterus," observed Trousseau s fine clinical intuition, "we have the same condition as in effusion; we know the beginning, but we do not know the

presence or absence of fever forms a criterion which is sus- y clinical experience. The forms of icterus, considered by be of an infectious nature, seem to be accompanied by fever. ht, therefore, call one form of icterus the febrile, and this ature would be preferable to others because based upon the

most marked symptomatic feature of the nosological group thus distinguished (Paletta[67]).

But the fever is not a constant sign of infection, and the gravity of the latter cannot be inferred from the degree of the elevation of the temperature. Yet we must bear in mind that there is a form of icterus which is rapidly fatal, and that it is considered to be of infectious origin and to be accompanied by hyperpyrexia. On the other hand, in catarrhal icterus there may occur a febrile movement which is not directly due to the jaundice but to the acute gastrointestinal catarrh. In spite of the fact that the fever may sometimes be absent, we believe the term febrile icterus to be preferable to infectious icterus in a large number of cases, for the latter term may be altogether erroneous. We do not wish to conceal the fact that catarrhal icterus itself has been considered by some to be of an infectious nature, but no clear demonstration of this has been given. As in the case of a gastrointestinal catarrh which may be thought to be of infectious origin but is not described as an intestinal infection when the cause is unknown, so a catarrhal icterus, while it is not denied that it may be of bacterial origin, is not included in the list of biliary infections.

The division of icterus into primary forms and those secondary to other intra- or extrahepatic lesions or to general infectious diseases is one accepted by all. We classify icterus according to clinical types as follows:

- Icterus
  - Primary
    1. Catarrhal (acute or chronic).
    2. Febrile.
    3. Grave (acute yellow atrophy).
    4. Emotional.
    5. Of the newly born.
  - Secondary
    1. To infections.
    2. To hepatic disease.
    3. To intrinsic or extrinsic stenosis of the bile ducts.

This classification, we repeat, is purely clinical and cannot be accepted from a pathological or pathogenic standpoint. An etiological and progressive classification belongs to the future.

Catarrhal Jaundice.

Etiology.

We have already stated our reasons for devoting a special section to catarrhal jaundice. The disease, from our standpoint, consists in a catarrhal inflammation of the mucous membrane of the larger bile ducts, propagated by extension from a catarrhal process in the duodenum. The embryological and anatomical relations of the parts

an explanation of the ease with which a catarrhal process in the nal mucosa will extend to the ductus choledochus and the larger cts. The latter are derived embryologically from the outward of the intestinal walls, and anatomically from the direct con- on of the latter.

etiological conditions therefore are in intimate relation to the ico-pathological condition of the intestines, more especially odenal portion where the ductus choledochus empties into the a of Vater.

form of gastrointestinal catarrh may result in inflammation mucosa of the ductus choledochus and the large bile ducts. f this biliary catarrh is of no clinical importance and might observed were it not that the swelling of the mucous mem- nd the increased amount of mucus form an obstacle to the n of the bile, which unless overcome by the secretory pres- the bile causes stasis, absorption of the bile by the lym- and the veins, and its appearance in the blood. Catarrhal is therefore an icterus due to stasis.

fundamental causes of catarrhal icterus are the same as those ointestinal catarrh. A too abundant and ill-regulated diet, stion of irritating substances and strong alcoholic drinks, y be considered causes of biliary catarrh which is never pri- its origin, but always secondary to some catarrhal lesion of stive tract.

most common clinical symptoms found are disturbances of nach and intestines, nausea, vomiting, eructations of gas, gastralgia, diarrhœa, constipation, etc., to which is super- yellowish tinge of the skin, the date of whose first appear- patient rarely can tell, and which rapidly increases.

ttempt has been made to connect the appearance of the jaun- a cold, or with some emotional disturbance. But while mentioned cause may by a further disturbance of the ab- circulation occasion a recrudescence of the catarrhal symp- ich of course facilitates its extension to the biliary passages, t admit emotional jaundice to a place with catarrhal jaun- gards either its mode of origin, its clinical symptoms, or , and we shall refer to it in its own place later.

ne cases the disease is attributed to disturbances in the cir- and in this connection we would observe that it is not un- find icterus in cardiac patients, in the hyposystolic period ffection, in pulmonary emphysema, chronic bronchial ca- d other affections of the respiratory apparatus when they ced disturbances of the cardiac function. In our descrip-

tion of hyperæmia of the liver we called attention to a special pathogenesis of the slight jaundice which often accompanies it and, as a rule, we have to deal with some one of these forms. We must, however, bear in mind that there may be a true catarrh of the bile passages caused by stasis catarrh of the digestive tract, and increased by the defective condition of the circulation in the bile ducts.

Some authorities (Neuschler, Fleischmann) have described a catarrhal icterus occurring at the menstrual period, a fact which it is difficult to explain. According to Senator there is a vicarious hyperæmia of the liver.

As to the influence of sex, age, habits, climate, etc., the same factors are present as in the case of gastroduodenal catarrh, which we hold to be the cause of catarrhal icterus. In our own experience this form of catarrh occurs more frequently in adult males than in women or children.

Morbid Anatomy.

On account of the uniformly benign nature of catarrhal jaundice, anatomical data are difficult to obtain. As in all catarrhs of the mucosa, after death the lesions, consisting chiefly of swelling and hyperæmia of the mucous membrane, either disappear or become greatly reduced. Although not always possible of demonstration, it is certainly permissible to take it for granted that this congestion and turgescence are found.

The increase in the mucous secretion is noticeable even in the cadaver. A layer of more or less thickened mucus mixed with desquamated epithelial cells is found upon the internal surface of the mucosa; in some cases plugs of mucus are found in the bile ducts occluding the lumen and thus interfering with the flow of bile. These are easy of demonstration by pressure upon the gall-bladder, which is usually full; the bile either flows with difficulty or not at all into the intestines, and by increasing the pressure a compact greenish-white plug of mucus will be squeezed out from the ductus choledochus, after which the bile flows freely. The formation of the plug is, however, not an essential condition to the narrowing of the lumen of the vessels, since the simple swelling of the mucous membrane, which usually disappears after death, may cause complete or partial stenosis. It is to be noted that in the region where the stenosis occurred during life the biliary duct is pale in color, while above it, from retention of bile, the color is yellow.

The intestinal portion of the ductus choledochus is that which exhibits the most marked lesions, from the fact that the morbid process is most easily transmitted to this part. Above the point of

sis we find retention of the bile and dilatation of the bile vessels; stenosis, as is usually the case, involves the ductus choledoch, the retention is complete, and the liver is enlarged and of an greenish-yellow color. If the stasis last a certain length of serious lesions of the parenchyma may be produced, such as we already discussed in connection with hypertrophic biliary cirsis.

companying catarrh of the ductus choledochus we may find of catarrh of the gall-bladder and of the cystic duct.

Symptoms and Course.

e relation of catarrh of the ductus choledochus to gastroduocatarrh enables us to understand why catarrhal icterus is preby a period in which there are symptoms of gastric or intesisturbance. Anorexia, pyrosis, nausea, vomiting, diarrhœa or pation or the two alternately, etc., are the morbid signs which first to appear in a catarrhal icterus. During this condition tient begins to assume a jaundiced appearance, which is usurst perceived by his friends; the gastrointestinal symptoms e, and intestinal acholia occurs with all the symptoms of stasis .

ring already (see p. 477) described the symptoms caused by trance of bile into the circulation, and its deficiency in the al tract, we shall not repeat the description. We will merely hat this form of jaundice follows the type of absorption e from defective excretion of bile, so that a special coloration tissues, the presence of normal biliary pigments in the blood ine, a marked slowing of the heart's action with relative g of the pressure, cutaneous pruritus of varying intensity, but often constitutes the worst subjective symptom, xanthopsia, the most frequent manifestations of the cholæmia, and, on the and, obstinate constipation, the characteristic discoloration of s, the interference with the digestion of fats, and the disturbthe digestion and absorption of nitrogenous substances and rbons are the manifestations of the intestinal acholia.

e symptoms of autointoxication never appear in this disease, the hepatic cell remains unaffected. This may be demonby the test for alimentary glycosuria and by the ratio of the minated. Neither are there symptoms of nephritis; these ny the other forms of icterus, of which we shall speak later. bjective examination of the liver is usually of importance. id before, after biliary stasis it is enlarged and the inferior

margin of hepatic dulness therefore extends two or three fingers' breadth beyond the costal arch, while by palpation the inferior border of the liver can be felt through the abdominal walls. Palpation is usually painful. There may be spontaneous pain due to distention of the peritoneal covering of the hepatic gland. In other cases the patients are conscious only of a sense of pressure or of tension in the right hypochondrium, and in the epigastrium. When the occlusion is in the ductus choledochus, a large amount of bile may accumulate in the gall-bladder, which becomes stretched and swollen and may be felt by palpation or outlined by percussion, especially when the abdominal walls are not thick or tense.

If the jaundice be prolonged, the general condition of the patient deteriorates; in consequence of the disturbed chemism and absorption in the intestines, nutrition is impaired, and the patient becomes thin, weak, and depressed. In some cases there is a special condition of physical and moral asthenia (jaundiced disposition), characterized by general apathy, irritability, and great moral depression, so that the patients become irascible, intolerant, and even hypochondriacal. The term bilious temperament has arisen from this special character of the nervous condition, and the word bilious is often used as a synonym for choleric or angry.

When the gastrointestinal tract is not especially affected, there is no fever, but, on the contrary, as we have several times had occasion to observe, the temperature may be lowered even to the extent of one degree. This is probably due to the action of the biliary acids and pigments upon the thermogenic centre. If fever should occur with catarrhal icterus, it may be caused by intestinal complications or by secondary infection of the biliary passages.

The disease usually runs its course in a few weeks. The urine begins to clear, the fæces assume their normal coloring, there is less flatulence, the pulse becomes more frequent, and arterial pressure rises. At the same time the general condition improves, and a healthy coloring begins to appear beneath the yellow tint of the skin. The skin, however, continues to be slightly jaundiced, even after biliary pigment has altogether disappeared from the urine, and this is because of the great affinity between biliary pigment and the protoplasm of the Malpighian cells, which is so marked that it would seem the skin is unable to free itself from the stain without desquamation of the epidermal layer. Hence, even when the patients feel perfectly well, the jaundice persists for a while, but gradually disappears.

In some cases the course of the disease is longer, sometimes lasting several months. A rare clinical type passes into a chronic condition of catarrhal icterus with permanent occlusion of the bile ducts,

h finally results in the most serious anatomico-pathological al-
ions in the cells and in the periangiocholitic connective tissue
Biliary Cirrhosis).

Prognosis.

rom the course of the disease, it is easy to see that the termina-
of catarrhal icterus is usually favorable, the occurrence of a
ic condition being altogether exceptional. The prognosis there-
s, with this one exception, a good one.

Diagnosis.

hen icterus occurs after symptoms of gastric catarrh, the diag-
of catarrhal jaundice is usually easy. In some cases, however,
is possible. Icterus appearing after gastrointestinal disturb-
may not be due to a disturbance of the digestive tract, but if
ic disease accompany it, may be the manifestation of a serious
bance of the liver. Moreover, if the physician question too
tently as to a previous history of gastrointestinal troubles, he
y a species of suggestion cause the patient also to lay too
emphasis upon any such signs, and thus lead to a mistaken
sis. Hepatic disorders accompanied by jaundice, more espe-
hypertrophic biliary cirrhosis or hepatic carcinoma, may be
verlooked. We have already seen how insidiously both of
diseases may begin, the first because of the long period in
the jaundice remits and reappears, sometimes in relation to
ure of the food taken, and the second because it follows a la-
urse which may often begin with acute symptoms of icterus.
e cases the errors are the more liable to occur because there
symptoms which might aid in a differential diagnosis, and the
lone of the malady will decide the matter.

rivate practice, especially when one is called in consultation
outset of the disease when no data exist in regard to its
there is the danger of diagnosing a simple catarrhal jaundice
s in which the classical phenomena of biliary cirrhosis or of
of the liver subsequently develop. For this reason it is well
tremely reserved in the prognosis in view of the diseases of
the jaundice may be the preliminary symptom. The state of
es is an important guide to the diagnosis. In hypertrophic
cirrhosis they are diarrhœal, bovine, and colored with bile,
n catarrhal icterus they are cretaceous and grayish-white in
When icterus appears in old age, and instead of diminishing
s in intensity after the fourth or fifth week, we should sus-
presence of a malignant neoplasm of the liver.

Catarrhal jaundice sometimes simulates biliary calculus. When the attacks of colic seem to be dyspeptic rather than hepatic in their nature, and not very painful, jaundice rapidly occurring in connection with them, we may infer that they are due to gastroduodenal catarrh. The previous history is of great assistance in the diagnosis, as there will usually have been painful or other phenomena indicating lithiasis; an examination of the fæces, too, is important, as it is often possible to find calculi which have been eliminated. In the case of calculus, the jaundice usually appears rapidly, does not attain very great intensity, and disappears quickly with total remissions of the pain in the epigastrium and hypochondrium.

It is evident from what has been said that a diagnosis of catarrhal jaundice should not be made without consideration of the history of the case and a careful physical examination, nor until all other hepatic affections which are ushered in with jaundice have been excluded.

Treatment.

The chief therapeutic indication is regulation of the diet; this will serve to cure the gastrointestinal catarrh, which is the immediate cause of the jaundice, and at the same time to place the liver in the best physiological nutritional condition. By reason of the intestinal acholia, not only are the digestion and absorption of fats interfered with, but also those of the albuminoids and the carbohydrates; therefore milk, which from the ease with which it is assimilated causes the least chemical work, forms the best food for these patients. Some practitioners, having in mind only the indigestibility of fats in acholia, do not believe in a diet of milk because of the cream which it contains, and prefer a rigid regimen of lean meat, white bread, light soups, vegetables, cooked fruit, etc., overlooking the fact that the pancreatic juice possesses a special ferment capable of digesting fats (steapsin). We believe this view to be unreasonable. To be entirely preoccupied with the defective digestion of fats without considering the increased difficulty in the digestion of albuminoids and carbohydrates, to be impressed by the amount of butter contained in milk and not to realize that it is almost impossible to exclude all fat from the food, to prefer to the easy digestion of fat the more difficult digestion of other alimentary substances, ignoring the fact that the small amount of fat ingested, if not acted upon by the pancreatic juice and absorbed, is expelled in the fæces without causing any special fermentation, means a very one-sided view of physiological pathology and a disinclination to adopt a rational therapy.

A rigid milk diet implies the exclusion of all irritating beverages,

as wine, beer, liquors, etc. The milk may be rendered more tible or more palatable by the addition of alkalies, aromatics, en of coffee or tea. A second important indication is the regu- ı of the bowels, which, from the absence of the normal stimulus ɜ bile, are apt to become torpid. For this purpose the use of gogue cathartics is advisable. Rhubarb and senna have ac- d a good reputation in practice because of their myokinetic r and their action in increasing the flow of bile by the propaga- ɔf the peristaltic movements from the intestines to the ductus lochus. They may be administered daily without fear of irri- ; the intestinal mucous membrane, and to a certain extent they ɔme the swelling of the mucosa and the accumulation of mucus ɜ ductus choledochus, which are the immediate causes of the y stasis.

e should also prescribe alkaline waters, which, as we have al- seen, exert a beneficial influence upon the hepatic function. of Carlsbad, Vichy, Castellamare, and others are useful, and ɔe taken at intervals during the course of the day.

me authorities have laid stress upon the necessity for intestinal ɛction, because of the absence of the antifermentative action of le, and have especially recommended salol, naphthol, the sali- s, etc. The best intestinal disinfection, however, consists, as ve repeatedly said, in an exclusive milk diet.

rhardt has made the attempt to provoke a flow of bile into the nes by overcoming the obstacle by mechanical means, and in ɛases in which the gall-bladder was distended and palpable he ded by appropriate manœuvres in compressing it and at once ing permeability of the ductus choledochus. Following in his ps, several other scientists have, especially in cases of pro- catarrhal jaundice or chronic jaundice, applied massage or ctric current to the dilated gall-bladder in the hope of stimu- its muscle fibres and thus increasing the pressure of the bile ercoming the obstacle to its circulation. This idea of mechan- re has led to some absurdities, such as the advice to patients ce, jump, ride, etc. Even apart from this last suggestion, cannot be recommended in earnest, there is little to be expected ıechanical treatment. The manœuvre proposed by Gerhardt is en feasible, and when possible is painful; it is, moreover, a ɔmatic form of treatment, rather than one addressed to the or to the cause of the disease; even if the obstacle in the choledochus is overcome, as the catarrhal conditions remain ted, it may easily return. Neither does massage nor faradiza- ve good results, at least according to our experience. This can

be readily understood when we consider how few unstriped muscular fibres there are in the walls of the gall-bladder and that these have probably undergone alteration from the stasis of bile.

The treatment proposed by Krull[65] in 1877, consisting in the daily use of cold-water clysters, is very useful in catarrhal as well as in other forms of jaundice, as we shall see later. According to this writer, this treatment causes rapid improvement of the gastrointestinal symptoms, and after a few injections the bile reappears in the fæces, the ductus choledochus becoming quite free from obstruction in from six to eight days. According to Chauffard, this method is useful in cases of acute icterus, but absolutely useless in the chronic forms. Our own experience is in favor of Krull's method, but we should never advise the injection of 2 litres of cold water, as such a large amount of fluid would tend further to weaken the atonic intestinal walls. Instead of this, we recommend two injections daily (morning and evening) of half a litre (one pint) of cold water (10° to 20° C.—50° to 68° F.), the fluid to be retained in the rectum until the desire to expel it becomes irresistible. The mechanism of this treatment consists in the provocation of peristaltic contractions which are carried from the intestines into the large bile ducts, and facilitate the flow of bile. Cold, rather than a large amount of fluid, is the essential factor to this result.

This rational measure is of course to be accompanied by treatment addressed to the symptoms; those deserving the most attention, because of the discomfort they cause to the patient, are the cutaneous pruritus and the bitter taste in the mouth. Medicine gives but slight relief of either. For the itching we should prescribe vapor baths, the partial baths of acetic acid recommended by Murchison (acetic acid, 250 gm.; water, 13 to 14 litres), chloroform lotions, anointing with unguents and oils, and the alkaline bromides given internally; for the bitter taste, the mouth should be rinsed with borate of sodium rendered aromatic by peppermint water. Bitters and aromatics are to be given to stimulate the appetite, and each symptom as it arises is to be appropriately treated.

Benign Febrile Jaundice.

This variety includes many idiopathic forms of jaundice, which occur independently of previous gastrointestinal lesions. They differ greatly in their course and general aspect, but possess two common etiological properties which, according to Chauffard, are the cause of their benignity, and that is the preservation of the integrity of the hepatic cell and unimpaired renal functions. The

ervation of these functions explains the rarity of the autoin-
ation which so often is the cause of death in hepatic dis-
. This condition cannot, however, be looked upon as absolute
invariable; there are progressive gradations instead of a precise
ation between the normal and the pathological functioning of
epatic cells and the kidneys. Therefore a line of demarcation
ot be drawn between benign and grave or fatal icterus, and, as
ve already stated, in practice we constantly see cases of benign
ice which later become of a grave type. From the little that we
in regard to their pathogenesis, it would seem that the cause in
veral types is one and the same, and consists of an infective or
probably of a toxic agent.
e presence of fever forms a diagnostic sign between these be-
orms of jaundice and true catarrhal icterus, and so does their
of onset, which usually resembles that of an infective disease.

Etiology and Pathogenesis.

was long since noticed that cases of icterus occurred more fre-
y in the autumn and the spring than at any other season,
which originated the name of autumn and spring jaundice.
s especially noticeable in large cities, and in Paris there is so
jaundice at certain seasons as to have given rise to the saying,
des mois on il pleut de la bile à Paris."
degrees, as the attention of practitioners was drawn to the
nce of the disease at certain seasons, it was noticed that there
rue epidemics of it. Diamantopoulos, Ganchey, Fröhlich,
e, etc., noticed small epidemics at different times in various
es in which the cases were sometimes all benign, while in
some cases were grave. True epidemics have been noted in
s' barracks (Laveran, Arnoult, and Coyne), the conscripts
sually most liable to the attack. According to Hennie and
rd, no immunity is conferred by the disease, but rather a
predisposition to it results from the attack.
ould seem (and many persons attribute great importance to
t) that alcoholism may cause febrile icterus. Its mode of
s variously interpreted, and is probably complex; direct and
action of the alcohol upon the cells, intestinal catarrh with
ation, and the production of certain toxins capable of acting
ir greatest energy in the liver, all probably have a share in
ation of the disease. Overindulgence in irritating, unwhole-
odstuffs, irregular or too abundant meals, the use of tainted
c., may in some cases cause febrile icterus.

Of importance to the right understanding of the pathogenesis and the occurrence of these forms of jaundice is the fact that they may follow inhalations of sewer gas.

Landouzy, Shirl, Ducamp, and Barrelli have reported important cases of the disease, evidently directly due to the absorption of putrid gases, occurring in the workmen whose duty it is to clean out the sewers. This form of the disease has also been noticed in persons following certain special trades, as, for instance, in butchers and tanners, when it may possibly have been due to toxic emanations from putrefying meats or skins (Fiedler, Vierordt, Chauffard).

The morbid agent, then, may consist of toxic substances developed during the decomposition of organic matter. The disease is found in adolescence and in adult life, rarely in childhood or old age. In pregnant women, as a rule, it assumes a grave type.

From a consideration of the facts which we have briefly mentioned, pathologists have been induced to study the bacteriological condition of the biliary passages, thinking that the pathogenesis of these morbid processes might be attributed to some special infection of these passages. It is not our intention to enumerate the various bacteriological data found by the different experimenters, because they lead to no practical result. We will mention the chief ones only, as from them can be seen how many and various are the micro-organisms which different investigators have found and to which the disease has been attributed. According to Jaeger, the morbid agent of some cases of febrile icterus which occurred epidemically in the barracks at Ulm was a proteus (capable of producing the disease in birds), and Bordoni-Uffreduzzi, even previously to Jaeger, described a similar proteus. Freund found a special diplococcus, Neelsen a characteristic bacillus, not pathogenic in animals, however; Goirde found a bacillus in the urine which was very similar to the bacillus coli communis. These positive results, which however proved nothing in regard to the cause of the disease, have been discredited and contradicted by reason of the result of important researches by Bastianelli Bignami, and Ducamp obtained during life; these, in view of what has been said about the possibility of post-mortem infection of the liver, are of far more value than the results of researches upon the cadaver. It is evident that not the slightest support to or relative demonstration of the theory of a special infective agent in febrile icterus has been given by these bacteriological researches; it would even seem that they disprove it altogether.

Kelsch holds that the following conclusions are tenable: 1. Idiopathic febrile icterus (including catarrhal icterus), whether sporadic or epidemic, is an infective disease; 2. The pathogenic agent is de-

d outside of the body; 3. It is developed in marshes, sewers, ch in vegetable and animal substances undergoing decomposi- nd in polluted waters.

hough there is no positive proof as yet of the theory of an in- origin of this affection, yet its course greatly resembles that ctious diseases. There is, as we shall see presently, a stage pre- ry to the jaundice characterized by general phenomena. We , however, from this fact alone argue that there is a specific on, the more so that a toxic or infective-toxic origin would to explain it. Studies upon the action of toxins in animals hown that there is always a short prodromal stage which re- s that of infectious diseases. An intestinal infection, exoge- r endogenous, with the secondary production of toxins which absorbed and reaching the liver would determine the occur- f jaundice, would satisfactorily explain this circumstance.

nical facts give more support to the theory of a toxic origin of than they do to that of an infective or infective-toxic patho- . The icterus due to inhalations already referred to, and the presented by artificial jaundice produced by inhalations of retted hydrogen or by injections of toluylenediamine, etc., confirm this view of the case. What are the toxic agents, and way they are absorbed and act upon the liver, are yet unan- questions.

ough in the actual state of our knowledge the toxic theory the most tenable, yet it is not improbable that as bacteri- rogresses we may have a satisfactory demonstration of the in- genesis of the disease.

Pathological Anatomy.

anatomico-pathological lesions, which are the immediate the morbid process, are enshrouded in as much obscurity tiology and pathogenesis. Although it has attracted the at- of pathologists, there have been but few occasions for post- examinations, either because recovery is the usual result, or the jaundice has been looked upon as an epiphenomenon of nown infectious disease, especially typhoid fever, to which l result was attributed. In other cases the jaundice, which s a benign type, assumed later the aspect of grave icterus, lesions sought for were those of the latter form of disease. sely this class of cases the general anatomico-pathological ns reported have been very similar to those of the infectious Browodowski and Dunin, Heber, Nauwerk, Mazzotti,

Bastianelli and Bignami, Tessari and Battistini found grave lesions in the liver and kidneys, consisting chiefly in fatty degeneration and necrobiosis of the glandular cells, often accompanied by infiltration of embryonal cells; there were at the same time grave lesions of the other organs, congestion of the lungs and brain, marked splenic tumor, ecchymosis of the epiploon, mesentery, and adipose capsule of the kidneys, lesions which, as we shall see, especially characterize the grave forms of icterus. We may therefore conclude that there is no pathological anatomy peculiar to the benign forms of icterus excepting catarrhal jaundice.

The pathological anatomy of benign febrile icterus is, by physiological induction rather than by actual demonstration, generally held to be the same as that of catarrhal radicular angiocholitis. Whether the origin be infective, toxic, or infective-toxic, it is altogether probable that the primary lesion occurs in the radicles of the bile vessels, inducing catarrhal lesions of the mucosa with tumefaction capable of causing a certain amount of retention of the bile, by reason of the lowered excretory pressure in this portion of the biliary system (Barth and Besnier) and its subsequent absorption into the lymphatic vessels and venous radicles. This would be a true stasis jaundice, but would be more benign than the usual form because the morbid agent, toxin or bacterium, would produce a functional excitement of the hepatic cell, with the secretion of a greater amount of bile, or of bile which is thickened and laden with pigment (polycholia or pleiochromia); and increased production of bile being superadded to the impeded flow, the jaundice would be easily produced and yet there would be no conditions favorable to intestinal acholia.

Another gradation in the intensity of the jaundice exists. Following the glandular hyperactivity, nutritional and anatomical lesions occur (due to either the intensity or the duration of the morbid cause) characterized chiefly by fatty degeneration, or in more severe forms by primary necrosis. At the same time the pathogenic agent, because of the changes which it has itself brought about in the protective function of the liver by the anatomical lesions caused, cannot be eliminated or transformed, but, accumulating in the blood, reaches the kidneys, the protective function of which is closely united to that of the liver. The morbid agent induces grave anatomical lesions in them as well, and these are in very large measure the cause of the malignancy of these forms of icterus. The above is the anatomico-pathological process as explained by induction. Actual knowledge exists only in regard to the most severe form of all, acute yellow atrophy.

Similar reasonings rather than actual indisputable facts sustain

gument for radicular angiocholitis, and these are chiefly based the primary lesions of the bile radicles found by Stadelmann in terus produced by poisoning by phosphorus, toluylenedia- etc., and referred to under the pathology of icterus.

Symptoms.

e symptoms in benign febrile icterus vary greatly in intensity than in kind. We have stated several times that in practice not see a special morbid type, but rather a variety of forms ing from the most benign to the fatal.

e prodromal stage, very similar to that of the infectious diseases, ewhat short, and is characterized by gastrointestinal disturb- (loss of appetite, a bitter taste in the mouth, nausea, dyspep- .), and what is still more important, by general phenomena, s headache, fatigue, malaise, muscular and articular pains, ight elevation of temperature. In rare cases this stage is ab- nd then the icterus is the first thing to occur. In other cases, other hand, the prodromal symptoms may be very serious, the nay reach 40° C. (104° F.), and there may be epistaxis, gastric testinal hemorrhages, and great weakness. The severity of ection may in a measure be predicted from this pre-icteric but in a few rare cases there may be slight initial symptoms d by somewhat severe jaundice, or grave prodromal symptoms her in a light form of icterus.

jaundice soon appears, either gradually or rapidly in the forms. A careful examination of the urine will show that the initial period of icterus there will be a true urobilinuria, the transformation of the bilirubin into urobilin by the renal ium, when but a small amount of the pigment is found in the The same condition may be observed in the last stage of the , when the amount of pigment set free in the blood serum lly decreases.

eral authorities, considering catarrhal icterus to be one form e idiopathic types of icterus, state that they are usually accom- by intestinal acholia. We, however, who for reasons previ- iven, have considered catarrhal icterus by itself, hold that, as when there is no marked stenosis of the large bile ducts ropagation of the morbid process, these forms of icterus are anied by a greater secretion of bile, so that the fæces become eal and so loaded with bile as to respond clearly to Gmelin's

ether with the jaundice appear all the symptoms dependent

upon it, which we have already described in detail. An objective examination of the patient shows, in addition to the symptoms of cholæmia, an hepatic swelling and a certain amount of pain upon palpation; the liver, as a rule, passes from two to three fingers' breadth beyond the costal arch; it is smooth, and the antero-inferior border is unaltered. Percussion will sometimes demonstrate that the area of dulness is increased above as well as below, but to a lesser extent. The spleen is also swollen and possesses all the characteristics of an acute enlargement, this being due, according to those who sustain the theory of infection, to an infective agent.

The fever, which is slight in the prodromal stage, may entirely disappear with the occurrence of the jaundice, but in more severe cases there is either an elevation of temperature or a persistence of a high initial temperature. The fever is of no special type, but is usually continuous-remittent. On the other hand, it sometimes assumes the form of intermittent hepatic fever, of which Semmola recently had an interesting case.

The patient was a man of 30 years, of good family, who after alcoholic excesses was the subject of general disturbances and a slight evening rise of temperature. After two days of this, jaundice rapidly appeared, together with a marked chill followed by high fever, which remitted and was followed by profuse sweating. The jaundice became more severe, and the fever preserved the above-described characteristics for a week and became of an intermittent quotidian type. Injections of quinine produced no effect. Later the fever became continuous-remittent, and fell by lysis with the remission of all the morbid symptoms.

The general condition of the patient depends upon the gravity of the affection; it is usually good, but may sometimes assume a grave aspect when the symptoms of the disease are severe. In a few cases, more especially in those which become grave in type, there are symptoms of dissolution of the blood, accompanied by hemorrhages into the mucous membrane and skin, symptoms of a fatal ending. The condition of the kidneys is of great importance in these forms of icterus. As a rule they remain normal, and this is a characteristic of the benignity of the lesion. Occasionally albuminuria or even slight hæmaturia may be present, in which case the prognosis should be reserved, the chief compensation of cholæmia being found in an unimpaired renal function.

Chauffard was the first to investigate the amount of urine and of urea in benign icterus. In the period of invasion, and when the disease is established, both are considerably reduced. It would seem that the purifying powers of the kidneys are insufficient for the work required, and that the nitrogenous products of organic combustion

ven water itself find their elimination interfered with and there- ıccumulate in the blood. But when the disease draws to a when the infective or intoxicating agents disappear, there is ıble increase of urine and of urea, which may reach to great ıts, while at the same time there is a marked elimination of bile nts. We thus have produced what Chauffard calls a *polyuric trogenous crisis*, which greatly resembles similar occurrences in urse of the infectious diseases. The general phenomena sub- ith rapidity, there being usually defervescence by crisis, but atomical lesions of catarrh of the biliary passages disappear gradually.

ger and Surmont have made a special study of the urinary during these crises, and have ascertained that, while they are l or diminished at the height of the disease, during these crises ıcrease to a great extent, a fact not attributable to the increased nt, because they persist even when the urine becomes decol- . There would seem to be a marked elimination of toxins ac- ıted in the blood during the height of the disease.

have ourselves had the opportunity of witnessing these poly- ıd nitrogenous crises of Chauffard in benign icterus, and have the marked influence which they exercised upon its course; in perience they are by no means a constant occurrence. In one of cases these crises never once occurred; the morbid pheno- gradually decreased, and the urine gradually increased in t and in the elimination of bile pigments.

ıe observers have noted in these forms of icterus symptoms of iency of the hepatic cell, especially alimentary glycosuria. casionally continues even after the jaundice has disappeared, ves as a sign to the physician of the persistent functional le- the cell. Girode[69] reports such a case in which a relapse of ease rapidly produced death.

Course.

course is as varied as the result. There are several types of ease.

lly benign febrile icterus, accompanied by mild general symp- ıd a slight febrile movement, ends in complete recovery after e of from three to four weeks. On the other hand, there may ıdual increase in severity of the symptoms until they become f a grave icterus, with high fever and severe general symp- nd characterized by hepatic and renal insufficiency. Even ırms may be recovered from, and as a rule the remission of ms is accompanied by the above-described crisis.

In other cases there will be all the symptoms of prolonged icterus, similar to those of catarrhal jaundice. The disease may last several months, and may finally become chronic. The jaundice then persists; there is enlargement of the liver, and remissions and recrudescences of the icterus may be noticed, being accompanied by elevations of temperature; they resemble biliary hypertrophic cirrhosis. The anatomical lesions may be very similar to those of the latter disease. It will be remembered that, when considering its etiology, we expressed the opinion that biliary cirrhosis might be due to permanent occlusion of the bile ducts.

The diagnosis in these cases is extremely difficult; careful note must be made of the previous history, which, with a progressive diminution of the area of hepatic dulness during the course of the disease indicating biliary cirrhosis, may throw a little light upon the subject.

Relapsing febrile icterus, sometimes called Weil's disease," varies in its course from the usual type, and is by some considered to be a distinct affection. Since Weil first called attention to this form of febrile icterus, many others have claimed priority in its description, Mathieu in particular. But it has been observed that, even if Mathieu and previously to Mathieu, Bouchard, Lanceraux, Arnould, Coyne, and others, had described a few cases of febrile icterus followed by a relapse, the credit of having studied its clinical characteristics from a different standpoint and of having called the attention of pathologists to it, belongs to Weil, and since that time an ever-increasing literature upon the subject has emphasized the importance of this form of febrile icterus.

In our opinion, however, it does not deserve to rank as a distinct disease, but should rather be regarded as a special form of the usual affection, since it is highly probable that the etiology is the same as in the other varieties of febrile icterus. It might easily be due to a difference in the virulence of the virus or in the amount of toxin produced, or even to the varying reactions of the organism. The anatomical lesions are indeed so similar as to be almost indistinguishable from those of febrile icterus of a certain degree of severity. Bacteriological researches have not given any results which would justify us in considering it a distinct disease, and although bacteriology is constantly reaching out into wider fields, it has not yet discovered the pathogenic agent.

Certain special micro-organisms have been met with, such as the bacterium coli commune, of which we have already spoken; the typhoid bacillus has also been found, which has led some authorities to consider Weil's disease as a "hepato-typhoid." This view, held by

Vierordt, Longuet, and many others, has been entirely dis-
d by the researches of Dupré, who proved that in cases of
l in which there was infection of the bile passages this special
jaundice never occurred.
ording to Karlinski, there is a true recurrent typhoid in which
ction acts especially upon the liver; in some cases which he
in Herzogovina he found the Obermeier spirillus in the
These differing results of researches have led many (Maz-
auwerk, Fraenkel) to believe that Weil's disease had a vary-
logy. But as we have already stated, it seems to us that the
is that of the other forms of febrile icterus, from which it
differs in its course. It is characterized by the severity of the
ms and by the relapses. After a prodromal stage which is
by grave general phenomena and accompanied by high fever
e outset, we have the rapid appearance of the icterus, which
intense and be accompanied by diarrhœal and bile-laden
he liver and the spleen are enlarged, the urine is notably re-
nd usually contains albumin and casts. These symptoms
to be alarming for from eight to ten days, and then with a
crisis rapidly subside; there is defervescence by crisis, the
condition improves, and the icterus gradually disappears.
ng to Mathieu the splenic tumor persists, and may be taken
n that the attack will return. After about a week of apparent
cence the general phenomena reappear, more especially the
hich may be ushered in by severe chills, while the jaundice
comes marked. The relapse is usually of shorter duration
ess severity than the first attack. A urinary crisis generally
omplete remission of all the symptoms after from six to eight
t in rare cases the return to a normal condition is much
nd defervescence is by lysis.
g and tedious convalescence has occasionally been noticed.

Prognosis.

evident that the prognosis will vary according to the varying
s and course of the disease, and cannot be given with exact-
pt in special cases. The condition of the renal secretion
he best criterion of the severity of the attack. The most
symptoms are a bad general condition, phenomena of auto-
ion, and symptoms of hæmatolysis.

Treatment.

There remains little to be added to what we have already said about the treatment of catarrhal icterus. The same therapeutic indications will guide the physician in these cases. A strictly milk diet and symptomatic treatment are always to be adopted. Intestinal disinfection is of the utmost importance, and to this end alkaline hyposulphites should be given with the milk diet, insoluble antiseptics and copious injections not being advisable.

The injections of cold water recommended by Krull are of great benefit, and Chauffard has shown that they have an important influence in the causation of the polyuric and nitrogenous crises with the occurrence of which all the symptoms of the disease disappear.

The patient should observe all precautions during convalescence to avoid a possible relapse, and this will chiefly be accomplished by a careful and appropriate diet.

Icterus Gravis.

(Acute Yellow Atrophy of the Liver.)

Although benign febrile icterus is not characterized by any anatomical lesion of the hepatic cell and of the kidneys, the opposite is the case in the grave forms of icterus, for in these there are marked lesions of both, these lesions constituting the serious and even lethal nature of the affection. No physiological compensation is possible when lesions of both kidneys and liver exist, and symptoms of autointoxication appear and rapidly lead to a fatal issue.

This disease has received a great variety of names, according as the various authorities laid most stress upon the anatomico-pathological process, the nosographic form, or the nature of the disease. Budd called it *fatal icterus*, Oranam *essential grave icterus*, Lebert *typhoid jaundice*, Wunderlich *pernicious jaundice*, Momeret *essential hemorrhagic icterus*, Rokitansky *acute yellow atrophy*, Frerichs *acute diffuse hepatitis*, etc.

We are unable to accept an anatomico-pathological nomenclature, because it often happens that there is no inflammation, but rather a degenerative process, nor does the widely accepted term of acute yellow atrophy seem justifiable, because the hepatic lesion is not always characterized by a reduction in volume of the liver. We have, therefore, adopted the name of *icterus gravis*, following Jaccoud.

Especially must we be careful to distinguish primary icterus gravis from the grave forms of icterus secondary to many hepatic diseases,

rom aggravated icterus. We have already spoken of the possible rence of rapidly fatal jaundice in the course of hepatic diseases er, biliary cirrhosis, venous cirrhosis, etc.), and this is second- cterus. When considering benign febrile icterus we alluded to ıct that a jaundice with light initial symptoms sometimes passed nperceptible degrees or with rapidity into a grave condition h ended fatally. This is aggravated icterus. It is our intention especially to consider grave primary icterus.

Etiology.

ıe disease is more frequent in women than in men, almost in roportion of two to one. While far more usual in youth and life (twenty-five to forty-five years), it has been met with in old ıd in childhood.

e relationship between acute yellow atrophy and pregnancy is oticeable; out of eighty-eight cases observed by Thierfelder in ı thirty were in pregnant women. It would seem to be never in the first three months of gestation, and is most frequent he fifth. It is rare for it to develop after parturition.

erus gravis may be the direct result of a preëxisting infectious , especially abdominal typhoid, relapsing typhoid, septic dis- and puerperal infections. Engel-Roimers twice noted it in the ary stage of syphilis.

ıe endemics of icterus gravis have at times been noticed, as, for e, in the same house and among members of the same fam- .rnould reports ten cases in soldiers who occupied the same

ressing emotions, grief, fear, sudden fright, overwork, vene- cesses, the immoderate use of alcoholic beverages, especially ken in large amounts at a time, are contributing causes to the ment of grave icterus.

lly, direct causes of the disease, which, however, differ as to omical lesions produced, are poisoning by arsenic, antimony, ove all, phosphorus, Munk even going so far as to say that in ll cases of the disease there is phosphorus poisoning which ıaps been concealed. This, however, is an exaggerated state-

Pathological Anatomy and Pathogenesis.

first and most important anatomico-pathological sign of a e of acute yellow atrophy is the atrophy of the liver. This ay be reduced to one-half or two-thirds of its normal size;

or even to one-fourth. The chief reduction is in its thickness, so that the thinned and diminished liver is found in a shrunken state under the diaphragm and towards the spinal column, covered over by loops of intestine which are usually inflated with gas. The hepatic peritoneum not having undergone a diminution in size equal to that of the liver, is too large for its contents and is shrivelled and wrinkled. The diminution in weight may reach 400 to 500 gm. (14 to 17½ oz.). The surface of a cut section is of an intense saffron color, but as a rule is not uniform but mottled, or there may be more or less extensive spots of a reddish color (red atrophy). The parenchyma is softened and friable, sometimes almost fluctuating, and the surface of a cut section does not present the lobulated appearance of normal liver tissue.

Atrophy of the organ occurs only at an advanced stage of the disease. When death has taken place rapidly, the liver is only slightly or not at all diminished in size, nor are the consistence and color as described above, but resemble the conditions found in biliary stasis, accompanied by a greater friability of the tissues. The gall-bladder is almost empty or contains a mucous fluid, scarcely tinged with yellow. Little bile or blood is found in the parenchyma of the organ. A microscopic examination of the portions most affected will show the hepatic cells to be almost entirely destroyed and replaced by a special amorphous substance, infiltrated with embryonal cells, hæmatoidin, fat granules, biliary pigment, and sometimes by crystals of leucin and tyrosin.

The majority of the cells are, as a rule, not completely destroyed; they persist, but their protoplasm almost entirely disappears and is replaced by fat droplets and granulations or true crystals of bile pigment. According to Cornil and Ranvier, the cloudy swelling and biliary infiltration are the first stage of the degenerative process, which in its last stages causes the breaking up and the destructive softening of the cells.

The red spots perceptible to the naked eye are the most advanced lesions of the process. When the parenchyma has been entirely destroyed, if death do not occur, the lymphatic vessels carry away the fat, the granular detritus, and the bile pigments, leaving only connective tissue and vessels more or less filled with blood.

In initial lesions, on the other hand, the changes are less marked; the hepatic cells are the seat of granular swelling, and the parenchyma contains a larger number of extractives. The blood-vessels, especially the hepatic veins, are altered; they are poor in blood and contain a notable amount of leucin and tyrosin. The most important changes, however, concern the bile ducts, which exhibit all the signs

radicular angiocholitis; the smaller canaliculi are often occluded
ıe granular adipose detritus, which would thus appear to be the
e of the icterus. According to Frerichs, however, the obstacle to
tion is the external compression of the biliary radicles by the
ate formed at the periphery of the lobules. Cornil, in one case,
l the interlobular canaliculi dilated and full of cells, and also a
ed development of a network of bile ducts, such as we referred
der biliary cirrhosis.

he kidneys are always affected to such an extent that the dis-
has been described as *hepatonephritis* by some authorities;
are present all the signs of a parenchymatous nephritis. The
onship existing between hepatic and renal lesions has already
discussed in the section on general pathology, and we shall here
y add that the early appearance of the renal lesion, which soon
ıes so severe, would seem to indicate that it is due to the same
as the hepatic affection. The spleen is usually enlarged, soft,
laccid. The blood is thin, grayish black, only slightly coagu-
and contains altered hæmoglobin, which, according to Quin-
l, does not possess its normal respiratory powers. Somewhat
ive effusions of blood may be found in many of the organs, as,
stance, in the peritoneum, stomach, intestines, spleen, mesen-
ıleuræ, lungs, etc. There may be fatty degeneration of the myo-
ım; œdema has been found in the lungs and in the brain. Fi-
fatty degeneration has been met with even in the voluntary
es.

ving thus briefly described the anatomico-pathological lesions
teristic of grave icterus, the first question which arises in re-
o the pathogenesis is whether we have to do with an inflamma-
: a degenerative process. This question was allowable in the
hen the two processes were considered to be absolutely dis-
ut not at the present time, when we know how intimate is the
ıship between the two morbid lesions, so much so in fact that
ıetimes cannot tell whether a tissue is the seat of inflammation
degeneration, as the former almost always merges into the
y imperceptible gradations. It is, however, proper to inquire
of the processes is the most marked. The grave lesions of
atrophy are more especially degenerative in nature, fatty de-
ion of the cells with a tendency to complete destruction being
a characteristic of this disease. It would seem that there is
ial inflammatory period of short duration, for the frequently
ible enlargement in the beginning, the proliferation of the
ınd of the cells which is sometimes found, together with the
ment of new bile ducts, would seem to indicate that this is the

case. This stage is, however, very short, and is quickly followed by the degenerative stage; the rapidity and the prevalence of this process is probably related to the intensity of the morbid cause.

More serious questions in regard to the pathogenesis remain to be answered. What is the direct etiological factor, and what is its nature? Is it infective or toxic? Is the disease a general one involving the liver, or is it an hepatic affection followed by general symptoms?

The morbid form of the disease, its clinical course, and the climatic conditions which favor its development have always suggested an infective origin, and this is the theory at the present day. Many authorities regard it as a general infectious disease, with a special localization in the liver, and Buhl, Bamberger, Coutain, and Murri incline to this view because of the grave lesions found from the outset in the kidneys. According to others (Demme, Liebermeister, Rosenstein) the primary localization of the infection is in the liver, that in the other organs, especially the kidneys, being secondary, and the grave general phenomena are due to accumulation in the blood of the toxic products which have been neither destroyed by the liver nor eliminated by the kidneys.

Many investigators have made bacteriological researches in the hope of finding the pathogenic organism of this disease. The results obtained by Klebs, Tomkins, and Dreschfeld, Hlava, Boinet, and Guarnieri—who from the blood of two patients who died of grave icterus made cultures that showed a special bacillus, which, however, did not completely reproduce the disease in animals—have not received further confirmation, and by the differences existing between them indicate the doubtful nature of the bacteriological data. Neither can much importance be attached to the results obtained by Hanot relating to the presence of the bacterium coli commune in the liver affected by acute yellow atrophy, nor to those of Nepveu and Bourdillon, or Girode, the first two of whom found the streptococcus pyogenes and the second the staphylococcus pyogenes aureus, because the cases were probably streptococcal or staphylococcal infection complicated by acute yellow atrophy. Important negative results have been obtained by Kahler, Pincherle, Dupré, and, above all, by Gabbi, who by his researches upon living blood and hepatic fluid, and his inoculations of living animals, demonstrated the complete absence of any micro-organism.

Although these experimental results do not entirely exclude an infective etiology, since the further perfecting of the technique of experimentation may succeed in demonstrating the existence of a special micro-organism, yet they certainly tend to invalidate the theory, the

e so that autointoxication would explain the morbid phenomena ılly as well. From the latter point of view, there would appear to toxic origin of grave icterus, similar to that previously described ıe case of benign icterus. Autochthonous toxins, or else toxins de- l from intestinal infections, are absorbed by the portal circulation act directly upon the hepatic cells, causing in the first place func- ıl insufficiency which allows of the free passage into the blood of oxic products produced in intestinal chemism, and then a marked omical lesion, while by their entrance into the bile they induce ılar angiocholitis. The grave cholæmia produced, the incom- decomposition of nitrogenous matters, and the total suppression ıe liver functions (true acholia of Jaccoud) further aggravate the toms. The only possible compensation is found in the kidneys, e intimate relation to the hepatic functions has already been al- l to several times. Very shortly, however, the highly poisonous s, in their elimination, act upon the kidneys, and produce first a orary period of overactivity, followed by grave functional dis- nces and anatomical lesions. The intense intoxication accounts ıe grave symptoms which appear in relation with the nervous m, the blood, and all other organic functions.

ıe analogy between the lesions of grave icterus and those of horus poisoning, which is so marked that the anatomico-patho- l data may easily be confounded, and the relations between n febrile icterus and grave icterus, which are so close that the may result from the former, would tend further to corroborate eory of a toxic pathogenesis. Moreover, certain symptoms of icterus which are supposed to be characteristic of infections ılly not to be explained upon that hypothesis. We have already that the fever is not always, and is perhaps never the proof of esence of an organized morbid agent, since it would seem to be dent upon the action of the toxins produced by the bacteria than upon the presence of the bacteria themselves. In connec- ith the splenic enlargement, which is so characteristic of this e, Gabbi calls attention to the fact that Ponfick's experiments he biliary acids and Mya's with pyridine, both hæmatolytic , resulted in enlargement of the spleen from the accumulation blood corpuscles in that organ. The intoxication caused by yellow atrophy would cause an intense hæmatolysis, which account for the enlargement of the spleen.

may, therefore, conclude that while it is not possible to en- exclude an infective origin of the disease, the cause is more to be toxic, as it is in the whole series of icterus, from the t to the most severe; the variation in the course would be in

relation with the intensity of the autointoxication, and with the functional and anatomical lesions of the hepatic cell and the kidneys.

Symptoms and Course.

There are two stages in grave icterus: a prodromal stage very similar to that of the benign forms, and a second period characterized by symptoms of great gravity.

In the first stage we usually have gastrointestinal disturbances of more or less severity. Anorexia, nausea and vomiting, diarrhœa, a dull pain in the epigastrium and right hypochondrium, are the initial symptoms, and certainly do not suggest the graver phenomena which occur later. Jaundice appears, as a rule, within a few days, and according to Frerichs begins in the face and neck, whence it extends to the whole body.

This prodromal period may be entirely absent, the disease commencing with severe symptoms, usually consisting of a chill accompanied by great depression, or more rarely by nervous excitement, intense headache, diffuse pains in the articulations, especially in those of the spinal column, and repeated vomiting. In even rarer cases the course may be so acute as to be fatal in from two to three days.

In the cases with a somewhat prolonged prodromal period the malaise increases, the icterus becomes more intense, the temperature rises and soon attains a high degree, and the characteristic nosological type of disease appears, with the jaundice, the grave nervous symptoms, and the hemorrhages associated with this form of icterus.

The jaundice, whether ushered in slowly or bruskly, may become very marked, its severity appearing to be in some way related to the duration of the process. Sometimes, especially when the disease is secondary to other morbid processes, there may be a true *green jaundice*. Some cases have been met with in which the icterus was absent (Bamberger), and this is especially apt to occur in the cases which are rapidly fatal. The jaundice is accompanied by the usual symptoms. It would seem, from observations of Semmola, that in the early stages of the disease the fæces are rich in bile, while later they become dry, cretaceous, and grayish white, as is always the case in intestinal acholia. This condition is quite in accordance with the functional and anatomical condition of the hepatic cell. An initial hyperactivity manifested by polycholia or pleiochromia is followed by diminution and then complete absence of the biliary function from cellular destruction, at the same time that radicular angiocholitis and

perilobular exudation, according to Frerichs, interfere with the tion of the bile.

utaneous or mucous hemorrhages may occur from the first, or in the advanced stage of the disease. Petechiæ or extensive effu- of blood may be noticed upon the skin, and it is not unusual to epistaxis, hemorrhages from the gums, bronchorrhagia, hæma- sis, hæmaturia, metrorrhagia, etc. These are in direct relation e grave lesions of the blood and consequently of the vascular resulting from the hæmatolytic action of the principal constitu- of the bile and above all of the toxic products which pass into ccumulate in the circulation because of the hepatic and renal iciency.

ervous symptoms are especially characteristic of the disease. begin as irritative phenomena; persistent headaches, which are imes the worst feature of the disease to the patient; marked ssness; insomnia, which is sometimes persistent; occasionally m, which may be so furious as to require the strait-jacket; ate hiccough, tremulousness of the lips and tongue, difficulty in lation, spasms, convulsions of certain muscles, gnashing of the and attacks of trismus are the symptoms most frequently ob- when the jaundice has made its appearance and the clinical e is complete. General epileptiform convulsions are rare, and chiefly when the kidneys are involved. The above-described oms usually last two or three days, and then give way to a sed condition which is far more dangerous. The excitement es, somnolence becomes marked, the sensorium is gradually sed until finally the patient enters into a coma, which ends in

External stimulation is of no effect, the pupils are dilated not react to any stimulus, there is incontinence of urine and sometimes subsultus tendinum may persist in certain muscles. exceptionally the stage of excitement is lacking and the grave oms begin with a marked depression of sensibility and great ness.

have already seen that these symptoms are due to intense oxication following hepatic insufficiency. Cholæmia, and uræ- m the renal lesions induced, certainly assist in their produc-

fever in the first stage of the disease is high and of a continu- ittent type; in some cases, however, it is rather slight (38° 100.4° to 102.2° F.). With the appearance of the nervous phe- there may be hyperpyrexia, or complete remissions; in a eptional cases all the morbid symptoms have been observed e temperature was constantly below the normal (*algid icterus*

of Hanot). The fever, like the nervous symptoms, depends upon the degree of the autointoxication, and the variations in temperature may perhaps be due to the quantity or the quality of the toxic products. The pulse in the early stages of icterus may be slow, but soon becomes frequent, small, and compressible. It has sometimes been known to assume the character of a cerebral pulse (slow and full) with the occurrence of the nervous phenomena. There is always dyspnœa, even when the fever is not high, and when there are no noteworthy lesions of the thoracic organs. This would seem to be due to the changes in the hæmoglobin, noted and studied by Quinquaud.

An objective examination, especially of the liver and spleen, is of the utmost importance to the diagnosis. As is evident from the demonstrations of pathological anatomy, the liver is diminished in size, the area of dulness of the left lobe being the first to disappear; at a later stage the remaining portion of the inferior boundary ascends more and more, and in marked cases a narrow strip in the right axillary line is all that remains of the area of liver dulness. Wunderlich makes mention of a depression in the hepatic region. According to some authorities, hepatic pain is never absent, in spite of the profound depression of sensibility; when pressure is made over the right hypochondrium the patients writhe and twist themselves about. In a few cases a slight increase in the area of the liver dulness has been noticed in the early stages of the disease. In cases with a rapid course, reduction may not follow the initial increase in size.

The splenic tumor may be considered to be a constant sign, but should there be grave gastrorrhagia and enterorrhagia it will diminish in volume.

Gastrointestinal symptoms are never absent in acute yellow atrophy. In addition to the hemorrhages we frequently have vomiting, diarrhœa, or more often obstinate constipation, while the tongue is dark in color as if burned (Chauffard) and the lips and gums are fuliginous.

Owing to the disturbances of circulation, various eruptions are found upon the skin, such as erythema, urticaria, roseola, scarlatiniform eruptions, etc. In pregnant women, profuse hemorrhages occur and induce abortion.

The anatomical lesions of the liver, and the frequent implication of the kidneys account for the importance of the alterations in the urine. This is diminished in amount and towards the end of the disease there may be anuria. Its color depends upon the degree of jaundice; we obtain the usual reactions of the normal and pathological bile pigments (bilirubin, urobilin). A gradual decolorization of

rine during the course of the disease is a bad sign, as it indi-
complete disintegration of the hepatic cell or total renal insuffi-
y. It is a matter of dispute whether there is an increase of urea
first stages (Brouardel and Bouchard), but when the disease
ly established its reduction is marked and sometimes enormous.
quaud found that the elimination amounted to only 0.2 gm.
s.) in the twenty-four hours. The salts are also diminished.
presence of albumin and of casts indicates the kidney lesion.
the decrease in urea there is an increase in the extractives
in, hypoxanthin, kreatin). Of the greatest importance is the
ce of leucin and tyrosin, which Frerichs considers a pathog-
ic sign. We spoke of this in the section on general pathology,
ndeavored to give the most satisfactory interpretation of the
In one case there was found an increase in the aromatic oxy-
with absence of leucin and tyrosin. Sarcolactic acid has been
oxyamygdalic acid, peptonoids, etc.
rmont found that there was diminution in the toxicity of the
according to the renal lesion, and an increase in it in the rare
n which there was a beneficial "urinary crisis."

Duration and Prognosis.

rapidly fatal course to which we have alluded is quite ex-
al. As a rule, death occurs within the first two weeks. Very
he course of the disease is protracted beyond the fourth week;
especially apt to occur when the initial stage has been long.
is the almost invariable result, and occurs either with grave
phenomena or without profuse hemorrhages, or else as the
ence of the uræmic condition. Bouchard considers myosis
important diagnostic sign of uræmic poisoning; mydriasis
e condition present in hepatic autointoxication.

prognosis is not always fatal, as we might be inclined to
since there have been some well-authenticated cases of cure
en the icterus was accompanied by grave nervous symptoms,
orrhages, by reduction in size of the liver, and by renal
These cases are, however, altogether exceptional, and not to
ed upon. But we should not altogether despair of a recovery,
ly if we are able to ascertain that the kidneys are not in a bad
n. In his practice Semmola has seen only one case of icter-
s which terminated in recovery, but it was a case in which the
age was more or less benign, and the later stages only were
ristic of grave icterus with a reduction of the area of liver
nervous phenomena of excitement, and marked albuminuria

and hypoazoturia; he has never seen a cure of a genuine case of acute yellow atrophy.

As Bouchard, Hervouet, Brouardel, and Chauffard were among the first to point out, the favorable issue is heralded by the "polyuric and nitrogenous crisis" already described, which, according to the last-named author, is never absent in a case which ends in recovery. An abundant watery or biliary diarrhœa and profuse sweating are good prognostic signs (Mossé). By these means the system is rapidly relieved of all the toxic products which are the direct cause of the grave condition and of death. They are followed by rapid improvement; the cutaneous and mucous hemorrhages cease, the nervous symptoms completely disappear, while objectively we may ascertain a progressive increase in the size of the liver and a disappearance of the pain in the hypochondrium. Convalescence is long because the organism requires a considerable amount of time to repair the ravages of the disease, and during its course the greatest care should be taken to commit no excesses which might cause a return of the grave symptoms.

Senator[7] believes in the curability of grave icterus, more especially when it is due to syphilis.

Diagnosis.

The diagnosis of this disease can be made only in the later stages, the symptoms of the first stage being indistinguishable from those of ordinary febrile icterus, or even from simple catarrhal icterus. When the jaundice appears, however, accompanied by nervous phenomena, hemorrhages, a decrease in size of the liver, and a splenic tumor, and when at the same time there is marked hypoazoturia the diagnosis is easy.

In some cases a general septic condition might be confounded with this disease, but the clinical history, the presence of endocarditis (ulcerative), the symptoms relating to the reduction in size of the hepatic area, and the nature of the fever will prevent error.

The symptoms of phosphorus poisoning greatly resemble those of acute yellow atrophy of the liver; but there are vomiting, intense gastralgia, and enterorrhagia, the liver is enlarged, nervous symptoms seldom occur, and, according to Strümpell, leucin and tyrosin are not found in the urine. An important diagnostic point is the fact that in phosphorus poisoning a period of three or four days occurs between the acute gastroenteric symptoms and the appearance of the icterus, during which time there is a complete and often deceptive remission of the symptoms.

Yellow fever may be distinguished from acute yellow atrophy by

eographical distribution, its brusk invasion with rachialgia and vomit, the biliary diarrhœa, frequent hæmaturia, and high con- us fever.

Treatment.

he treatment of this grave form of icterus is that of febrile ic- which we have already described. A rigid milk diet, intestinal fection (alkaline hyposulphites), cold-water enemata, and symp- tic treatment are the methods at our disposal.

n view of the incomplete oxidation of albumin due to the renal is, some authorities have advocated oxidizing medication (in- ions of oxygen, benzoate of sodium, and even the subcutaneous ion of turpentine). This treatment is undeserving of considera- The human body is not a test tube nor a chemical furnace, an we expect to obtain in it the reactions which are artificially ced in the laboratory. The incomplete reduction of albumin not mean that oxidation is imperfect from lack of oxygen; if æmoglobin is unable to absorb it, it is not due to its scarcity in spiratory tract. Both facts are merely the result of complex onal and nutritive disturbances in the organs which preside hese biochemical processes. Treatment by oxygen therefore is e therapeutic fad.

Emotional Icterus.

at jaundice of some degree of severity could, without prodromal oms, occur suddenly as a result of intense mental emotion was ed in remote times, was subsequently denied because of the lty in accounting for its pathogenesis, and has of late, as the of accurate observations, been reinstated in its proper place. notions which give rise to this jaundice are usually of a de- ng nature. Rendu reports the case of a child in whom jaun- curred within three-quarters of an hour after fright occasioned ing to undergo catheterization. Chauffard tells of a case in a ho during a quarrel was obliged to exercise an enormous t of self-control to prevent himself from committing violence. reports the case of a man (during the Commune) in whom ic- appeared just before he was shot. In our own experience a asserted that jaundice appeared within half an hour after he hat his son had committed suicide.

special characteristics of this form of jaundice are its rapid ance, the almost complete absence of intestinal disorders and , intestinal acholia (although Chauffard held that there was al period of polycholia), and the absolute benignity of the af-

fection, which as a rule disappears in from four to five days. In some exceptional cases its course is longer, and similar to that of catarrhal jaundice.

Two theories have been advanced in explanation of the pathogenesis of this disease. Potain believes that the psychical injury causes a centrifugal reflex which reaches the liver by way of the splanchnic nerves, causing dilatation of the blood-vessels and perhaps exciting a flow of bile. This would be icterus from pigmentary polycholia.

The action of the splanchnic nerves upon the hepatic circulation not being clearly explained nor the occurrence of this reflex process experimentally demonstrated by this somewhat artificial theory, we prefer the view held by Lépine, that a spasm of the large biliary ducts is caused reflexly by the emotional injury. This theory has moreover been completely sustained by the experimental researches of Oddi who demonstrated the existence of a true sphincter of the ductus choledochus, which was capable of contraction under reflex stimulation, as well as the existence of a special reflex centre in the spinal cord. Therefore it is easy to conceive of a spasmodic contraction of this sphincter, with a resulting obstruction of the biliary circulation, and absorption of the bile by the blood. Nor does the persistence of the icterus beyond the time which would seem natural for a simple biliary spasm militate against this view, because we know that even when the biliary circulation is restored, by reason of the affinity of the Malpighian reticulum for the biliary pigment the latter leaves it very slowly, or does not leave it entirely, the cutaneous color disappearing only when the skin desquamates.

Some points in the pathogenesis still need to be cleared up; for instance, it is difficult to understand the rapidity with which the jaundice arises, when we know that it is produced slowly even when the ductus choledochus is tied.

The prognosis is good, and the treatment is based upon considerations which we have already several times enumerated. In addition, anodynes, more especially the bromides, are to be given.

Icterus of the New-Born.

From the second to the fourth day after birth, it is not unusual to note the rapid appearance of a diffuse jaundice, especially of the limbs. General and digestive symptoms are entirely absent. The affection would seem, however, to occur more commonly in debilitated infants rather than in the robust. The course is benign; in one or two weeks the jaundice gradually disappears without leaving any note-

hy trace. Should other morbid lesions arise the result may be

.

'he pathogenic theories of the disease may be gathered into three ps:

. The hæmatogenous origin (Virchow, Ebstein, and others), in r words, the breaking up of the red blood corpuscles in the first d of life, and the metamorphosis of the hæmoglobin in the cir- ing blood into bile pigments.

Origin by absorption from stasis into the bile passages. Vir- and Bamberger have suggested a possible occlusion of the s choledochus or of the large bile ducts by plugs of mucus; chs and Naunyn changes in the circulation, more especially a ished pressure in the portal vein after parturition; Birch- hfeld explains the phenomenon by the fact that after birth d venous hyperæmia of the liver occurs, causing œdema of n's capsule and pressure upon the interlobular bile ducts.

Diminished absorption of bile by the intestines. The meco- is very rich in bile pigments (Quincke).

e most reasonable of these theories appears to be that which ates the jaundice to the greater destruction of blood corpuscles the change in the external surroundings. This does not mean e bile pigment is derived from the hæmoglobin in the blood t, but rather that pigmentary polycholia, inspissation, and of the bile are caused by the increased destruction of cor- s, and there is of course, increased reabsorption. That icterus orum may also be due to an altered biliary function has been by the presence of the biliary acids in the urine.

Secondary Jaundice.

(Secondary Biliary Infection.)

en describing the various hepatic diseases we called attention al instances to the presence of jaundice, laying especial stress s pathogenesis, and in the chapter upon icterus gravis we f the possibility of its resulting from other hepatic diseases. subject we shall therefore not return. Neither is it our inten- lescribe all the forms of jaundice which may follow general us diseases, for this would be to invade a field which properly to others. We desire to speak of those forms of icterus only ccur from internal or external occlusion of the bile ducts.

Etiology.

The most frequent cause of internal obstruction of the bile ducts is a biliary calculus; more rarely we have vesicles of echinococci passing through the ducts, or the development of the multilocular cysts throughout the biliary system. Conditions so rare as to be altogether exceptional, consist in the immigration into the bile ducts of the ascaris lumbricoides, grape seeds, cherry pits, etc., which by their presence cause an obstruction to the flow of bile. Even the distoma, as we have already seen, may develop in the bile passages and cause a stasis, producing an icterus from reabsorption. Finally, neoplasms, especially of a cancerous nature, may have their primary development in the bile ducts.

With equal frequency stenosis or complete obstruction of the biliary passages may occur from a pathological condition of neighboring organs. Cancer or round ulcer of the duodenum, the first by its development and the second by processes of cicatrization, may invade the opening of the ductus choledochus; cancer of the stomach or colon, tumors of the uterus, ovaries, pancreas, mesentery, retroperitoneal lymphatic glands, and omentum, aneurysms of the hepatic or mesenteric artery, enlarged kidneys, neoplasms of the kidneys, and finally chronic peritonitis with the formation of cicatricial fibrous bridles, etc., may either by compression or by forming adhesions with the vessels of the hilum of the liver produce stasis of the bile and thus cause a secondary icterus.

Finally, a rare cause of secondary icterus is found in right diaphragmatic pleurisy and acute perihepatitis. These act, not by causing pressure or stenosis of the bile vessels, but by interfering with or preventing the respiratory movements of the diaphragm, thus limiting or annulling their beneficial effects upon the biliary circulation. What these are we can realize by recalling how the increased intra-abdominal pressure in inspiration assists in the excretion of the bile.

Morbid Anatomy.

It is unnecessary to describe here all the lesions which we have already enumerated as the causes of biliary stasis. We shall therefore confine ourselves to a description of those which are caused in the liver from occlusion or stenosis of the bile ducts, and which by their consequent infective complications are of the greatest diagnostic and prognostic importance.

In whatever portion of the biliary system the obstacle is situated,

ile ducts above the point of stenosis will be found to be dilated listended from the secretory pressure of the bile, the part below more or less empty and discolored. The walls of the bile ves- above the obstacle are sometimes thickened, but in some cases are on the contrary markedly thinned. When this is the case, epatic biliary apoplexy is liable to occur with more or less de- ion of the hepatic parenchyma, or the rupture of large bile into the peritoneum.

the stenosis persists the specific constituents of the bile in the above it are reabsorbed and replaced by a muco-serous fluid hed by the mucous membrane of the ducts, a fluid which, lly enough, takes on a greenish-yellow tinge. In the first stage s process the liver is enlarged and full of bile, but later, if the le persist, all the anatomico-pathological consequences occur we have already described in the chapter upon biliary cirrho- d need not repeat. In the other organs and tissues will be the lesions characteristic of icterus, described in the preceding rs.

r special concern is to describe the results as regards infection, follow any obstruction to the excretion of the bile. When treat- the general pathology of the liver we mentioned the aseptic con- of the bile and the conditions which favored its loss of this eristic, and we dwelt especially upon the fact that the bile is adily infected from the intestine when its flow is obstructed, en the obstacle is near Vater's ampulla (Netter). This is why tion of intestinal origin is liable to arise when there is any in- or extrinsic obstruction to the biliary circulation, and especially is situated near the mouth of the ductus choledochus. In e of intrinsic obstructions of the bile ducts, there are some conditions which favor the occurrence of infection. Thus when s have emigrated from the intestines they may infect the ctly, their bodies being the agents upon which the micro-or- of the intestines are carried. In the case of calculus, it has ted that the more numerous the calculi the more probability of infection, as they migrate with greater frequency into the s. If a calculus, instead of having a smooth surface, is rough ular, the lesions which it causes in the bile ducts are so many s for infection of either biliary or hæmatic origin. Finally, infections occurring in individuals suffering from choleli- ay become localized in the biliary passages as a place of sistance, and thus occasion biliary infection.

e are three anatomico-pathological varieties of infection of passages: *catarrhal*, *suppurative*, and *ulcerating angiocholitis*.

The first named greatly resembles that form of disease which we have already considered, and is a true catarrhal condition of the mucosa, anatomically similar to catarrh of other mucous membranes, consisting of redness, swelling, increased mucous secretion, etc.

The most common form of angiocholitis secondary to an infectious process is the suppurative, and of this there are several types.

Biliary abscess, formed at some part of the wall of the bile duct, the size of a pea or of an almond, and filled with a yellowish or greenish (from the presence of the bile) muco-pus, its walls formed of embryonal tissue, or if it be of long standing of fibrovascular tissue. It may be single, multiple, or formed by the confluence of several areolar abscesses.

Disseminated miliary abscesses of varying sizes, but usually smaller than the first variety which infiltrate the whole liver, giving it in some cases a sponge-like appearance.

Suppurative angiocholitis has been experimentally produced by ligation of the ductus choledochus without observing the rules of asepsis (Charcot, Garbault).

The ulcerative form is the most rare. The ulcers, which are usually circular in form, are of varying number, and are usually found in the gall-bladder or in the larger bile ducts, as they are usually the result of biliary calculi.

The suppurating abscesses and the ulcers may cause grave general or local lesions. Thus the opening of an abscess into the branches of the portal or the suprahepatic veins may give rise to a grave septic or pyæmic general condition, ulcerative endocarditis (Netter and Martha), purulent meningitis (Josias), suppurative pylephlebitis, etc.

When an obstacle of any nature, intrinsic (calculi, etc.) or extrinsic (tumors, cicatricial bridles), compresses or causes stenosis of the duct of the gall-bladder, we shall first have stasis and dilatation, followed by dropsy of the gall-bladder. When secondary infections occur they may chiefly affect this organ, producing abscess of its walls or true empyema (suppurative cholecystitis).

Many researches have been made of late, especially in the French and Italian schools, with the object of determining the pathogenesis of biliary infection, and we may venture to draw the conclusion that the etiology varies, as there are many forms of bacteria capable of migrating under favorable conditions from the intestines into the bile ducts and there causing infective angiocholitis. To describe in detail all the researches which have been made would be to enter upon a theoretical discourse of little clinical or therapeutical value. We will merely mention, among the bacteria that have been found, the streptococcus pyogenes, the staphylococcus pyogenes aureus, the

erium coli commune, the typhoid bacillus, and the diplococcus of nkel.

Experimental researches have also been made with a view to reucing infections of the bile ducts. Suppurative angiocholitis been caused by a direct injection into the biliary passages of the erium coli and the staphylococcus pyogenes. Gabbi caused urative angiocholitis by injecting the bacterium coli directly into lood of a rabbit. The intestines are the usual point of entrance is micro-organism, a fact which is the more readily understood se the intestines are its ordinary habitat.

abbi's experiments would seem to prove that the pathogenic -organisms may be introduced through the blood, but the reses of Corrado,[72] on the other hand, appear to demonstrate his is an impossibility.

Symptoms.

aving already discussed and explained the symptoms and the genesis of stasis jaundice, and shown the variations in the of catarrhal and other idiopathic forms of icterus, besides havmonstrated in the section on biliary cirrhosis the anatomicological and clinical events following prolonged stasis of the e will not burden our readers with what would be a mere rep- of symptoms, although referring to secondary icterus. We merely call attention to the fact that the course, issue, and sis of secondary jaundice depend absolutely upon the cause ing it. If it is a removable cause (biliary calculus, echinococughter cysts, intestinal parasites, removable tumors of the inal organs, floating kidneys, diaphragmatic peritonitis, etc.) ndice disappears with the cause. If it cannot be removed of the biliary passages, a large calculus wedged into a large ct, malignant tumors of the liver and neighboring organs, ial bridles, chronic peritonitis, etc.) the jaundice will become . In that case symptoms of cholæmia will occur accompanied stinal acholia; the liver becomes enlarged, sometimes attaining mous size; the gall-bladder is immensely increased in volume mor) and filled with bile from the increased bile pressure, so ors in diagnosis are liable to occur (echinococcus cysts, ab- etc.); the general nutritive condition becomes gradually , and after a while all the morbid phenomena of biliary s occur.

symptoms which arise after an infective angiocholitis is esd are still more deserving of consideration. Infection of

the bile passages may be introduced by a prolonged latent period. In practice we frequently meet with cases of this disease which prove rapidly fatal from pyæmic and general septic symptoms occurring during the course of a jaundice from stasis, and at the autopsy are found lesions which from their stage of development must necessarily have been of a more ancient date than the symptoms led us to believe possible. In some cases, on the other hand, latency is entirely absent, the characteristic symptoms occurring in the initial stages of the biliary infection.

As a rule, objective symptoms on the part of the liver are lacking. Increased pain in the right hypochondriac region and a relative increase in size of the hepatic gland may be entirely overlooked, or may be attributed to one of the frequently occurring periods of recrudescence of the morbid phenomena in icterus from stasis. In still another class of cases there will occur a grave infection of the blood from the opening of hepatic abscesses into the branches of the portal vein or into the suprahepatic radicles, resulting in symptoms of septicæmia, ulcerative endocarditis, meningitis, or finally, propagation of the suppurative process to the vena porta, causing a purulent pylephlebitis, death being the inevitable and rapid result.

More frequently in this disease we find a symptom which is of the utmost diagnostic importance, namely, fever. This was first studied by Charcot, and has received the name of *hepatic* or *biliary fever*.

It may have one of three types (Accorimboni): that of *single abscess*, an *intermittent*, or an *irregular* type.

The single abscess is ushered in by a severe initial chill, followed by a tolerably high temperature, which usually ends in sweating (hepatalgic fever of Charcot). This form of fever accompanies hepatic colic, that is to say, the passage of a calculus through the biliary passages causing a temporary obstruction. It may accompany or follow the pain, but does not depend upon it, since it may sometimes occur when there is no pain.

Intermittent hepatic fever, on the other hand, occurs in cases of permanent intrinsic or extrinsic obstruction of the ductus choledochus (Charcot). Its course is precisely similar to that of malarial fever, starting with a severe chill, reaching a degree of temperature which is sometimes very high, and ending in profuse sweating. It is chronic, and as a rule takes on the quotidian type, but it may more rarely assume the tertian or quartan. There may be complete and prolonged remissions, but it is sure to reappear suddenly.

The irregular type may be intermittent, or remittent, or the attacks

be of several days' duration. It often assumes a continuous-retent form, with grave depression of the patient's strength.

As Reynard, Brouardel, and Charcot were the first to point it is of the utmost diagnostic importance to note the behavior urea in this form of fever. While in fevers of other origin amount of urea is increased because of the increased organic bustion, in hepatic fever we have marked hypoazoturia. This, ve have before remarked, must be due to the hepatic insufficy which becomes more pronounced during the febrile movet.

Charcot investigated the pathogenesis of hepatic fever, even besecondary infection of the biliary passages had been the subject udy. It would appear to be the direct result of the absorption the bile passages of a pyretogenous septic principle, abnormally d in the bile. The name of *biliary septic* fever is therefore justie, and from its origin Charcot compares it to uroseptic fever. r researches have shown Charcot's hypothesis to be correct when are infective lesions in the bile ducts. Some authorities (Dupré, ger) hold the view that the fever is due to migration into the l of the pathogenic micro-organisms of angiocholitis, but this y needs further substantiation. It would seem more likely that line with Charcot's theory) biliary infection gives rise to special s which enter into the circulation and produce the characteristic

Prognosis.

nis depends upon the primary disease. Infectious processes of liary passages usually result in death, but they would appear to casionally curable. Prognosis should be reserved, since the osis itself is a matter of such great difficulty.

Treatment.

ie rules already given for the treatment of icterus apply with force to this affection. In a few cases surgery may intervene nove the cause of the disease (tumors, calculi).

treat or even to prevent biliary infection, recourse should be o intestinal antisepsis by means of milk and the alkaline ulphites; up to a certain point the use of the salicylates or lic acid and of salol is to be recommended, for they are elimiby the bile and may accomplish a certain amount of biliary psis. Injections of quinine are absolutely useless to reduce er. Chauffard asserts that he has often observed intermittent

hepatic fever fall after the administration of 4 to 5 gm. (60 to 75 grs.) of salol.

Symptomatic treatment is often called for and serviceable.

Biliary Lithiasis.

(Biliary Calculi; Gall-stones; Cholelithiasis.)

Although biliary calculi were first observed by Keulmann in 1655, the relation existing between them and the special morbid symptoms of biliary calculi was recognized only about fifty years ago. The study of this disease was pursued in a manner diametrically opposite to that usually followed; it began with a study of the pathological anatomy, and thence proceeded to observation of the clinical facts. The first chemical analysis of biliary calculi was made by Galeati, but their true chemical composition could not be fully understood until their principal constituent, cholesterin, was discovered.

The later researches of Joucroy and Thénard and those of Portal, Bocillard, and Bouisson, cleared the chemical field, while the valuable work of Jaconneau-Dufresne demonstrated the precise relationship between biliary calculus and the special symptoms of the disease.

Etiology.

Biliary lithiasis is of tolerably frequent occurrence, especially in certain parts of the globe. It is most common in northern regions, and some have endeavored to prove that this is due to the more fatty diet of these countries. This theory has not received clinical support, for Schüppel demonstrated the rarity of the disease in Finland where fatty substances form the chief articles of diet.

Nor is greater weight to be attached to the opinion that cholelithiasis is caused by a scanty use of water, or an excessive use of acid drinks, or the drinking of water rich in lime salts, the action of these alimentary conditions upon the chemical composition of bile being extremely problematical. To hold that the inspissation of the bile is due to the small amount of water ingested, its acidification and richness in lime salts (which are the cause of the precipitation of the cholesterin), to the excessive use of acids or lime water, is to reduce pathological facts to preëstablished laws without taking account of the natural compensatory processes which maintain the organic equilibrium.

Of greater importance are the individual conditions which contribute to retard the circulation of the bile. Biliary calculus is more

uent in women than in men. The statistics of Stein, Durand-
lel, Schroeder, etc., completely establish this to be a fact, and
may consider it proved that two-thirds of the cases occur in
en. Pregnancy, sedentary habits, etc., have been supposed to
unt for this peculiarity. Age has a certain influence. It has been
ed that the disease is more frequent from forty years upwards,
cases are on record of its occurrence in youth and even in the
y born. A sedentary life, taking of meals at long intervals, and
circumstances appear to be etiological factors of this disease,
ugh Naunyn[73] holds a contrary opinion.

ll the above-mentioned causes act by interfering with the free
of bile and causing its retention in the gall-bladder and bile ducts.
lult or advanced age, in women, in those leading a sedentary life,
take meals at long intervals, and when pregnancy diminishes
uscular work, reduces the number and depth of the respirations
lowers intestinal peristalsis, we find conditions which interfere
the free flow of bile, which therefore remains stagnant beyond
ormal period in the biliary passages.

yr, who upholds the chemical theory in regard to the pathogene-
cholelithiasis, thus reducing the formation of a calculus to a
process of crystallization, believes the two essential conditions
chemical alteration of the bile and a retarded circulation.
idual and hereditary predisposition also play a part in the
gy of calculus. Biliary lithiasis is in fact largely dependent
a diathetic condition which belongs with the arthritic diseases,
as gout, rheumatism, urinary lithiasis, obesity, diabetes, and
a, which, as Bouchard has so well demonstrated, depend upon
etardation of metabolism. This relationship has been mis-
stood by many, but we have become convinced from practical
ience that it is of the utmost importance.

the previous or family history of patients suffering from cho-
asis be thoroughly investigated, we shall usually be able to
some hereditary taint or the account of some disease which will
m the fact of a relationship between general nutritive disorders
iliary lithiasis. We must bear in mind that the causes referred
ve, relating to the mode of life, the quality of the food, etc., are
quent and common that the wonder is that the disease does not
more often than is the case. For this reason we do not find it
le to attach much importance to them as etiological factors, but
e rather in the relationship between cholelithiasis and the ar-
diathesis. This theory assumes a stronger position the more
tudied in the light of clinical facts.

Physical Characters.

Calculi may be found in any part of the biliary system, but occur with most frequency in the gall-bladder. They originate in this situation and in the small biliary ducts. They are not found in the ductus choledochus nor in the cystic or hepatic ducts except when they emigrate and become impacted therein. Their size, number, and shape vary greatly in different cases. We must in the first place distinguish gravel from true calculi, it being possible for both forms to exist together. Gravel is formed of the most minute granules resembling the finest sand, mixed with and suspended in the bile, which therefore appears to be more or less thickened. It is nearly always dark in color, greenish or even blackish, quite exceptionally grayish or a whitish gray. It is usually found in the gall-bladder but may sometimes occur in the intrahepatic ducts.

Biliary calculi may vary from the size of a millet seed to that of a pea, a pigeon's egg, or even larger. Some have been described as from 10 to 15 cm. (4 to 6 in.) in length and not less in circumference. That calculi of the gall-bladder attain such dimensions can be easily understood, when we remember that their size may be limited only by that of the cavity which contains them.

Their form also varies, and usually depends upon the place where they are formed or to which they are carried, they being subject to modifications in size and shape. In the gall-bladder they are usually rounded and more or less oval, but sometimes they are angular or full of knobs. When the calculi are numerous, they may be cubical, tetragonal, octagonal, and more or less regular according to the reciprocal positions of their surfaces. The cavity in which they are situated being limited in extent, as they develop they tend to lose their round shape and take on a polyhedral form, from attrition of their opposing surfaces. Sometimes calculi in the gall-bladder form true articulations, a projection from one fitting into a depression or a cavity in another.

Rarely a solitary calculus has been found of a size sufficient to fill the whole gall-bladder and a part of the cystic duct, and hence pear shaped like the cavity containing it. Calculi of the bile ducts are usually cylindrical, and often branch dichotomously; sometimes they are hollow, the central lumen being filled with inspissated bile. In number they may be from one to hundreds, or even thousands. Some cases have been reported in which there were five, six, and seven thousand (Naunyn, Otto), and in some cases the liver was as if calcified and could not be cut with a knife.

The color is usually dark, but varies, according to the chemical
nposition of the calculus, from a more or less grayish-white and
enish-gray to green or black, these variations depending upon
amount of bile pigment which the calculus contains. The con-
ence of the calculus also depends upon the chemical composition;
rule, it may be crushed between the fingers, but it may be hard
calcareous. The specific gravity varies, but is always more than
of water in newly formed calculi; as they dry they may become
ter.

As to their chemical composition, cholesterin is their most fre-
nt component. Then in the order of frequency we have bilirubin,
lly in combination with carbonate of calcium and other rarer
(phosphate and sulphate of calcium, phosphate and car-
ate of magnesium, traces of iron, copper, and manganese), the
ry and fatty acids, mucin, and epithelial cells from the bile
ages.

n structure calculi are very variable, and may be divided into
main classes, homogeneous and mixed. In the first the chemi-
nd physical composition are the same from the periphery to the
e. In the second a cut section usually shows three concentric
s: 1. A cortical layer of a deeper color and harder consistence,
ntrically stratified, formed of bilirubin or of lime and bilirubin;
central nucleus composed of mucus infiltrated by pigment or
me foreign body; 3. A median radiated zone, composed of
sterin of a light color and more or less transparent.

iliary calculi have been found with more than one nucleus (some-
as many as five), in which case there would seem to have been
a of a number of small calculi each one of which retained its
ucleus.

ncerning the chemical composition of the different varieties of
tones the reader is referred to the article on "Diseases of the
ladder," below.

Pathogenesis.

though the pathological anatomy and the symptomatology of
calculi are well known, the pathogenesis is still obscure. Ex-
ental research has been directed to the elucidation of this im-
t part of hepatic pathology, but the results obtained do not
tely nor clearly explain the morbid process.

e first effort of investigators was directed to the reproduction of
by introducing foreign bodies into the gall-bladder of the dog,
ndeavoring to bring about a catarrhal condition of the mucosa,
e results were invariably negative. Occasionally some of the

elements of the bile would be precipitated (Marcantonio), but never cholesterin which is so frequent an ingredient of calculi.

Two principal theories are held by investigators, the chemical and the anatomical. According to the chemical theory, valiantly upheld by Thudichum and by Bouchard, calculi are formed by precipitation of the cholesterin from special chemical changes in the bile. Cholesterin is dissolved in the bile by the presence of alkaline bile salts and soaps of sodium and potassium which, however, dissolve a certain determined amount only. If, as a result of the nutritional exchanges in the tissues, nerve tissue especially, cholesterin is formed in large amount and is eliminated by the bile, it cannot all be dissolved, and is precipitated. Even when it is not increased in amount it may precipitate, if the alkaline reaction of the bile is neutralized or changes to acid. Two factors may contribute to this condition, either a permanent excess of organic acids in the fluids of the body, or their insufficient intraorganic combustion. At the same time the organic acids set at liberty a larger amount of lime than normal from the tissues, and this, being eliminated in the bile, forms calcium soaps and bile salts of calcium, both of them insoluble. The diminished amount of the glycocholate and taurocholate of sodium and potassium further contributes to the precipitation of the pigment which they hold in solution.

This theory has been violently opposed, especially by Naunyn and his school, the chief objections being that the proportion of cholesterin in the bile is constant, and is not found to be increased in cholelithiasis, and that the bile contains an amount of biliary salts, soaps, and fats greater than is necessary to dissolve its cholesterin.

A theory which occupies a position between the chemical and the anatomical is that the cholesterin is precipitated because of catarrh of the bile passages leading to the formation of acid mucus which transforms the glycocholic acid in which the cholesterin is held in solution in the bile into cholic acid, which is not capable of dissolving it. This hypothesis is, however, untenable, Naunyn having shown that cholic acid does dissolve cholesterin.

Meckel and more recently Naunyn have abandoned this theory, and hold to the anatomical, considering that the calculus is formed by a special catarrhal condition of the bile passages (lithogenous catarrh), whose function it is to separate the cholesterin and the salts of lime, and which, by reason of this inflammatory process of the mucosa, increase in amount. Calculi having by this theory been proved to depend upon catarrh of the bile passages, a cause for the catarrh was next sought; the researches so far made in this direction

ıld tend to demonstrate a bacterial origin. The finding by Naunyn
ı special bacterium in certain intrahepatic calculi, the precipitation
ıilirubin and lime by the bacteria of putrefaction, the fact that
lelithiasis may follow typhoid infection (Dupré, Dufour), the
ıonstration of the microbic origin of other calculi analogous to
biliary, such as salivary calculi (Galippe, Maiocchi), are all facts
ch go far towards proving the parasitic origin of lithogenous ca-
h, which is the fundamental condition of biliary lithiasis.

The unsatisfactory nature of all these theories is, however, evident,
the question can be resolved only by further and more profound
ies. To our minds the most important pathogenic condition
ld appear to be the chemical composition of the bile. It is evi-
that the cholesterin, lime, and pigments must exist in a relatively
ortional amount in order to be in a state of solution, and that
ld this amount be altered, precipitation of one or more of these
tituents might follow. It is, moreover, presumable that, just as
ıe case of the gastric juice and other organic secretions, devia-
in the normal composition of the bile may occur, due either to
ional conditions of the hepatic cells or to altered chemical com-
ion of the blood coming to the liver from the gastrointestinal

he composition of the bile will necessarily vary according to the
ances from which it is derived, and the special biochemical ac-
of the secreting cells. For this reason the study of the patho-
is of cholelithiasis should be directed especially to investigation
e chemical variations which the bile undergoes. Perhaps when
have been accurately determined in their relation to the quality
portal blood, and to the processes of gastrointestinal digestion,
ay obtain some light upon this subject which, in spite of the
researches and theories, is still shrouded in darkness.

ıe symptoms of biliary lithiasis will vary according to the seat
ize of the stones. For their consideration, as well as for that of
eatment of this condition, the reader is referred to the article on
ases of the Gall-bladder."

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EASES OF THE GALL-BLADDER.

BY

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DISEASES OF THE GALL-BLADDER.

ANATOMY.

THE gall-bladder is situated in a fossa or depression on the under face of the right lobe of the liver, the quadrate lobe being to the

When distended it projects from one-half to three-quarters of nch below the edge of the liver just beneath the ninth costal car-ge. When the body is erect, the long axis of the gall-bladder runs ards, backwards, and outwards. The fundus is in immediate con- with the hepatic flexure of the colon, the body of the gall-bladder ing upon the first portion of the duodenum. The gall-bladder is ated above the great omentum, and in dealing with it surgically should always be borne in mind, as it contributes materially to safety of operations. The gall-bladder is pyriform in shape. It nds almost directly backwards, deviating only a little to the right upwards. It measures from two and one-half to four inches (65–nm.) in length and from one to one and one-half inches (24–36) in diameter in its widest part. It holds from one to two and alf fluidounces (30–75 c.c.), and is always full except when external pressure is brought to bear upon it. Its upper surface ached to the liver by connective tissue to within one-half to one of the tip of the fundus. The lower surface is covered with oneum, which occasionally entirely surrounds the viscus and s a mesentery to attach it to the liver. The neck of the gall-ler curves upon itself, and with the cystic duct into which it es describes the letter "S" at the transverse fissure of the liver.

he wall of the gall-bladder consists of three coats: the serous, ibrous and muscular, and the mucous. The serous coat cov-nly two-fifths of the circumference—that on the under side. ibrous and muscular coat contains more of fibrous than muscu-ssue and has very little power of contraction. It has, however, asticity, and when the bile pressure increases the gall-bladder s; when it decreases it again contracts. I have demonstrated he gall-bladder, with the normal bile pressure, will expel on erage only about thirty minims, or about five to six per cent. e contained bile, by its inherent contractile power (*Medical d*, March, 1894). The mucous coat is raised in rugæ—bound-

ing polygonal spaces which are largest at the fundus. These rugæ form circular elevations in the neck of the gall-bladder which act as valves and are supposed to play an important part in cholecystic colic and in the formation of hepatic calculi. The mucous membrane is lined with columnar epithelium and contains many mucous glands. Pressure upon the surface expels a green slimy fluid containing small grayish-white clumps. The clumps consist of clusters of agglutinated epithelial cells. These are credited with forming the nuclei

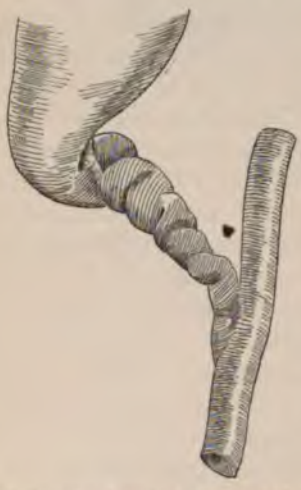


Fig. 46.—Curvature of the Neck of the Gall-Bladder, Flexion of the Cystic Duct, and Angle at which it enters the Common Duct. (Heitzmann.)

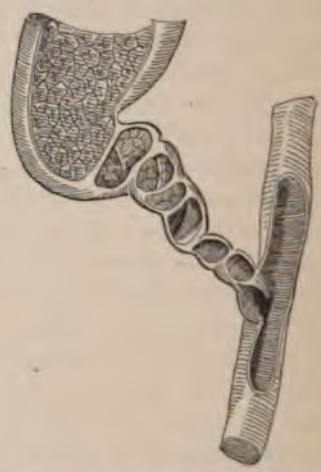


Fig. 47.—Same as Fig. 46, with the Canals Open, showing Heister's Valves.

for gall-stones either when expelled completely from the gland or when protruding from the opening in the gland duct. The mucosa at the neck of the gall-bladder contains a delicate anastomosis of blood-vessels and a fine plexus of lymphatics, which account for manifestations of sepsis from infected lesions of the gall-bladder. The lymphatics of the gall-bladder empty into the hepatic glands, which are situated in front of the portal vein, between the layers of the gastrohepatic omentum. These glands receive also most of the superficial lymphatics from the under surface of the liver.

The gall-bladder is very vascular, receiving its blood supply from the cystic artery, which is a branch of the right division of the hepatic. There is an extensive anastomosis of blood-vessels in all directions. The cystic vein empties into the portal, consequently septic thrombi of this vein if liberated would be arrested in the liver. All of the coats bleed profusely and for a long time when incised;

y are richly supplied with sensitive nerves and very painful when tated. When the fundus was irritated, the pain was referred to epigastrium in seventy per cent. of cases of cholecystostomy mined by the author. In sixty per cent. of the cases when the k was irritated with a sound the pain was reflected to the back on right side, just beneath the angle of the scapula. In eight per . it was referred to the left side, in the same relation to the ula, and in the remainder he cases in irritations of fundus, body, and neck, sensation of pain was lo- d at the site of the gall- der. These percentages esent approximately the lization of the pain from stone pressure in the us regions mentioned.

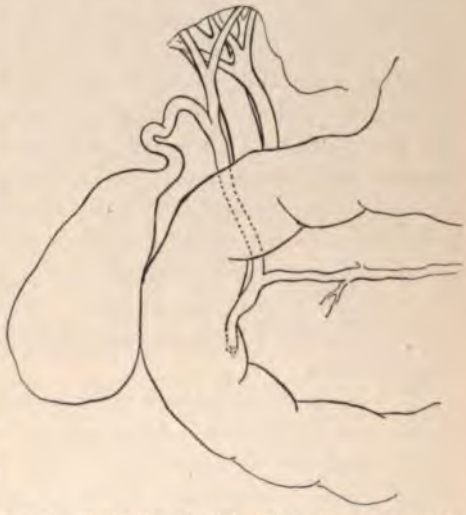


FIG. 48.—Illustrating the Relative Position of the Gall-Bladder, Ducts, Vessels, and Intestine.

he *cystic duct* is a tube welfth to one-eighth of ch (3 mm.) in diameter about one and one-half s (30–40 mm.) in length. nnects the gall-bladder the hepatic duct, and ined they form the com- duct. It is slightly sig- al in shape and curves upwards and backwards and to the right e first third of its course and downwards and to the left in the ining portion. It is situated in the lesser omentum, the hepatic being to the left and the portal vein behind. Its nerve and lar supply is even more extensive than that of the gall-bladder. e *hepatic duct* is formed in the transverse fissure by a branch each lobe of the liver. Its diameter is about one-sixth of an 4 mm.) and its length about two inches (50 mm.). Its branches ade up by the coalescence of the intrahepatic ducts which orig- in the interlobular spaces. These ducts are made up of con- e tissue and have a lining of short columnar epithelium resting basement membrane. The duct and vein have the same color re situated in close proximity. The former can be recognized ng situated above the artery while the vein is below. This is important anatomical point, as it assists the operator in differ- ng between the duct and the vein.

e *common bile duct*, ductus communis choledochus, is from one-

sixth to one-fourth of an inch in diameter (4–6 mm.) and nearly three inches in length (72 mm.). It conveys the bile from the union of the hepatic and cystic ducts into the duodenum. It passes downwards and backwards, continuing the course of the hepatic duct between the layers of the gastrohepatic omentum in front of the venæ portæ and to the right of the hepatic artery. Passing behind the first part of the duodenum, it reaches the descending portion and continues downwards on the inner and posterior aspect of that portion of the intestine covered by and often included in the head of the pancreas, and for a short distance in contact with the right side of the pancreatic duct. Together with that duct, it then perforates the muscular wall of the duodenum, and after running obliquely for three-quarters of an inch (18 mm.) between its coats and forming an elevation beneath the mucous membrane (ampulla of Vater), it becomes somewhat constricted, anastomoses with the pancreatic duct, and opens by a common orifice on the inner surface of the intestine near the junction of the second and third portions of the duodenum, three or four inches (75–100 mm.) below the pylorus. The distance from the fundus of the gall-bladder to the ostium duodenale is from six and one-half to eight inches.

The ducts are supplied with lymphatics that extend up along the portal vein to the interlobular spaces and empty into the lymphatic glands and the thoracic duct. The lymphatics that accompany the hepatic vein, particularly those on the upper surface, communicate with the thoracic lymphatics. In obstruction of the bile ducts these lymphatics carry the bile from the liver to the blood channels.

PHYSIOLOGY.

The hepatic and common ducts are tubes for the transmission of bile from the liver to the intestine. The cystic duct transmits the bile to and from the gall-bladder, and is joined to the hepatic duct at an acute angle. The gall-bladder is credited by physiologists with being the storehouse for bile. Landois states—and it is the consensus of opinion of physiologists up to the present time—that the liver is secreting continuously and part of the bile is stored in the gall-bladder and poured out copiously during digestion. Bile is excreted continuously, but the quantity excreted is increased during certain hours after the ingestion of food. Bile will be considered in this article as an excretion in accordance with the opinion of the majority of surgeons who have paid particular attention to this department of surgery. Physicians, as a rule, consider it a secretion. Experiments have convinced the author that the function of the gall-bladder

ıot that of a storehouse, and that its contents are not changed with h digestive act. The gall-bladder may be regarded as a controller ension of the bile circulation. An increase of the quantity of bile harged into the intestine takes place at two periods, namely, ween the third and fifth hours, and between the eleventh and thir- ıth hours after the ingestion of food.

The author has shown that the inherent contraction of the gall- lder, under normal conditions, expels on an average only thirty ims of bile even with stimulation of the neck of the gall-bladder the duodenal mucosa. When we consider the amount of bile eted daily (40 ounces, Murchison; 566 cc., Westphalen; 652 Ranke; 779 to 625 c.c., Coatman and Wilson), we can readily that if the small quantity which the gall-bladder is able to ex- (30 minims) were added to this large quantity, it would make ıppreciable difference either in the quantity or the current of entering the intestine at the time of digestion. The bile in gall-bladder consists mostly of mucus and is different in its ific gravity and consistence from that in the cystic and hepatic s, showing that there is an interchange between the cystic, com- , and hepatic ducts, *i.e.*, the varying tension of the bile circulation es the bile in the cystic duct to and fro, but does not move the bile ıe gall-bladder. In numerous cases after cholecystostomy, when gall-bladder had contracted to a narrow canal or sinus, the author rved that the bile was ejected intermittently every twenty or y seconds. In these cases the gall-bladder could have no pos- connection with the intermittency, contrary to the views of ɔn. From these facts the author has concluded that the liver tes increased quantities of bile periodically after ingestion of , and that the gall-bladder does not empty its contents intermit- y as stated by some physiologists.

AGENESIA.

cquired or congenital absence of the gall-bladder has become of interest since v. Langenbuch performed his first operation for its val in July, 1882. He proved in man what both Harlan and Cam- ıac had previously demonstrated on animals, namely, that the ladder is not necessary to life in mammalians. As a rule it is nt in carnivorous and omnivorous mammalia, but is absent in the rity of herbivorous mammals. The line cannot be closely drawn, ıembers of the same species, or even of the same family, may it uniformly present or uniformly absent. It was believed that in sence its place was supplied by dilatation of the ducts or ampulla.

The researches of Rex have shown that the dilatations or ampullæ may exist when the gall-bladder is present or are not formed in its absence, showing that they do not necessarily substitute each other. In my experiments of cholecystectomy in dogs, it was shown that a number of months after the extirpation of the gall-bladder the cystic duct became dilated to a miniature gall-bladder, attaining one-third to one-fourth the size of the original, the absence of the gall-bladder having no apparent effect upon the health of the animal. In nine out of thirteen cases of agenesia in adults reported by Courvoisier, there was a dilatation of some part of the duct. In six the hepatic, in one the two large branches of the hepatic duct, in another a number of the branches of the duct, and in one the ductus choledochus. He reports a similar dilatation of one or more of the large ducts in about the same proportion as above in cases in which there was occlusion of the cystic duct from pathological conditions, which leads to the conclusion that it may be compensatory and nature's effort to form a substitute for the gall-bladder. That the gall-bladder is not necessary to human existence or even to the enjoyment of good health, is shown by the number of cases recorded in the literature in which it was congenitally absent (Bergmann, Sandefort, Ziegler, Home, Eschler, and others), and in late years by the number of cases of cholecystectomy in which its removal has had no appreciable ill effect on the patient. Eschler recently reported a case of agenesia in a child two years old. There was an entire absence of the gall-bladder and of the fissure. No evidence was present that it had previously existed.

The inability to find a gall-bladder in an operation is not sufficient to designate the case as one of agenesia, as the gall-bladder may be buried in the substance of the liver and not show on the under surface (Wolfart). In these cases the cystic duct appears to come directly out of the liver (Vetter). Whether this condition was congenital or acquired it was impossible to determine in the case recorded. Vetter's case had two openings between the gall-bladder and the hepatic ducts. These were most likely pathological fistulæ. Of six other cases collected by Courvoisier, all were observed in children who died shortly after birth, and were accompanied by other agenesia of the hypoblast with one exception (Littré's case); in this the only agenesia was of the gall-bladder, it being noted that the ductus hepaticus was greatly enlarged.

Double gall-bladder has been recorded (Huber, Cruveilhier) in the form of two parallel gall-bladders divided by a septum, each having its separate duct (Rokitansky), or two distinct gall-bladders may exist with separate ducts. One may be within the liver and one

ithout, or they may be entirely separate and displaced. In cases of alformation the gall-bladder may be located in the normal position. horseshoe-shaped gall-bladder was reported by Schroer; it was tached to a floating liver. Diverticula resembling the gall-bladder,)parently originating from it or the cystic duct and containing cal-li, are not infrequently met with pathologically. These must not mistaken for double gall-bladders.

There is no recorded case of congenital absence of the ducts. ouble cystic duct has been found, one emptying into the ductus paticus, the other into the ductus choledochus. Double chole-)chus has been noted in which there were two insertions into the iodenum. Cases are recorded in which the choledochus emptied to the stomach. It is questionable in some of the cases whether ey were congenital canals or pathological fistulæ.

INFECTIONS AND ULCERATIONS.

Inflammations of the gall-bladder may be divided into three isses: (1) The catarrhal, chiefly traumatic, the result of irritation gall-stones; (2) the subacute or chronic, also most frequently ac-mpanied by gall-stones and mild degree of pus infection; and (3) ite infections of the gall-bladder with or without the presence of .culi. We shall now consider the last, as the first two will be taken in connection with cholelithiasis on account of their intimate asso-tion with it.

Acute Empyema of the Gall-Bladder.

Acute empyema or gangrene of the gall-bladder is associated with olelithiasis in seventy-four per cent. of the cases. In the remain-it occurs in zymotic diseases, as typhus fever, variola, etc.

Etiology and Pathology.

Whether the source of the infection in the majority of the cases is ough the portal circulation by way of the liver and down the bile nnels to the abraded gall-bladder, or by the arterial circulation ectly to the gall-bladder, or through the ductus choledochus and tticus from the intestines, is not easy to determine nor has it been isfactorily demonstrated. The infection may occur through any of these routes. It has been shown that living micro-organisms eliminated with the bile and in that way may be transmitted to gall-bladder, where under certain conditions they may find a fa-

vorable field for incubation, *i.e.*, a mucosa that has been abraded or ulcerated from contact with gall-stones. From a close analysis of the cases under my observation, it is my belief that the infection is received from the liver through the hepatic and cystic ducts. In the very acute variety there is at least an ulceration, and in some cases gangrene with a necrosis of a greater or lesser portion of the gall-bladder. The rapidity with which gangrene of the gall-bladder takes place depends upon the virulence of the infection and the tension at which the contents of the gall-bladder are retained. I have observed complete necrosis as early as seventy-two hours after the initial symptoms of infection. Four or five days is the most common period required for the destruction of the gall-bladder by gangrene.

In the early stage the gall-bladder is surrounded by adhesions to the adjoining viscera and omentum which endeavor to wall in the destructive process. These adhesions are life-saving to the patient, as they tend to prevent the extension of the inflammatory process. Later, when perforation takes place, the abscess may remain circumscribed by these protective adhesions after the gall-bladder has been destroyed. Pericystic abscesses occur without perforation by the transmission of the infective material through the diseased wall of the gall-bladder. The latter may remain circumscribed, or may produce a general suppurative peritonitis. The most rapid destruction of the gall-bladder takes place when pus is retained under pressure, as the circulation of the gall-bladder is impaired, its resistance is lessened by the pressure, and its rapid destruction is favored both by the biotic and toxic effects of the microphytes. The same pathological condition is observed in the appendix vermiformis under similar conditions. The quantity of pus in empyema of the gall-bladder is usually small, not exceeding a few ounces. When the process has existed for a long time, it may reach an enormous quantity, even several pints. The fluid of the empyema or abscess may be sanious or odorless, or pus of a thick creamy character, but it is most frequently, particularly in the very acute variety, exceedingly offensive, having the same odor as that generally present in appendical abscesses and attributed to the bacillus coli communis. It is thick, brown, contains some bile, and the débris of the gangrenous wall of the gall-bladder. The author has been unable to find reliable statistics concerning the micro-organisms commonly found in these abscesses.

Symptoms.

The history of previous attacks of colic accompanied by vomiting, and the voluntary information from the patient that during and immediately after these attacks there was considerable sensitiveness

beneath the costal arch in the right hypochondriac region or the epigastrium are of value in the diagnosis. The history of nic gastric disturbance with what the patient calls "frequent at- s of gastralgia" also leads to the belief that the patient had red from cystic-duct obstruction. If the patient is able to state the attacks were accompanied by an elevation of tempera- (99.5° to 101°), it is an additional evidence that he had suffered cystic-duct obstruction with a mild pus infection. The sudden t of severe pain, nausea, and vomiting, often accompanied by a of temperature, with great sensitiveness beneath the margin of right ninth costal cartilage and the absence of jaundice, are er evidence that the patient is suffering from cystic-duct ob- tion. In the acute cases in which gangrene of the gall-bladder esent or imminent, the temperature will be above 101°; the ute variety frequently presents a temperature of 101° but rarely r. When gangrene takes place the temperature ranges from to 104.5°. The pain in the early stage before necrosis and re of the gall-bladder have taken place is very severe, often col- when the duct is obstructed. As necrosis advances the pain es less, and when rupture takes place the temperature falls and e same time the sensitiveness and induration increase. From ginning the patient shows all the manifestations of a profound ; dry, coated tongue, mild delirium, dry skin, great thirst, a, vomiting, and frequently hiccough. When rupture takes and the tension of the pus is reduced the manifestations of diminish.

Physical Signs.

ese are great sensitiveness beneath the right costal arch, in- d upon deep inspiration; a circumscribed induration, some- smooth and elliptical in shape, again irregular and nodular, moves synchronously with the liver in the respiratory act; lness on percussion. The absence of peristalsis in the neigh- d of the tumor, inability to produce bowel movements, and symptoms of adynamic ileus are sometimes observed. The the induration often bears no relation to the size of the gall- r nor to the size of the abscess, but is indicative of the extent egree of the infiltration and adhesions. The border of the haped organ when free from adhesions describes an arc, the of which is the ninth costal cartilage. It can be swung farther left than a movable kidney; it not infrequently can be felt pro- from the margin of the liver, and the kidney can be outlined with one hand, while the gall-bladder may be grasped with

the other. It may be differentiated from a tongue-shaped extension of the right lobe by its smooth surface, its fluctuation, and by the absence of the sharp ridge that is present on the elongated lobe. It frequently accompanies the linguiform enlargement, and the margin of the liver may be distinctly outlined leading to the gall-bladder, to which it is usually firmly adherent. Jaundice is rarely present, i.e., gangrene of the gall-bladder does not occur with occlusion of the common duct, but with occlusion of the cystic duct.

Differential Diagnosis.

It is difficult, if not impossible, to differentiate between a gangrenous appendicitis when the appendix is located on the under surface of the liver and a gangrene of the gall-bladder. The writer has seen the appendix adherent to the surface of a gangrenous gall-bladder and *vice versa*. Perforation of the stomach or duodenum is accompanied by greater depression in the early stages and less pronounced septic manifestations. Rupture of hepatic abscess or of suppurating echinococcus cysts is also difficult of differentiation. Rupture is accompanied by symptoms of depression and collapse rather than by the symptoms of septic intoxication. Rupture of a non-infected echinococcus cyst is usually accompanied by urticaria and the symptoms of diffuse peritoneal involvement with depression instead of elevation of temperature. In pyonephrosis and perinephritic abscess the induration and sensitiveness are most marked in the loin and are accompanied by only a moderate elevation of temperature. The induration does not extend so high in the epigastrium and can usually be separated from the margin of the ribs.

Prognosis.

All of the cases collected by Courvoisier up to 1890 died between the third and fifth day. Such, however, has not been my experience. All of the cases operated on before the third day recovered. One operated on as late as the fifth day recovered. One operated on the fifth day died. From these facts it can be seen what a rapidly fatal disease it is.

Treatment.

The medicinal treatment of the disease may be summed up in a single sentence: Do not administer an opiate; it obscures the symptoms, renders the diagnosis more difficult, and contributes nothing whatever to the relief or cure of the disease or to the preservation of

life of the patient. This is a surgical disease from the onset, prompt surgical treatment is absolutely indicated. Perforation he gall-bladder from infection may take place within seventy-two rs from the onset, but peritonitis produced by infection through wall of the gall-bladder is usually limited up to that time. In a ase like this in which the diagnosis can be made with such ac- ıcy and the results of medicinal treatment are so fatal, there is xcuse for delay.

Operation.—An incision three inches long is made from the ninth al cartilage parallel to the outer wall of the rectus over the high- oint of induration. The peritoneum is opened, the adhesions are fully examined, and the induration is packed around with sterilized ze to prevent the pus from escaping over the healthy peritoneum n the abscess or bladder is opened. The adhesions are then ly separated along the under surface of the liver until the abscess ıll-bladder is reached. If the gall-bladder is still intact and gan- ous, an incision should be made in the wall an inch to an inch one-half in length and a glass drain of the same diameter in- d. No effort should be made to remove a gangrenous gall-blad- ıor to explore its cavity. The drainage tube or glass speculum, h is best for this purpose, retains an opening that is large gh to admit subsequent exploration without the danger of lacer- the adhesions, and if the gall-bladder be gangrenous it will ate and the slough may be readily removed. The object of the ary operation is the relief of the tension of the retained pus and s. The removal of the stones from the gangrenous gall-bladder be postponed until the active sepsis has subsided, at which time perator is master of the situation. All that is necessary is to ge out the pus and to make effective drainage; the cavity should e irrigated. When the gall-bladder is ruptured or there is a ystic abscess, the possibility of an infection of the lesser peri- l cavity through the foramen of Winslow must be borne in , and if the symptoms of sepsis continue after drainage in this of cases, the foramen should be enlarged, the cavity examined, ıe lesser peritoneal cavity drained by a tube drawn out through in over the apex of the right kidney.

rs. M., age 46, enjoyed excellent health all her life, never hav- ad attacks of colic or jaundice. She was large and very obese, ing two hundred and sixty pounds. On the evening of October 1895, she had some distress after eating a hearty meal. The fol- g morning at ten o'clock, she was attacked with severe pain in ght hypochondrium, followed by nausea and vomiting. The ncreased in severity, the nausea was persistent, and the tender- xtreme; there was no jaundice. Large doses of morphine did

not stop the pain. On the evening of the 28th, the pulse was 90, temperature 102°. On the morning of the 29th the pulse was 115, temperature 103°. The area of sensitiveness was increased; the abdomen was tympanitic; there was inability to produce bowel movements. Patient was seen in consultation at four o'clock on the afternoon of the 29th. Manifestations of profound infection were present; the tongue was dry and coated; pulse 122, temperature 104.4°; respiration increased and entirely thoracic.

Examination.—Heart and lungs normal; no icterus. Abdomen distended, tympanitic, very sensitive under the right costal arch, where marked induration could be detected; peristalsis absent; urine negative.

Diagnosis.—Acute cholecystic infection with duct obstruction.

Operation.—October 30th (morning), patient slightly delirious and greatly depressed, pulse 130, temperature 103.2°; tongue very dry. The usual abdominal incision was made for a cholecystic operation. The gall-bladder was very much enlarged, surrounded by omentum and intestine, and slightly adherent. When incised it did not bleed. All adhesions were separated from it and a cofferdam of gauze was built around the gall-bladder to the neck. Nine ounces of pus and bile were aspirated to relieve the tension. The opening was closed with a ligature. The wall of the gall-bladder was gangrenous. It was not opened until November 2d; when incised it showed all of its coats necrotic. A large quantity of pus escaped and a three-fourths of an inch glass drain was inserted. There was no bile; the necrotic gall-bladder was separated and escaped through the opening. After two weeks a calculus could be detected in the cystic duct by aid of the sound. No effort was made to extract it. Four weeks after the primary operation a pure cholesterin calculus, five-eighths of an inch in diameter, escaped. This was followed by a free discharge of bile. The fistula closed in six weeks, and the patient recovered.

This case illustrates how quickly the gall-bladder may be destroyed when infection products are retained under pressure.

Subacute or Chronic Empyema of the Gall-Bladder.

In empyema of the gall-bladder that organ is full of pus, with perhaps mucus and bile, but the pus predominates. The inflammation is usually subacute in character, contrasting markedly with the condition described in the last section. Cholelithiasis must be considered as the greatest etiological factor, as out of seventy-four cases of chronic empyema of the gall-bladder, including those published by Courvoisier, fifty-eight were accompanied by calculi. In approximately seventy-one per cent. of these the stones were located in the gall-bladder or in the gall-bladder and cystic duct. In only 2.7 per cent. of the cases were the stones confined to the ductus choledochus. As other etiological factors we have the infectious fevers, which favor a low type of pus infection, and we also have empyema accom-

ying suppurations of the gall-tracts not associated with calculi. e entozoa finding entrance into the gall-bladder may also produce pyema. The pathological condition of the gall-bladder differs maally with the cause and duration of the inflammatory process. mucosa is not infrequently ulcerated and abraded with submus abscesses and pustules. If the cysticus be obstructed by a large ie, the rule is that the gall-bladder is dilated, its wall is thickened, the pus contains no bile but considerable mucus, and not infrentlv the contents are a clear liquid. The gall-bladder may attain reat size under these circumstances, holding even two quarts rck). In only twelve per cent. of the recorded cases was the gallder contracted. Adhesions take place to neighboring viscera. In nic empyema of the gall-bladder pericystic abscesses occur withperforation, but they are more commonly associated with pertion and not infrequently the abscesses contain calculi that have ped from the gall-bladder.

Symptoms.

hronic empyema of the gall-bladder is usually ushered in with a cystic colic, followed by slight inflammatory reaction and a sense ight and pain in the right hypochondrium. The patient is naud and not infrequently vomits. After the emesis the severe pain ides. The temperature rarely exceeds 101°. There is marked erness in the region of the gall-bladder, which is increased on deep ration; tumor or induration may be detected, depending upon ize of the gall-bladder and the presence of pericystic adhesions. two or three days these symptoms entirely subside and the nt enjoys comparatively good health until another exacerbation place. The patient frequently gives a history of having had tack of the above character every three to six weeks over a period nths or even years, and usually attributes it to some indiscretion et. Sooner or later the case must have surgical treatment. Ocnally agglutination to the abdominal viscera takes place and the bladder is drained through the intestines. In others, the adhe- to the abdominal wall form and there is phlegmonous suppuration ting in a perforation with external opening. The pus or stone escape or be extracted through this opening. Rupture into the minal cavity with suppurative peritonitis also occurs. As a rule, istory of these cases extends over a number of months or years.

Physical Signs.

The gall-bladder can usually be outlined as a smooth, pear-shaped body extending down from the margin of the ribs. The kidney can be detected behind separate and movable. As the gall-bladder enlarges it frequently carries down with it the right lobe of the liver. When the abdominal wall is thin, the sharp edge of the liver can be detected passing transversely across the tumor, and the edge of the liver can be traced upwards and forwards, downwards and backwards from its adhesion to the gall-bladder. This is pathognomonic of enlargement of the gall-bladder. These patients should be examined during an attack, as the tension and sensitiveness of the gall-bladder are greatest at that time and the tumor can be more readily palpated. It can be moved from right to left for a considerable distance, and swings from the costal arch as its centre.

Differential Diagnosis.

The condition may be differentiated from abscess of the liver by its elliptical shape, its mobility, and the abruptness with which it protrudes from the lower margin of the liver. The same characteristics distinguish it from suppurating echinococcus cysts. In hydro- and pyonephrosis, the tumor is situated farther to the side, can be felt more distinctly behind, and is not so globular in shape. It cannot be displaced so far to the left. Movable kidney may be confused with empyema of the gall-bladder. The shape of the kidney and the ability to replace it in the right loin are points of value in the differential diagnosis, and further, while holding the kidney in one hand, the gall-bladder with the other may be felt to move up and down with the respiratory act. The tumor should never be punctured with an exploring needle to make a differential diagnosis, as the life of the patient would be immediately jeopardized by a leakage from the abscess. This dangerous procedure is very often practised at the present time, but should be condemned. Exploratory laparotomy is much safer than puncture. Furthermore, puncture does not aid much in the differential diagnosis, as there is nothing pathognomonic about the pus in empyema of the gall-bladder.

Treatment.

Medicinal Treatment.—The physician is most frequently called on to treat the patient during the exacerbation. In the interval between these attacks the patient suffers very little except from reflex gastric

urbances. During the attack local applications of hot fomenta-
s, brisk cathartics, and moderate-sized doses of phosphate of so-
n may be administered. After the attack, good results have been
ined by large doses of phosphate of sodium and by water cures,
icularly at Carlsbad. The diet should be regulated, as the patient
lly attributes the exacerbation to some change of diet, and many
s the attacks are associated with a particular article of food.
osure to cold also frequently precipitates an attack.

Surgical Treatment.—This must usually be resorted to for perma-
relief. Operations for chronic empyema of the gall-bladder are
led into three classes: (1) Cholecystostomy in two sittings;
cholecystostomy in one sitting; and (3) cholecystectomy.
n the gall-bladder is only moderately enlarged and adhesions
not present, a cholecystostomy in two sittings is always indi-
. A cholecystostomy in one sitting should be confined to cases
ich union between the gall-bladder and abdominal wall exists
e time of the operation, and the distending suppurating gall-
ler can be drained without disturbing the free peritoneal cavity.
gall-bladder is greatly elongated it may be dislocated into the
minal incision, the peritoneal cavity protected at its neck by
e and packing, and a cholecystostomy in one sitting performed.
e end of eight or ten days, after adhesions have taken place,
ll-bladder may be amputated down close to the peritoneum and
age kept up until the suppuration subsides, when the fistula is
d to close. When the gall-bladder is greatly elongated and free
adhesions, a primary cholecystectomy may be performed and
pening closed with a Czerny-Lembert suture. This operation
l always be performed when the cystic duct is permanently
led and the amputation should be made at the point of occlu-
If a previous cholecystostomy has been performed and a sinus
s, the indication is to do a secondary operation and amputate
ll-bladder at the point of occlusion of the cystic duct. When
dominal cavity is opened and the gall-bladder is found to be
ously enlarged and free from adhesions, in order to assist its
ion through a small incision aspiration may be performed,
ing taken not to allow the contents to come in contact with the
or hands of the operator. In all cases in which there is danger
escape of pus during the operation, a cofferdam of gauze
be placed around the field of operation. After prolonged
ge the gall-bladder contracts to a sinus, and if the cystic duct
ulous and the edge of the gall-bladder is below the surface of
n, the opening will close. If the cystic duct be permanently
d by either a calculus or cicatricial contraction, a permanent

fistula will remain until the gall-bladder is extirpated or the obstruction is removed. A thorough examination of the gall-bladder mucosa, the dilated cystic duct, and the cause of the obstruction may be made with the aid of the cystoscope which the author has had constructed for this purpose. The cystoscope consists of tubes varying in diameter from 5 to 20 mm. and in length, including stilet, from 85 to 105 mm. (see Fig. 49).

The results of the operation for chronic empyema of the gall-bladder are very favorable, the best being from the operation performed at two sittings. The one-sitting operation should be performed only when there is some special indication for the immediate opening of the pus cavity. A cholecystectomy should be reserved for a secondary operation, except when the neck of the gall-bladder is greatly elongated and the wall is free from adhesions, or when the cystic duct is permanently occluded.

FIG. 49.—The Cholecystoscope.

INFECTION OF THE BILE DUCTS.

ETIOLOGY.

This pathological condition is to be considered separately from abscess of the liver from all causes, and should be confined to primary suppurations of the gall-ducts themselves, due to the passage of septic material from the intestine up the gall-channels, or to infections of the gall channels following the passage of gall-stones, echinococcus cysts, ascarides, or other foreign bodies. In 74 cases Courvoisier found that 77 per cent. were due directly or indirectly to the passage of gall-stones and 23 per cent. to echinococci, intestinal worms, and carcinomata. In 42 per cent. of the cases the gall-stones were found in the hepaticus and choledochus; in 34 per cent. in the gall-bladder as well as in the hepaticus and choledochus, and the remainder in the gall-bladder and cysticus. This shows that the cause of the suppuration is most frequently due to impaction in the hepaticus and choledochus, and it appears that such an obstruction greatly favors inoculation and extension of pus infection. The Eberth bacillus was found in a post-mortem examination by Girode. Gilbert and Dominici produced suppurative cholangitis in animals by injecting pure cultures of Eberth's bacillus into the common duct. While the bacillus coli communis (Escherich's bacillus) is the common parasite

the biliary tract, it is pathogenic only under certain circumstances. intz reports a case of cholecystitis with the formation of gas in the er in which pure cultures of this bacillus were found. D. Alloco orted a case of cholecystitis and angiocholitis produced by pure tures of the bacillus coli communis. Statistics of these cases do show in any great number whether the infectious agents were phylococcus or streptococcus pyogenes or bacillus coli communis; three have been found separately and associated.

The pathological changes produced by the suppuration are swell-, injection, and œdema of the mucosa. If the intensity of the in-mmatory process is great the canals will be completely occluded, not infrequently a pericholangitis occurs. Ulceration and per-ation from such an infection occur most frequently around the ained calculus. The suppurative process may extend up the gall-ts into the liver, and a small or large abscess may be formed in it. abscess in the liver may contain gall-stones or may have direct mmunication with the suppurating duct. In these cases it is not ommon to have multiple foci of suppuration in the liver. These due not alone to the passage of pus along the ducts but to a sup-ative endarteritis or thrombosis of the hepatic artery. The pas-e of ascarides favors the advancement of suppuration along the t. Carcinoma opening into the tract or primary carcinoma of the ts also favors suppuration, and particularly carcinoma of the head he pancreas. Cases of suppuration following severe zymotic dis-s have been recorded.

Symptoms.

The symptoms of cholangitis are so closely allied with those of -stone impaction, and so frequently associated with the presence gall-stones, that it is difficult to differentiate between the two. re is present the evidence of obstruction of the choledochus or aticus together with chills and fever. The chills and fever are ost always preceded by colic. Shortly after the colic jaundice ifests itself. The pains or colic disappear but the jaundice con-es. As this disease is confined to the hepatic and common s, jaundice will be present as long as the obstruction from ma of the mucous lining exists. It must not be concluded that use the colic has ceased the calculus has been expelled into ntestine. More frequently it is retained in a diverticulum or in ampulla of Vater and continues as a cause of ulceration and ruction in the duct. Jaundice with an intermittent fever must r be interpreted as malaria. It is always infective and most fre-tly produced by septic cholangitis with impacted calculi. In

suppurative cholangitis without cholelithiasis the patient is depressed and intensely and continuously sick, and the entire hepatic area is sensitive to percussion and palpation. There is no intermission of fever. In the case of hepatic abscess there is a marked remission if not a perfect intermission. When high temperature and chills are due neither to abscess nor to infective angiocholitis, but to stone in the common duct, the jaundice is continuous, the chills are recurrent, the fever is intermittent, and in the interval between the attacks the patient is comparatively well.

Treatment.

The symptoms of sepsis are produced by a retention of the septic products of the cholangitis intermingled with bile, and the indication is therefore clear that the gall-tracts should be flushed or drained. The so-called cholagogues, calomel, podophyllin, etc., have been shown to be impotent to increase the quantity of bile. Large draughts of carbonated water should be administered as that greatly increases the secretion from the liver even if it does not increase the quantity of solids eliminated. The pain may be allayed with small doses of phosphate of codeine. The application of fomentations over the liver has a salutary effect. When the stone is retained in the common duct a cholecystostomy is indicated. After the inflammation has subsided, if necessary, a choledochotomy for the removal of the stone may be performed. It should not be performed as a primary operation, for patients suffering from cholæmia accompanied with sepsis do not withstand severe operations and usually die from sepsis and cholæmia by the fourth day. When the stone is in the hepatic duct, if it can be located, the duct should be incised and the stone removed, after the field of operation has been thoroughly protected with a gauze packing.

The *prognosis* in this class of cases is very grave. The great majority die before the end of the fifth day (Osler).

GALL-STONES.

Chemical Composition.

Gall-stones are composed chiefly of cholesterin and bilirubin. Next in frequency is found carbonate of calcium (Naunyn). Associated with bilirubin are biliverdin, bilicyanin, and bilifuchsin. Copper is always associated with bilirubin, apparently as the bilirubinate of copper, and iron is also intermingled, but in small quantities, rarely equalling one-tenth of the concrement. The protein forms

ıy contain the phosphates or sulphates of calcium; even mercury s been found as a nucleus.

Physical Characters.

Gall-stones may be divided into (1) laminated cholesterin, (2) re cholesterin, (3) common gall-stones, (4) mixed bilirubin, (5) e bilirubin, and (6) protein (Naunyn).

The *laminated cholesterin stones* are usually smooth on the surface l of varying colors, white, green, yellow, black, brown, and com- ations of these shades. They are made up of layers from one- th to one line in thickness, are friable and often crumble when . They are faceted. When incised the outer wall is usually crystalline, while the centre has a marked radiating crystalline earance and is usually composed of pure cholesterin.

The *pure cholesterin stones* are oval or egg-shaped, seldom faceted. y are rarely multiple. The size ranges from that of a cherry to of a pigeon's egg. They are warty on the surface, rarely smooth. irregularities are due to crystal formation on the surface. They transparent or semitransparent with a slight yellow tinge; many ain a nucleus of a dark brown color. When they are fractured show beautiful radiating crystals constructed from the centre. y are not made up of layers. Of these two varieties the second ists almost exclusively of pure cholesterin, while the first contains ty per cent. of cholesterin. The more the crystalline structure ominates the greater is the quantity of cholesterin, but even the matic layers contain a large percentage of that salt intermingled bilirubin or biliverdin. The green layers occasionally met with ich in carbonate of calcium.

The Common Gall-Stone.—In this class is included the majority of stones. They are of different sizes, shapes, and colors on the ce, are mostly faceted, not alone from friction but from moulding ıe early stage of their development when they are soft. The ominating color is yellow, next in frequency brown, less commonly , and but rarely green. They seldom attain the size of a cherry, ; more frequently about as large as a pea. The small ones when ly extracted from the gall-bladder are soft and very friable; they even resemble putty in consistence. When dried they become r and shrink considerably. When fractured they are found composed of layers of varying density and color. The centre still be soft and cheesy, but occasionally it is as dense as the hery. This is the class of stones that were supposed to have passed after the administration of olive oil, but a careful nation of them showed that they were not gall-stones but undi-

gested oil. Not infrequently some of the stones contain an alkaline fluid in the centre surrounded by a thin capsule. To the unaided eye the common gall-stone never has a crystalline appearance.

The Mixed Bilirubin.—They are usually of cherry size, never large, only two or three are found in the gall-bladder or in the ducts. When more than one is present they are faceted. They are composed of thick layers of a dark brown or reddish color. They contract when dried and not infrequently fracture, the outer shell peeling off. Towards the centre they are crystalline and often the nucleus is composed of pure cholesterin. Twenty-five per cent. of the outer layer consists of cholesterin, the remainder being principally bilirubin and the bilirubinate of copper, with a small trace of iron.

The *pure bilirubin stones* contrast markedly with the latter. They are never large, often as fine as sand, rarely attaining the size of a pea. They may be divided into two classes. The one is soft, rough on the surface, mulberry-like, crushes readily under the finger, and when dried shrinks materially or even crumbles. The other, of the same size as the former, is of very different form, irregular on the surface, or sometimes smooth; it is of a steel-gray or dark brown color; it is hard, but the larger ones are never solid; it consists almost exclusively of the calcium salt of bilirubin with a little biliverdin and very little cholesterin. In fifty per cent. of the cases the stones vary in size from that of a pea to that of a bean; in twenty-two per cent. they are as large as hazelnuts. In a small percentage of the cases they are from the size of a grain of sand to that of a grain of wheat, moulded on the edges to adapt themselves to the shape of other stones. In twelve per cent. they are as large as pigeon's eggs. One has been reported which was 7 cm. (2¾ in.) in length and 10 cm. (4 in.) in circumference. They are not infrequently moulded to the shape of the gall-bladder.

The *protein forms* consist of amorphous substances and incompletely crystallized cholesterin salts. They are rarely laminated, and the naked eye cannot detect their crystalline structure. The centre differs materially from the rest of the stone, being a foreign substance surrounded by almost pure cholesterin. Pure carbonate of calcium stones of large size have been reported (Frerichs). They are of varying colors and can be recognized by their hardness. Conglomerate stones of different colors and consistence are not infrequently found, the nucleus consisting of one salt surrounded by various layers of individual or combined salts. Foreign bodies, such as a tapeworm, a distoma, a needle, and a lime stone, have been reported as centres for these concrements (Lobstein, Bouisson, Nancke, Frerichs).

The common gall-stones, the mixed cholesterin, are found only the gall-bladder. Numerically they reach into the thousands. aunyn observed a case in which there were five thousand. The ry large stones which are found in the gall-bladder may be one to ree inches in length and one to one and one-half inches in diam- er. Whether they consist of cholesterin or of bilirubinate of cal- um is not yet determined. They are found in the gall-bladder, the ctus cysticus, and ductus choledochus. To the latter positions ey are probably forced from the gall-bladder. They are faceted. hen multiple they assume the shape of the cavity in which they are nd. Occasionally they are adherent to the mucosa, *i.e.*, granula- ns from the mucosa have burrowed into the meshes of the stone the stone has formed about them. Pure bilirubin salts have been nd in the gall-bladder, in the mucosa of the gall-bladder, and in intrahepatic bile channels.

The Physiology of the Formation of Stone.

The presence of cholesterin and lime in the bile is entirely inde- ndent of metabolism and of the character of the food taken. They ginate from the mucosa of the gall-tracts (Naunyn). Cholesterin present in numerous forms of pathological tissue metamorphosis l it is possible that its presence in the bile is the result of elimi- ion from the blood. It has been found that when a large quantity cholesterin is administered to rabbits it does not materially in- ase the quantity present in the bile (Jankau, Thomas). Inves- tions of the amount eliminated in various diseases were made by usch and no material increase was noted except when a calculus present in the gall-bladder. In these cases the bile was very rich holesterin. This is of great etiological importance, as it shows that presence of the calculus itself tends to produce an increased quan- of cholesterin, and that cholesterin is not a product of metabolism not a specific product of the liver. That crystallization of cho- erin should be increased by the presence of crystals already ned is well understood in the chemico-physics of crystallization particularly of that of hydrocarbons. It was found thatthe exudate n the diseased mucosa of the gall-bladder uniformly contained lesterin even though there was entire absence of bile in the gall- lder. Robbins found that seven per cent. of the solids of pus con- d of cholesterin. It is probable that the action of the bile on the liated epithelium of the mucosa of the gall-tract favors the for- ion of cholesterin. When the mucosa is diseased the exfoliation creased and also the cholesterin formation.

The excretion of calcium salts by the bile is not increased by the administration of large quantities of calcium in the food or in solution in drinks (Jankau). There is present a small quantity of lime salts in the bile even in the smaller gall-tracts. It is probable that part of this is produced from the mucin secreted in the gall-tract, as mucus always contains calcium. It has been demonstrated, however, that the bile from a fistula of the gall-bladder contains more of the salts of lime than the bile from the smaller tracts of the same individual.

The solubility of the various salts and combinations which are found in gall-stones was investigated by Happel. Cholesterin and bilirubin salts are not soluble in water. Cholesterin is held in solution in the bile by soap, fat, and alkaline glycocholates. Cholesterin is soluble in one-fourth to two and one-half per cent. of glycocholic acid and taurocholate of sodium at a temperature of 37° C. In twenty-four hours there will be dissolved in this solution one-tenth of the weight of cholesterin. A 2.5-per-cent. solution of soap under the same circumstances will dissolve 1.25 per cent. of cholesterin. Olein will dissolve five per cent. of its weight. Kausch concludes that the tenth part of the cholesterin in the bile is dissolved by the alkaline glycocholates. In addition to that there are present fat and soap. These also dissolve the cholesterin and compose one-tenth of the bile. The decomposition of glycocholic acid in the bile does not precipitate the cholesterin nor does the presence of decomposition appear to favor its crystallization, at least not at the body temperature. The presence of carbonate of calcium, lime water, or carbonic dioxide in small quantities lessens the tendency to the precipitation of bilirubin. Lime water in excess favors the precipitation, and if to the lime water there are added other alkalies and glycocholates the precipitation is again lessened.

The most interesting point in the formation of gall-stones is the origin of the nucleus or the crystalline centre around which the stone grows. There is ever present in the bile of the gall-bladder a greater or lesser number of epithelial cells. These are single or arranged in clumps and have undergone or are undergoing fatty or hyaline degeneration. When examined with the microscope these nuclei can be recognized as structureless white pearls within or escaping from the cells. These pearls consist of cholesterin around which gall-stones are formed. After a time the cholesterin crystallizes and side by side with the pearls may be seen other small masses with a brown, soft centre (bilirubinates), the centre being surrounded by cholesterin. Naunyn says that bile salts are protoplasmic poisons and under certain favorable circumstances produce cholesterin from the exfoliated epithe-

ım of the bile channels. It is a recognized clinical fact that depos- of bile salts in the tissues lessen the regenerative power of the lls. The second method of formation is recognized when brown d fatty flocculi, consisting of fat and cholesterin crystals which are frequently present in the gall-bladder, form centres for the forma- on of stones. Their chemical composition varies greatly. They nsist of fat, cholesterin, glycocholates, bilirubin, etc. These soft ımps may enlarge as such and be numerous enough to fill the entire ll-bladder (Naunyn). Again when concentration of the solid con- tuents takes place they may form a shell around the fluid part which mains in the centre, and thus is formed the hollow, friable stone ıich is not infrequently met with. When small agglutinations are dergoing crystallization, they form what is known as the mulberry ne. Stones are rarely formed by an agglutination of individual olesterin crystals.

Micro-organisms were found in calculi, particularly those of re- ıt formation by Gilbert and Dominici, and were credited by them h playing an important rôle in the causation of the concretions. eir experiments to produce calculi by injection of pure cultures ved a failure. The further development of these stones takes ce by two processes, namely, an addition of layers on the surface l the infiltration and crystallization of the salts of cholesterin hin. The layers are made up of pure cholesterin or combina- ns of cholesterin with bilirubin, biliverdin, etc., the surface be- ning hard and dense while the centre may remain soft and hollow, ıtaining a few drops of clear liquid. The crystallization of the ıline amorphous cholesterin substance advances towards the sur- e. Cholesterin is carried from without to the centre of the stone infiltration—a well recognized process in mineralogy. The bili- in is usually dissolved and removed as the process of crystalliza- ı goes on. The cholesterin is contributed by the epithelium of gall-tracts and is not derived from the bile itself. These forma- ıs grow even when the cystic duct is entirely obliterated and when re is no bile in the gall-bladder. All gall-stones are formed in gall-bladder originally, with the exception of the bilirubinate of ium stones, which are sometimes formed in the intrahepatic chan- s. The larger stones which are met with in the large gall-tracts probably carried there from the gall-bladder where they origi- ed, and may increase in size both as bilirubinates and as choles- n stones; but cholesterin stones of large size have been found edded in the wall of the large ducts or in diverticula of the ducts, were possibly developed in these positions.

The formation of gall-stones in the liver, that is, in the smal-

ler gall-tracts of the liver, takes place from the retention of bile and the precipitation of salts. They consist of bilirubin combinations. It is possible that the precipitation is favored by the presence of schizomycetes which are readily developed in the bile when it is stagnated.

The frequency with which gall-stones occur and the age in which they are most prevalent is best shown by Schroeder's statistics, which I here quote:

Age.	Number of post mortems.	Gall-stones.	Percentage of gall-stone cases.
0 to 20	82	2	2.4
21 to 30	188	6	3.2
31 to 40	209	24	11.5
41 to 50	252	28	11.1
51 to 60	161	16	9.9
Above 60	258	65	25.2

In men, 4.4 per cent. In women, 20.6 per cent.

As these statistics were compiled from the post-mortem records of a hospital which admitted patients of all ages, a very careful analysis of the cases in this particular was made. The statistics show that gall-stones seldom occur before the age of thirty, are more frequent from thirty to sixty, and occur very frequently after that age. They occur almost five times as often in women as in men, and in married women ten times as often as in single women. The mode of dress and results of childbirth are probably among the predisposing causes. Schroeder found that fifty per cent. of women suffering from corset liver had gall-stones. The tongue-shaped enlargement of the right lobe of the liver associated with cholelithiasis only occurs as a sequence of cholecystitis when the gall-bladder is elongated and brings down with it the adherent lobe of the liver. This tongue-shaped projection or hourglass contraction is very prevalent in countries where tight lacing is kept up from childhood.

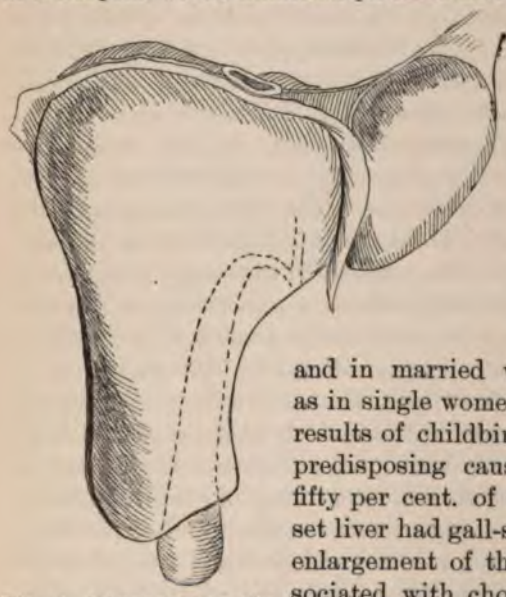


FIG. 50.—Illustrating the Tongue-Shaped Enlargement of a Right Lobe of the Liver and the Relative Position of the Enlarged Gall-Bladder in Cholelithiasis.

ht lacing and the pendulous abdomen following pregnancy mili- against the free exit of bile from the gall-bladder and favor the nation of stone. The great frequency of stone in the aged is bably accounted for by the excessive exfoliation of mucous helium, and thereby the increased quantity of cholesterin in gall-bladder accompanied by atony or loss of elasticity of the -bladder itself (Kausch). The author does not believe it is due n atony of the muscle fibres as stated by Naunyn, as the mus- fibres play a very minor rôle in the expulsion of bile. Fre- s believes that occupations requiring patients to sit many hours he same position favor the formation of gall-stones. That there a hereditary predisposition to the disease cannot be demonstrated, ough many observers have noticed that it has existed for three erations; the author has observed that in one family. Diathesis emperament appears to have little influence in the production of stones.

t is apparent that retention of bile in the gall-bladder favors the ation of stones, and as stones are not formed by precipitation of solid constituents of the salts in the bile, some other factors must e into play. Either the stagnant bile has a deleterious effect on mucosa or it favors the growth of microphytes. Bile in its nor- condition in healthy tracts is sterile. In bile artificially stagnated gature of the ducts micro-organisms such as staphylococci, strep- ci, and the bacillus coli communis, frequently develop (Charcot). e and many other micro-organisms may be eliminated from the l through the bile, but it is believed they play a small rôle in tiology of cholelithiasis, while they are credited with producing cystitis not infrequently. Retention of bile in the intrahepatic s, cicatricial formation, or cirrhosis of the liver, favors the forma- of bilirubin stones, and these may be carried through to the gall- ler and form the nucleus of cholesterin stones. The mucosa of gall-bladder has at times a villous appearance and on the tips of villi may be found deposits of crystals which form the nucleus stones (Mettenheimer). The foreign bodies that enter most ently into the production of gall-stones are found in the gall- ler. When one gall-stone has been formed, the tendency is to the uction of many others from the pathological changes produced in mucosa by the presence of the original calculus in the gall-blad- Circulatory disturbance or venous hyperæmia appears also to the formation of calculi.

Calculi in the Gall-Bladder.

Post-mortem statistics show that gall-stones are present in about one-tenth of the cases examined. Bollinger found gall-stones in 5.4 per cent. of all cadavera and two and one-half times more in women than in men. In Denmark Paulsen found that only 3.78 per cent. of bodies examined post mortem had gall-stones. This percentage is very much below the average for southern countries. An examination into the history of these cases shows that they produced symptoms that were recognized in only a very small percentage of the number. This is due to two causes: First, the gall-stones did not produce gross pathological conditions or symptoms sufficiently marked to permit of a diagnosis; second, the profession was not generally informed on the symptomatology of gall-stones in the gall-bladder. The changes produced in the gall-bladder by the presence of stones are many, depending upon the number and size of the stones, the comparative size of the gall-bladder and the stones, whether they are impacted in the cystic duct, and whether they are accompanied by infection; that is to say, they are not recognized in the gall-bladder by their presence *per se*, but by the pathological changes which they induce. The presence of stones in the gall-bladder produces a traumatic or catarrhal type of inflammation rarely reaching to suppuration. This is recognized by the hypertrophy of the wall of the gall-bladder; it is normally soft and elastic, but in consequence of the presence of stones it becomes cicatrized, dense, and in cases where no obstruction in the duct exists it becomes contracted in size. Courvoisier found the gall-bladder contracted in 80.25 per cent. of the cases of cholelithiasis. When the gall-stones are large and situated near the neck of the gall-bladder, producing retention of the secretion, there may be great enlargement of the viscus. Experimentally the gall-bladder has been so dilated as to contain fifteen pints (Durian). Clinically it rarely exceeds the size of a child's head. The direction in which the gall-bladder may enlarge varies. It may extend downwards, rarely to the left, most frequently it is found in the median line or to the right of the median line. The author has observed the fundus resting on Poupart's ligament. It may be elongated, narrow, or balloon-shaped and contain as much as a litre or more of mucus and pus or bile. This dilatation is the result of obstruction in the neck, the gall-bladder in some of these cases being thin and pliable; in others where infection exists it is thick, indurated, and œdematous. The writer has observed it as small as the nipple. It is not uncommon to find the gall-bladder hugging closely the calculi impacted within

or even to find elongations of the mucosa embedded within the nes. In other cases there is an incrustation of the gall-bladder l in which the mucosa is embedded to such an extent as to lead to suppose that there may be a true calcareous degeneration of mucosa itself. The latter has not been satisfactorily demonated.

When the gall-bladder is filled with calculi, it is not uncommon to diverticula project from the wall of the gall-bladder which may tain many calculi and these may be practically shut off from the ty of the gall-bladder. The pockets produced in this manner are results of softening or degeneration of the wall at the points where hernias occur, as many of the diverticula are covered merely with serosa of the gall-bladder, and some with connective tissue only, e of the gall-bladder wall being preserved. Whether this is a nary ulceration tending to the formation of hernia of the viscus, or condary atrophy of the walls, is difficult to ascertain, but it is more ly the former. The walls of the diverticulum are fortified by adons from the neighboring viscera. The form, shape, and number iverticula vary greatly. The diverticulum may exceed the size of gall-bladder and contain the only stone present in the case. Dicula may become sessile or pedunculated, and all communication the gall-bladder may be obliterated. A gall-bladder may be did into many separate sacs by cicatrices. These sacs usually comicate with a common centre. Bands may separate the cavities in h the stones are located to such an extent that it is impossible slodge the stone from one chamber into another. A condition of kind forms the greatest objection to cholecystostomy in two sit. Evidence of pericystitis, the result of infection through the of the gall-bladder from within outwards, is commonly found the history of cholelithiasis extends over a prolonged period. e adhesions are life-savers. They consist of omentum or aggluions of adjoining viscera, as the colon, duodenum, etc. When nfection is extensive or a suppurative pericystitis is present, or irulent type of infection exists, necrosis of the viscus which is tinated to the gall-bladder takes place and a cholecystic fistula med.

Symptoms.

alculi may exist an indefinite time without producing the slightest festation of their presence. Fürbringer states that the gall-stones ecognized only when they become displaced in the gall-blad- From reflex influences, from irritation of the mucous mem- by the stone, and more frequently from infection of a greater

or less severity, the mucosa vesicæ pours out an increased secretion which carries with it the stone into the cystic duct or on into the bowel. A sudden dislodgment by violence, excessive exercise, or a contraction of the abdominal wall may also precipitate an attack. When the gall-bladder is over-distended it becomes painful, its muscular contraction, its elastic reactive power, and the force of its accessory compressors are much increased. The prodromal symptoms of an attack are slight, beginning with a discomfort in the right hypochondriac region or epigastrium and gradually increasing in severity. There is an inability to digest certain articles of food, and during the attack the gastric disturbances are exaggerated even to vomiting. Pain, but not colic, is present and is often referred to the angle and inner margin of the scapula. There is obstipation and tympany. These patients have a temperature of 99° to 99.5° F. The above symptoms usually continue for three or four days and then gradually subside to recur as exciting causes arise. The great majority of gall-stone attacks are without jaundice either during or following the attacks. Unless the stone is impacted in the cystic duct colic is absent. Pain, tenderness, and fever may be present. Digestive disturbance is always present when gall-stones produce pathological conditions in the gall-bladder.

A history of previous attacks is usually given. Among the causes for recurrence may be mentioned exposure to cold, overexertion, prolonged journeys, etc.

Examination.—On palpation marked sensitiveness is detected in the right hypochondriac region, increased on deep inspiration. This may be so localized that the gall-bladder may be perfectly outlined. In these cases there is an absence of icterus. If the attack be accompanied by a pericystitis or if the gall-bladder be enlarged, a tumor can be felt.

We must differentiate this disease from ulcer of the duodenum with peri-intestinal adhesions, from infection arising from ulceration of the pyloric end of the stomach, and from malignant disease of the pylorus. The history and course of the diseases are very different.

Treatment.

This consists of rest in bed, warm applications, saline cathartics, and restricted diet. The mechanical treatment of Purckhauer appears not only useless but dangerous. It cannot be reasonably supposed to favor the advancement of the stone and may lead to perforation of the gall-bladder or gall-tracts or prevent adhesions to neighboring tissues or organs. The attack runs its course in from three

six days and may at any time be sufficiently severe to compel the ient to remain in bed. When once the calculi have produced ficient trouble to bring on an attack, the rule is that there are fre- ent recurrences. If the resulting pathological changes become re deeply seated, both the severity and the duration of the attack increased until finally surgical interference is obligatory. This uld consist in the removal of all of the gall-stones whether in the l-bladder or in diverticula.

The indications for operations on the gall-bladder in the absence icterus or the previous passage of stones are: 1. Reflex periodic estive disturbances accompanied by tenderness beneath the right tal arch; 2. Repeated attacks of mild pain or gastralgia with derness under the right costal arch, and an elevation of temperature ging from one to two degrees; 3. Recurrent attacks of colic in the it hypochondrium and the epigastric region accompanied by ked tenderness over the gall-bladder during the attack of pain, h or without elevation of temperature; 4. Tumor in the region of gall-bladder with reflex disturbance or local tenderness. The sitiveness in the gall-bladder can best be brought out by a full in- ation when the finger can be pressed down against the tumor.

The operations indicated are cholecystostomy, cholecystectomy, cholecystenterostomy, depending upon the pathological changes sent.

Obstruction of the Cystic Duct.

This may be caused by (*a*) concrements, (*b*) cicatrices, (*c*) neo- ms, or (*d*) valvular formations.

From a practical standpoint the cystic duct includes the narrow ion of the neck of the gall-bladder as well as the duct itself, and pathological lesions and treatment of both are the same. As the ents in the cystic duct are constant to or fro, it is not probable calculi are formed in that duct. The size and shape of the es found in the duct are as manifold as those found in the gall- der. They are usually firmly impacted in the duct and rarely w of the passage of bile. Occasionally a ball-valve condition has observed in which the bile can readily flow in but cannot escape. rticula are common, the stone only partially occluding the canal. n the calculus is very large it may be forced from the gall-blad- and dilate the duct to its junction with the hepatic (see Fig. 51). size attained in one of my cases was five-eighths of an inch in meter. When the duct is completely occluded there is an ectasia he gall-bladder caused by the accumulating mucus in the sac. is absent, there being usually a hydrops or an empyema of the

gall-bladder, depending upon the presence or absence of infection. The fluid from the gall-bladder in these cases often has no trace of bile. It may be thin and watery and vary from that to the consistency of a colloid substance. Occasionally it contains blood, which indicates that there is probably a malignant growth in the gall-bladder.

FIG. 51.—Dilatation of the Cystic Duct, as far as its Junction with the Hepatic, by a Gall-stone. From a case of cystectomy. The gall-bladder was 7½ inches in length; the calculus measured 1½ inches.

Cicatricial occlusions in the ductus cysticus are almost always the sequence of cholelithiasis. Ulceration and infection of the duct take place during the passage or retention of a calculus, and following this is cicatricial contraction with complete occlusion of the canal. Congenital occlusion has been reported a number of times, but it is of little importance to the clinician. As a sequence of the occlusion of the cystic duct, there may be contraction or dilatation of the gall-bladder. Contraction exists in forty-eight per cent. of the cases recorded. Obstruction of the cystic duct has been found present in a number of cases of pathological cholecystenterostomy in which the fistula communicated with the duodenum, jejunum, colon, or stomach. The cystic duct may be occluded by compression of the duct from without, as by a cancer originating in the gastrointestinal tract or occurring secondarily in the liver, or it may be compressed by infected lymphatic glands which press around the vein and close the duct, as in typhus fever and cholera. Cicatricial bands from a pericholangitis have been found to be a rare cause of obstruction, but contraction of the hepaticoduodenal ligament combined with movable kidney was shown by Weisker to be a frequent cause of obstruction to the cystic duct. He further showed that hydrops and concrements were frequent conditions in the gall-bladder when the right kidney was movable or when there was hydro- or pyonephrosis present. Obstruction of the cystic duct by ascarides and presence of these worms in the gall-bladder and common duct have been frequently reported. They have even escaped from cholecystic fistulæ to the surface of the body. Displacements by hydatid cysts or intracystic rupture of hydatids may produce occlusion of the duct and be followed by a dilatation of

gall-bladder. Primary carcinoma at the neck of the gall-bladder y also occlude its lumen.

Symptoms.

The clinical picture of occlusion of the cystic duct will vary with nature of the occlusion, also with its degree, its duration, and the sence or absence of infection. As the mechanical occlusions occur t commonly from gall-stones, the symptoms of calculus obstruc- will alone be considered. There is given a history of previous cks of pain in the right hypochondrium, accompanied by nausea omiting, obstipation, and slight fever which subsided in a short , and if the calculus has passed through the ducts in a previous ck we shall get a history of associated jaundice. The patient lly attributes these attacks to gastric disturbance or calls them stralgia." So common is this that no case of gastralgia should llowed to pass without a careful examination during the attack, ne differential diagnosis can be made at that time with great cer- ty. Icterus very rarely follows these attacks. The attack will nitiated with pain of a colicky character in the right hypochon- m or in the epigastrium, gradually increasing in severity and hing its height in two or three hours after the onset. Nausea and iting are frequent. The temperature does not rise above 99° to °, unless a severe infection be present. The pain increases in in- ty as long as the occlusion exists and the calculus continues to ad- e in the duct. The patients are physically depressed, but never go collapse because of this obstruction. At the end of three or four s, most frequently after severe emesis, the pain suddenly sub- , the sensitiveness remaining for two or three days. The method lief is by the calculus falling into the gall-bladder.

physical examination shows diaphragmatic respiration very lessened or absent, rigidity of the hypochondriac region by ular contraction of the abdominal wall, and increased resist- below the margin of the ribs, it being impossible to induce atient to relax the muscles. The pain is greatly increased on ure and is much more severe when a full inspiration is taken. sensitive area can be well outlined if the examination be con- d with care and judgment for fifteen or twenty minutes. In ic cystic-duct obstruction the patient keeps a constant guard his right hypochondriac region. He will not allow his clothes brushed or his abdomen to be pressed against. False motions ted towards the patient's abdomen are resisted. A slight slap ne abdomen will make the patient flinch very much. Even mooth, oval, distended gall-bladder may be felt. It may be

freely displaced, and moves up and down with the motion of the liver. It is elastic and fluctuates. Great care must be exercised in differentiating between a distended gall-bladder and a tongue-shaped elongation of the right lobe of the liver produced by corsets. Many times they are associated, the enlarged gall-bladder carrying with it the right lobe of the liver. When both are present the gall-bladder can be felt projecting from beneath the sharp edge of the liver unless the abdomen be too thick for successful palpation. When the tumor is moved laterally its centre of rotation is the costal arch. The hand can be pressed in between it and the kidney. The tumor is in rare instances adherent to the anterior abdominal wall. The varying size of the gall-bladder depends upon the presence or absence of occlusion. During an attack it is large and sensitive, in the intervals it is soft and cannot be palpated. These are conditions of intermittent hydrops of the gall-bladder. Goldwitz claimed that the intermission was due to paralysis of the muscular wall. The author cannot support that opinion, as he does not consider the muscular development of the gall-bladder sufficient to produce such conditions, but attributes it to the ball-valve action of the stone in the cysticus or to the complete obstruction of the duct.

Differential Diagnosis.

Obstruction of the cysticus should be differentiated from hydatid of the liver by the sessile base and the absence of pain in the latter, the pedunculation and history of intermittent pain in the former. It should be differentiated from tongue-shaped elongation of the liver by the sharp edge of the latter and the conformity of this edge to the shape of the liver, the absence of pain or tenderness on pressure, and absence of fluctuation. The lower point of the liver may, however, be round and resemble a gall-bladder. In the case of gumma the tumor is often multiple, rarely projects abruptly from the liver, is not spasmodic in its manifestations, and there is often a higher and more continuous temperature. In carcinoma there is enlargement of the whole liver, with irregular nodules on the surface, and icterus is present. Cyst of the pancreas may be differentiated by its deep situation, the longest diameter being transverse to the body, and its fixed position. The edge of the liver can be frequently felt to move up and down over this tumor. Carcinoma of the pylorus may be differentiated by the history, the emaciation before the tumor could be detected, the absence of fever, the irregular nodular character of the tumor, and the situation of the tumor close to the median line. Movable kidney and hydro- and pyonephrosis, are characterized by the

t that we are able to separate the tumor from the liver, the greatest bility being parallel with the long axis of the body, and by the minence of the tumor behind, as well as by the evidence obtained m the urine. From tumors of the omentum on the right side it is ctically impossible to differentiate obstruction of the cysticus. ey are of such rare occurrence that they scarcely come under con- eration. Malignant disease of the colon or fæcal impaction is nifested by the shape, mobility, resistance, nodules, and previous tory. Appendicitis, when the appendix is situated close to the r, is marked by the history, initiated with sharp pain, nausea, l vomiting, and very early elevation of temperature, above 101°, ch is not common in cystic-duct obstruction, except when there nfection with acute empyema or gangrene of the gall-bladder. In great majority of cases of appendicitis the tumor can be separated n the margin of the liver; the rapid formation of the tumor in ap- dicitis, and its gradual formation in cystic-duct obstruction are gnostic points of value. The history of previous attacks, with or nout jaundice and with or without elevation of temperature, is important in the differential diagnosis. If the case be exam- l before and during an attack, the differential diagnosis can be le with great precision from all the conditions mentioned. These s are as frequently duplicates of each other as are cases of ap- licitis.

Treatment.

The treatment should be divided into three classes: (1) Prophy- c treatment against attacks, (2) the treatment during attacks, (3) the radical treatment.

Occupation, diet, and exposure act as exciting causes. Most of patients can attribute the attack to certain kinds of diet, and if can be withdrawn they have a much longer respite. Exposure old is another frequent cause. Prolonged travelling over rough s is one of the exciting causes often alleged by patients residing e country. Water cures, particularly at Carlsbad, hot baths, milk , all appear to lessen the frequency of the attacks. Large doses nosphate or hyposulphite of sodium taken in a glass of hot water t and morning have apparently the same salutary effect. The ment during the attack should be the use of sedatives, as phos- e of codeine, muriate of pilocarpine, saline laxatives, hot fomen- ns in the region of the gall-bladder, and absolute rest. A care- ecord should be kept of the temperature, the character of the and the location of the tenderness. If the patient has jaundice g the attack it is possible that the calculus or many of the cal-

culi may pass through the duct into the intestine. The stools should be carefully sifted for their detection. When jaundice does not occur it may be concluded that the stone is still in the cystic duct or gall-bladder. These attacks may occur every two or three weeks or may not recur for months. The writer has reported a case in which they occurred regularly at the time of menstruation. The woman was subsequently operated on and found to have three large gall-stones, one of which was impacted in the cystic duct.

The radical treatment depends upon the causal relations and consists of cholecystostomy, cholecystenterostomy, or cholecystectomy.

Intrahepatic Gall-Stones.

The literature is fairly rich in instances of intrahepatic calculus, but an analysis shows that the cases have been very defectively recorded. The reports of autopsies and operations do not show conclusively that the stones were of intrahepatic origin, that is, that they did not escape through perforations of the gall-bladder into the liver or to the surface of the liver, or that the suppurating gall-bladder was not adherent or embedded within the liver substance. There is, however, a sufficient number of accurately recorded autopsies to prove that calculi are formed in the smaller hepatic ducts.

The symptoms and clinical history are not sufficiently distinctive to admit of a diagnosis. The diagnosis of cases reported as operated on for the removal of gall-stones from the liver should be questioned, as the entire pathogenesis of these cases cannot be recognized in an operation. Gall-stones have been removed from abscesses and bile cysts of the liver.

Gall-Stones in the Hepatic Duct.

Gall-stones in the ductus hepaticus have been observed and while they are not of frequent occurrence, still they play an important rôle in cholelithiasis. They are rarely found, except in cases in which gall-stones are present in other ducts or in the gall-bladder, or in which there is evidence that they have previously existed at these points. The hepatic duct contained gall-stones in less than one per cent. of post-mortem examinations in which gall-stones were present in some part of the tract. Their presence therefore might be accounted for by their expulsion from these points into the hepatic duct. One or both of the principal branches might be occluded and the number of stones present has varied greatly. Courvoisier reports a case in which all the ducts, even to the small hepatic bifurcations, were

ll of gall-stones and there had been no symptoms produced or in-
cations of their presence until twelve days after an operation for the
dical cure of hernia. The great majority of stones are undoubtedly
gall-bladder or cystic-duct origin, as in 59 cases collected by Cour-
isier 56 had gall-stones in either the gall-bladder or the common
ct; and of 51 cases of calculi of the hepatic duct of which the exact
ndition was given, 45 had contraction or obstruction of the hepatic
ct, showing that retardation of bile favors the formation of stone.
seems that the pressure and tension in the gall-bladder force the
culi from the gall-bladder into the common duct, and when the
ter is obstructed they are forced up the hepatic duct. This opin-
is supported by the fact that in 45 out of 51 cases of stones pres-
in the hepatic duct, there was also obstruction to the common
t by calculi. There have been cases reported of primary car-
oma of the duct, secondary sarcoma, obliteration of the duct from
atricial contraction, congenital imperforation of the duct which is
ually associated with that of other ducts, compression of the he-
ic duct, malignant growths, occlusions by echinococcus cysts, by
arides, etc. These cannot be recognized during life.

Symptoms.

The chief symptoms of obstruction of the hepatic duct are a sen-
on of pain and discomfort in the region of the liver, and gastric
urbance, particularly vomiting. The pain is not often colicky
has no distinctive character to lead to a diagnosis. Jaundice
present in a greater or lesser degree in forty-one per cent. of
cases. When the calculus in the hepatic duct or in the liver sub-
ce is found in a dilated sac, and there has been produced an
phy or cystic condition of the bile channels tributary to that
, there is rarely sufficient bile retained to produce jaundice.
everal of the cases in which jaundice was present there was also
bstruction of the common duct. Fever of an intermittent type
present only when infection existed or when liver abscess was
ciated with it, and then in only twenty-two per cent. of the
s. The disease is unfortunately associated with gall-stones in
r positions, in the ducts or gall-bladder, in such a large number
ses that a differential diagnosis between them cannot be made;
the attention of the physician and of the patient is drawn to
iver by these complications. The occasional occurrence of gall-
es in the hepatic duct and liver substance undoubtedly explains
e of the cases in which a continuation of the symptoms exists
the common and cystic ducts and gall-bladder had been freed
perative procedure.

Treatment.

A careful examination of the hepatic ducts should be made in every operation on the cystic or common duct. An excellent exposure of the under surface of the liver and hepatic ducts may be made by elevating it with a Sims' speculum. If a calculus is located it should be treated on the same principles as if found in the cystic or common ducts, except that cholelithotripsy by palpation or puncture with a needle should be tried in preference to incision into the duct.

The medicinal treatment is the same as that for cholelithiasis in general. Regulation of the diet, prolonged water cures, attention to clothing, etc., are also of service.

If suppuration be produced around a calculus in the liver and an hepatic abscess result, it should receive appropriate surgical treatment.

Obstruction of the Ductus Choledochus.

These may occur from (*a*) gall-stones, (*b*) foreign bodies, (*c*) neoplasms, (*d*) obliteration, or (*e*) compression.

In the Pathological Institute at Basle, of 2,520 sections, there were 255 instances of cholelithiasis. In 10 of these cases the choledochus was obstructed. In practically 10 per cent. of all the cases gall-stones were present and in 0.4 per cent. the gall-stone was in the common duct. These statistics should be borne in mind when considering the symptoms of cholelithiasis, as they show why jaundice is not more frequently present. Gall-stones in the common duct are very often associated with gall-stones in the gall-bladder, less frequently in the cystic, and rarely in the hepatic duct.

Calculi occur in the common duct from three sources: First, and most frequent, by being expelled from the gall-bladder through the cystic duct into the common duct; second, by their passage from the liver and hepatic duct into the common duct; third, and least frequent, by their formation in the duct itself. The size and physical characteristics of the calculi in the common duct have all the variations that have been found among those contained in the gall-bladder, cystic and hepatic ducts, and the liver, as in the great majority of cases they have been propelled from these sources. The gravel that is not uncommon in the gall-bladder is rare in the common duct, perhaps, because stones of such minute size would not be retained there but would pass on into the intestine. The range in size is that from a pea to a pigeon's egg. The number varies greatly, depending upon the dilatation of the duct and the size of the stones.

The position of the stone in the duct in 177 cases collected by ourvoisier was as follows: In the opening of the duct, 17; in the iddle, 19; close to the duodenum, 20; in the papilla, 41; and the ıtire duct was full in 26. It should be noted that in a majority of ıe cases the stones were near the duodenal end, which is the small- t part of the canal.

The results of gall-stones in the choledochus may be consid- ed under two heads: (1) The effect upon the bile circulation above e obstruction, and its constitutional result, and (2) the local ef- ct upon the tract itself. The local effect upon the canal depends on the size of the stone, the length of time it is retained in one sition, the shape of the stone, and the presence or absence of in- tion. The stone is propelled through the duct by the contraction its circular muscular fibres (Naunyn). The bile pressure itself s little to do with the advance of the stone, it being equal to a lumn of mercury only 24 mm. high. The greatest pressure exerted stones from behind is when they are impacted in the cystic duct, is pressure being due to pathological accumulations within the ll-bladder. Large calculi are propelled through the common duct wly, and are frequently retained in one position for a long time, ducing ulceration and necrosis of the mucous surface, and not in- quently perforation. When a number of calculi are retained in the ct they often produce diverticula. These cases are not always ompanied by jaundice, as it is possible for the bile to pass between around them on through the duct. Occasionally with the presence a large stone we have a sacculated condition of the gall-bladder l a ball-valve action of the calculus against the opening (Fenger). e catarrhal inflammation produced by the passage of a stone is stly confined to the choledochus; but when this is accompanied an infection from the duodenum, it may extend up the ducts and iate a general cholangitis, which is very frequently fatal. After eration of the duct and after the stone has passed on into the duo- um, we have as a sequel cicatricial contraction, even to complete lusion. The formation of carcinomata in the duct is generally posed to be a result of the irritation of retained gall-stones. The ılts of perforation of the duct and the septic manifestations from erations will be considered under separate headings.

Another result of obstruction of the duct is to produce a dilatation ll of the tracts above with the exception of the gall-bladder, and of t also except when the former presence of a gall-stone had caused ickening of its wall. The degree to which the common duct may ome dilated is very great. It has been described as eight inches g and five inches wide (Frerichs). Terrier reports four cases in

which it was as large as the intestine. The duct may be sacculated and appear as a cyst. The intrahepatic ducts may be so dilated that the whole liver is a fluctuating mass with channels as large as the little finger.

The most frequent cause of closure of the duct by foreign bodies is echinococcus cysts. They originate in the liver and rupture into the duct, the daughter cysts producing the occlusion. Ascarides obstruct this duct the same as other ducts above mentioned. Foreign bodies or gases cannot pass into the choledochus from the intestine unless the canal has been pathologically dilated, that is, by the passage of stones or other foreign bodies. Of neoplasms of the duct the most common are carcinomata. The other tumors recorded have been polypi, fibroids, and villous growths. The carcinomata exceeded in number all of the others and were recorded as epithelioma, adenocarcinoma, scirrhus, cylindrical-celled, etc. In the great majority of cases the growth was primary, though it has occurred secondarily from carcinoma of the pylorus. It is noteworthy that gall-stones were present in seventy-eight per cent. of cases of carcinoma. Cicatricial occlusion of the choledochus was shown in a large percentage of cases to be a sequence of cholelithiasis with subsequent cicatricial deposit in the wall of the duct itself (Benzançon).

The congenital obstructions of the choledochus are often associated with obstruction of the hepaticus or cysticus and are most likely due to syphilis, although this has not been demonstrated.

Closure of the duct by compression from without may be caused by growths in the neighborhood of the duct. The most frequent of all is carcinoma of the head of the pancreas. Next in frequency is carcinoma of the duodenum, and next to that is carcinoma of the pylorus. Aneurysms, hydatids, and abscesses in the neighborhood have been known to produce occlusion by pressure from without.

The gall-bladder was atrophied in 80.4 per cent. of the cases of obstruction of the choledochus by gall-stones, and dilated in 19.6 per cent. In obstructions from other causes than gall-stones the gall-bladder was dilated in 92 per cent.—an important factor in the differential diagnosis (Courvoisier). The fluid in the dilated tracts varied considerably. In a small percentage of the cases it was purulent, and in the greater number of these the pus was confined to the gall-bladder; in other cases the fluid was clear mucus, all traces of bile being absent; but in the majority of the cases it was a thick, dark-colored bile. When the pus is contained in the tracts it may lead to abscess of the liver, or to necrosis and perforation of the wall, especially when it is accompanied by a calculus.

Symptoms.

The symptoms vary with the character of the occlusion. With ıll-stone obstruction there is often a history of previous obstruction the cystic duct, or a previous cholecystitis. In the initial stage ılic is the predominating symptom. The attacks of colic may have isted for years before the calculus enters the common duct. The mptoms of obstruction of the common duct in the order of their currence are: Sudden onset of colicky pain, nausea and vomiting, nsitiveness in the right hypochondriac region, normal or subnormal nperature except in infected cases, occasionally collapse, presence bile in the urine, and two or three days later icterus. The only mptom in addition to those of cystic-duct obstruction, and one ıich is never present in occlusions of that duct, is jaundice. In ute attacks in which the gall-stones are small and expelled through e ducts, jaundice may occur on the second or third day after the set of pain. It will continue as long as the obstruction at the com-ɔn duct continues, and disappears three or four days after the latter relieved.

The history of intermittent attacks of jaundice with the recog-ion of gall-stones in the fæces confirms the diagnosis. The pain biliary colic is due to an overdistention of the duct, as we find dilatation of any muscular or non-muscular channel. Its inter-ttent increase is not due to spasm but to periodic occlusion or ad-ıcement of the stone to a new position. That it is not due to ɜtruction alone or to spasm of the duct is shown by the absence colic in all malignant occlusions not associated with calculi, and the absence of colic after the bile pressure has been relieved by a ɔlecystotomy although the stone has been allowed to remain in the ne position in the common duct. The nausea and vomiting are re-t and may have accompanying them all the symptoms of adynamic ıs.

The fever in obstruction of the choledochus is due (1) to local eration, (2) to angiocholitis, and (3) to retention of infected mate-l from the gall-bladder behind the impacted calculus. Among metastases from these infections may be mentioned multiple ıcess of the liver, subphrenic abscess, empyema, endo- and peri-ditis. The collapse is a general manifestation of the pain and the ex action through the sympathetic. Bile in the urine and icterus the evidences of the admission of bile into the circulation. In early stage of obstructive jaundice there is enlargement of the ɔr from a dilatation of the small bile channels. The bile is ab-

sorbed by the lymphatics, carried into the thoracic duct, and admitted into the blood through that channel. If the thoracic duct were ligated there would be no jaundice from the obstruction. When it is ligated experimentally after jaundice is present the jaundice disappears (Vaughn Harley). The hepatic cells become smaller and smaller, depending upon the degree and continuance of the obstruction.

Jaundice with gall-stones is due (1) to obstruction in the common or hepatic ducts, (2) to inflammation and swelling of the mucosa of the ducts producing obstruction. This inflammation or angiocholitis may be the result of the passage of the stone through the ducts, or it may be an extension of the inflammation originating in the gall-bladder and propagated along the ducts. This latter variety of jaundice is not associated with colic unless the calculus obstructs the duct. The intensity of the jaundice depends on the completeness and duration of the obstruction. I cannot agree with Pick that jaundice may be produced by reflex disturbance excited by the presence of the stones in the cystic duct or gall-bladder.

Diagnosis of the Cause of Obstruction.

The differential diagnosis between calculus obstruction and other varieties of occlusion of the duct is of great importance. If the duct obstruction comes from other causes than calculus there are no pathognomonic symptoms or distinctive manifestations. Gall-stone obstruction can be diagnosticated with reasonable probability by the following complexus of symptoms: (1) History of previous attacks of colic (gastralgia) with or without jaundice, accompanied by vomiting and local tenderness in the region of the gall-bladder; (2) colic followed in a few days by jaundice; (3) recurrence of jaundice of short duration; jaundice of long duration, sometimes even years, when preceded by colic; previous attacks of jaundice with the passage of gall-stones in the fæces; (4) the development of an intermittent type of fever after the jaundice has existed for some time.

When the enlargement of the liver is due to gall-stones the organ is soft and spongy at the edge. The edge is round and can be easily indented while the enlargement from secondary carcinoma is hard and nodular. The enlargement caused by gall-stones never takes place so rapidly nor to so great a degree as that due to secondary carcinoma.

The most common cause of obstruction of the choledochus, aside from gall-stones, is cancer of the pancreas. The differential diagnosis between cicatricial obstruction and non-malignant neoplasm is impossible except by exploratory incision and careful examination. The jaundice in these cases may appear suddenly, but the prodromes

e very marked, the emaciation, the lassitude, the presence of tumor the epigastrium and the absence of colic aid us in the differential agnosis. In malignant obstruction the jaundice is permanent and not accompanied by fever. The purpura and capillary hemorrhages om the mucous membranes have no specific relation to gall-stone struction. They are merely the result of cholæmia from any cause.

Intermittent attacks of jaundice in rapid succession when the stones nnot be found in the fæces may be due to any of three causes in the llowing order of frequency: (1) Large calculi in diverticula or in lated gall-ducts acting as ball valves; (2) tumors, within or without, compressing the canal; (3) intermittent catarrhal conditions. In e first two there is no actual intermission but rather a remission the jaundice and pain; in the latter there may be entire intermission. When long intervals of time, as months or years, intervene between the attacks, they are practically pathognomonic of gall-stones. ntinual icterus of years' duration is rather indicative of gall-stones n of other obstruction. Icterus of over fifteen months' duration y be considered as due to gall-stones or at least produced by a n-malignant obstruction. To differentiate mechanical from catarl jaundice, it has been suggested that one-sixtieth of a grain of lrochlorate of pilocarpine be given twice daily for two weeks. If jaundice and subjective symptoms continue, there is some menical obstruction, gall-stones, cicatrices, or neoplasms, while if the ndice be catarrhal it will disappear under that treatment.

The next symptom of importance is fever. Charcot attributed fever to the retention of bile and called it "intermittent hepatic er." Schüppel considered it due to inflammation. It is of an inmittent character and often accompanied by chills. In the order the frequency of their occurrence the intervals are 8, 14, 5, 4, 3, 2 days, the most common being the 8-day variety. In practiy all of the cases in which fever was present in Courvoisier's colion there was ulceration or suppuration of some part of the gallt. Schüppel and Quine's observations confirm the theory that fever is of septic origin.

Treatment.

The treatment of obstructions of the choledochus may be divided three classes: (1) Symptomatic treatment during the attack; (2) prevention of return of the attacks; (3) the radical removal of cause of the obstruction. When the obstruction is due to gallses the onset is usually severe, the pain is intense, it is accomied by nausea, vomiting, and obstipation. The patient becomes ressed and has a frequent, weak pulse. The skin is cold and

clammy, the expression is anxious, and in some cases there are symptoms of profound collapse. These symptoms are met as they arise by opium for the pain, preferably morphine administered hypodermically in large doses of from one-third to one-half grain. Repeated doses of morphine may be given by the mouth every two to four hours if the pain should continue; hot applications to the epigastrium or a prolonged hot bath, full doses of spirit of chloroform and compound spirit of ether, and, if symptoms of collapse are marked, hypodermic injections of strychnine or caffeine, large quantities of strong coffee, and alcoholic stimulants should be administered. When the calculus escapes into the duodenum the pain suddenly ceases, but sensitiveness remains for several days.

Attention should be given to prevent a recurrence of the attack. The best results have been obtained by prolonged courses of treatment at certain alkaline springs. There has been no rational explanation given as to why certain mineral waters should favor the passage of gall-stones or lessen the frequency of the attacks when the stones have not been passed; but clinical observation furnishes ample proof of the beneficial effects of prolonged mineral-water treatment. The most celebrated of these spas is Carlsbad; others, such as Kissingen, Homburg, Marienbad, Ems, Vichy, Mountain Valley, Contrexéville, and Hathorn, are also credited with having beneficial effects in these cases. When it is impossible for the patients to visit the springs, the medicinal salts or water may be prescribed. Large quantities of water, from two to four quarts a day, should be taken for from five to eight weeks. The symptoms gradually disappear, and the frequency of the attacks may diminish under this treatment without the detection of gall-stones in the fæces. Other forms of treatment, such as a mixture of turpentine and ether, chloroform, salicylate of sodium, etc., are of very questionable utility. Olive oil was once a favorite remedy for this condition, and the literature abounds with reports of cases treated successfully by this method, many claiming that calculi were passed; these were usually described as of the soft, round variety, but they were undoubtedly saponified oil. The diet, occupation, or other influences which have previously precipitated an onset should be avoided.

When the obstruction resists medicinal treatment for a number of days or weeks, the radical removal of the cause of the obstruction must be undertaken. If the patient is very icteric and the icterus has existed for a long time, every prolonged operation must be avoided, or the patient will succumb as a result of the hemorrhage and cholæmia within two or three days. This hemorrhagic tendency is lessened by the administration of large doses of chloride of calcium for several

ys before operation (Mayo Robson). The administration of this t is certainly based on good physiological principles, for the chlo- e is one of the most diffusible salts of calcium, and the fibrin med in the coagulation of blood is an organic salt of this metal. administration should be practised in these cases as well as in læmic purpura hæmorrhagica. So great has been the mortality in lical operations for obstruction of the choledochus with the pres- e of severe icterus that it is questionable whether it should ever undertaken. The indications in these cases are (1) to relieve the læmia, and (2) to remove the cause of the obstruction. It is there- e advisable to do a primary cholecystostomy when the cystic and atic ducts are patulous and the gall-bladder is of sufficient size to mit of the operation. Since a large percentage of the cases of ob- ıction of the common duct have calculi in the cystic duct or gall- ldder it may be necessary to remove them to prevent their advance- ıt into the common duct—an additional reason for making this nary operation.

The author has devised the following operation for the relief of re cholæmia: When the gall-bladder is very much contracted or cystic duct is occluded the cholæmia may be relieved by taking ıntage of the pathological condition of the small bile channels of liver, viz., their varicose condition. Some attain the diameter of little finger. An incision two inches long is made over the surface ıe liver, the peritoneum is accurately sutured to Glisson's capsule one of the dilated bile channels. An incision an inch or an inch one-half long is then made through the wall of the dilated duct. s is kept patulous by suturing the edges to the muscular fascia or toneum. By this simple procedure the bile may be drained. n the cholæmia has subsided a radical operation may be made he removal of the obstruction to the hepatic or common duct. ıis cannot be accomplished, the patient suffers the inconvenience permanent biliary fistula, but life is not jeopardized. After the dice has disappeared a second operation may be performed for emoval of the cause of the obstruction. The patient is then in ıdition to withstand the depressing effects of the operation, and langer of hemorrhage, which is so great in icteric patients, will entirely disappeared. If the obstruction be a gall-stone in the non duct the method of procedure will depend on its location. n the obstruction is from tumors around the duct they should moved if possible. For this purpose the foramen of Winslow be enlarged and the tumor extracted, as was a dermoid tumor case reported by Arthur D. Bevan.

ı malignant occlusion of the duct, as from cancer of the head of

the pancreas, no operative interference should be instituted, not even a cholecystostomy or a cholecystenterostomy, as these patients are cachectic and their vitality is so much reduced that a large percentage of them succumb from the fourth to the seventh day after the operation, and even if the operation be successful life is not much prolonged.

Prognosis.

In cases of obstruction to the common duct the prognosis will depend upon the cause of the obstruction. A great majority of the cases of occlusion of the common duct by calculi are of short duration; the patient recovers from the attack in a week or ten days and remains well until there is a recurrence of the condition. When the calculus is large and is retained in the common duct for more than three or four days the dangers are cholæmia, perforation, and sepsis.

Cholæmia, when there is dilatation of all of the tracts above the seat of obstruction, is accompanied by enlargement of the liver.

Perforation into the free peritoneal cavity, which may result in a general septic peritonitis, sometimes occurs after an exertion. A circumscribed abscess may form and subsequently empty by necrosis and perforation of the bowel, or appear in the epigastrium and be drained from without. The gall-bladder may become adherent to the stomach or intestine at the point of impending perforation, and when the latter occurs a permanent communication results. The perforation may take place into the substance of the liver and give rise to an hepatic abscess.

As a gall-stone passes through the duct it produces a greater or lesser abrasion of the surface and makes an atrium for infection. This infection may appear as a general septic cholangitis and terminate fatally within two or three days from the onset of the symptoms. Fortunately it does not often assume this form. There may be an infection of the ducts with a transmission of the infection to the liver and the formation of multiple hepatic abscesses. The lymphatics may be infected, with involvement of the glands, or a perihepatic abscess may form. The most common manifestation of sepsis is that produced by ulceration of the ducts as indicated by the recurrent attacks of the chills and fever, which may continue as long as the duct remains obstructed and terminate fatally from the constitutional effects produced by the sepsis without the formation of secondary abscess.

Complications.

Oddi describes the cardiac complications of gall-stones. They are of microorganic origin, septic endocarditis being the most com-

on. Septic peri- and myocarditis also occur. They should be nsidered as metastatic infections, the same as from other infectious seases. A pericholangitis, with or without suppuration, may occur om an infection from within or without the duct, and a perforation the wall may take place. This pericholangitis may be suppurative, it it is most commonly adhesive, and the duodenum, gall-bladder, d liver become matted together. This is one of the most com- on difficulties encountered in operations upon the gall-tracts. The mmon and cystic ducts and often the gall-bladder are buried in ese adhesions and can be recognized only after a careful separation the lines of cleavage.

Perforations of the Gall-Tracts.

The most interesting pathological phenomena in connection with seases of the gall-tracts are the efforts made by nature to protect e peritoneal cavity when perforation of the tract is about to occur. Out of 499 cases compiled by Courvoisier, in 8 there were perfora- ns and fistulæ established between different parts of the gall-tracts mselves; in 5 the vena portæ was perforated; in 70 the perforations ened into the peritoneal cavity; in 49 into circumscribed pockets hin the peritoneum; 3 perforations were retroperitoneal; 13 opened o the stomach, 83 into the duodenum, 1 into the jejunum, 1 into the m, 39 into the colon, 7 into the urinary tract, 24 into the pleura l lung; externally through the abdominal wall, 196. In some the cases the perforations occurred into two or even three of se different places. These 499 fistulæ were reported in the his- ies of 1,800 cases. In these statistics the number of perforations he intestinal tract amounts to 137. This figure cannot, however, considered an approximate estimate of the number of times per- ation occurred into the intestines as compared with the num- of external perforations (196), as numerous intestinal perfora- s were undoubtedly overlooked or the diagnosis was not made, le the great majority of the external perforations from gall-stones recorded; however, the figures plainly outline the general ten- cy of the perforations of the gall-tracts. The history of the s of perforation between the gall-tracts themselves is of little in- st, as the pathological condition cannot be diagnosticated except ing operation or post mortem. The same may be said of the per- tions into the portal vein of which there are only 5 recorded cases. perforations in these cases were due either to gangrene or to ssure necrosis produced by the stone; in 3 of the cases the stone jected into the vein. Geigel recently reported a case in which

the pressure of the gall-stone produced a perforation of the choledochus and admitted the stone into the portal vein. There was a thrombus of the vein which broke down and produced multiple hepatic abscesses.

Perforation into the Peritoneal Cavity.

Of the seventy cases of free perforation into the peritoneal cavity only one was diagnosticated *intra vitam* without operation. To this the following case seen by the author may be added.

Mrs. O'B——, age 31. Previous history: Always enjoyed excellent health with the exception of the diseases of childhood. No previous attacks of jaundice or colic. She had complained of slight epigastric disturbance for a number of years. No tenderness was noted in the right hypogastric region.

Present illness: February 19th, 1893, ten days before the patient was seen in consultation, she was delivered of a child after a prolonged but otherwise normal labor. During the last expulsive pains the patient began to suffer from pain in the right hypochondrium, which remained sensitive after labor was completed. Patient felt weak, but had no unpleasant symptoms until four hours after delivery. She then presented a condition that was diagnosticated angina pectoris. The pulse was 140 and weak. There was great difficulty in breathing and deep inspiration was painful. Nausea and vomiting soon occurred; the skin became cold and clammy, the pupils were dilated, and dissolution seemed imminent. Twelve hours later these symptoms subsided somewhat, although the pulse remained very rapid and the breathing was still difficult. The temperature was under 100°. The tenderness in the abdomen continued. The patient was seen only with the aid of artificial light, and jaundice, if present, was not noticed. The urine was high-colored from the second day. Her condition continued about the same until the writer saw her on the tenth day. She was pulseless, pupils dilated, intellect clear, skin cold; she complained of great thirst, and there was sighing respiration; the pulse was 160, and there was an anxious expression.

Examination revealed heart and lungs normal; the abdomen was distended, tympanitic, and sensitive to pressure. It could be divided into two areas by a line drawn from the left ninth costal cartilage to the middle of the right Poupart's ligament. The region above and to the right of this line was flat on percussion, sensitive, resisting, rather sharply outlined. Below the line there was hyperresonance or tympany; there was also absence of severe sensitiveness on pressure here, and there was no induration nor evidence of fluid. The urine was found to contain a large quantity of bile. The induration was immovable. There was slight œdema of the skin over this area; the surface of the tumor was smooth and painful. The bowels moved regularly; the bladder was not distended.

A diagnosis was made of rupture of the gall-bladder with accumulation of bile in the peritoneal cavity. The patient was in such bad condition that an operation was not deemed advisable. The follow-

ıg morning she was comatose and dying. In order to confirm the iagnosis an aspirating needle was introduced, and a quart of bile as withdrawn from the enlargement. It contained a few flocculi. he margin of the induration which had circumscribed the large ac- ımulation of bile could then be easily outlined. No gall-stones were etected. Death occurred two hours later. An autopsy was not per- itted.

The danger of early perforation after the onset of symptoms is eatest in the acutely infected cases with obstruction to the ductus sticus. I have observed cases in which the gall-bladder was com- etely gangrenous within forty-eight hours after the onset of symp- ms. While perforation from the pressure of an impacted calculus es occur, it is not so common as the necrosis and perforation m infection. It can be readily seen that while the bile in the nor- ıl condition is not septic, in the great majority of cases of perfora- n there is present a pathological process in the gall-bladder, and bile becoming thereby infected the danger to life when it is ad- tted into the free peritoneal cavity is great, as a general peritonitis ıally follows. Where the perforation takes place and the adhe- ns retain the infective material and form a circumscribed intra- itoneal abscess, the danger is very much less, as a secondary ne- sis of the bowel or abdominal wall may take place and drain the cess without producing a general suppurative peritonitis.

The *symptoms* of perforation of the gall-tracts into the free perito- l cavity will depend in a great measure on the quantity of pus or discharged and the virulence of the infection produced. In the e of gangrene the onset is violent; marked symptoms of peritoneal olvement, great pain, local tenderness, nausea and vomiting, ileus, collapse follow each other in rapid succession until the patient cumbs to the absorption of the products of the pathological proc- in the peritoneal cavity.

The course is very similar to that of a perforative peritonitis from endicitis, only not so fulminating and destructive in character as latter is apt to be.

The *differential diagnosis* between appendicitis and gall-bladder oration may be made by the history of previous attacks associated jaundice and the location of the dulness, induration, and sensi- ıess. If the case is not of the fulminating variety the increase in ırea of dulness in the right hypochondriac region may be noted hour to hour or day to day. Respiration is very much more promised in peritonitis produced from gall-stones than in that luced by perforations of the appendix. There is no pathogno- ic symptom of the condition.

The indications for *treatment* are the same as in perforative peritonitis from any other cause, that is, prompt surgical interference. Cases of circumscribed peritonitis or circumscribed intraperitoneal abscess, the result of perforation of the gall-tracts, run an entirely different course. The symptoms are those usually present in cholelithiasis with a history of repeated "gastralgias," gastric crises, often associated with jaundice. At the time of the perforation there is a history of acute inflammatory disease in the right hypochondriac region, accompanied by local tenderness, sometimes by ileus, pyrexia, and induration; the size of the induration is no indication of the quantity of pus. These cases run a subacute or chronic course unless there is a secondary perforation of the abscess into the peritoneal cavity. From the number of intestinal fistulæ recorded it is presumable that a large percentage of these cases empty into the gastrointestinal tract like perityphlitic abscesses.

The differential diagnosis between empyema of the gall-bladder and pericystic abscess, except when the latter is very large, cannot be made, nor is it very important from a practical standpoint, as both have the same primary operative indications—external drainage as soon as the diagnosis of suppuration is made.

Perforation into the Gastroenteric Canal.

The *symptoms* of perforation of the gall-tracts directly into the gastrointestinal canal are very obscure. In the great majority of cases large gall-stones that are passed through the intestinal tract find their entrance into the intestine through perforation of the wall of the gall-bladder or ducts and are not admitted through the dilated bile channels (Thiroloix, Létienne, James Collins). This statement is supported by the large number of cases of ileus and those in which large biliary calculi have passed per anum without there having been a history of jaundice, as well as by the fistulæ between the gall-tracts and the intestines found post mortem and during operations. The symptoms of perforation of the gall-tracts depend not so much on the perforation itself as upon the tissue, organ, or cavity into which it perforates, *i.e.*, perforation of one portion of the gall-tract into another does not give symptoms, while perforation into the urinary or intestinal tracts, into the peritoneum or pleura, gives symptoms peculiar to the pathological changes produced in these organs or cavities. That it is not impossible for large calculi to pass through the ducts into the intestine, is shown by post-mortem observations. Of thirty-six autopsies in which large concrements had escaped into the intestine, in only three cases could it be demonstrated that they passed the entire distance through the duct, showing that this route is the exception.

e duct may be enormously dilated, as is shown by the large calculi
t are now frequently removed by operation from the cystic and
nmon ducts, particularly from the lower third of the latter. The
hor has successfully removed a calculus seven-eighths of an inch
length and five-eighths of an inch in diameter from this position.
st-mortem and surgical observations show that the most difficult
tion of the duct for the stone to pass through is between the am-
la of Vater and the ostium duodenale. At this point a necrosis of
wall of the duct and of the duodenum takes place and a perfora-
n often occurs through which the calculus passes into the intes-
e. Kirmesson and Rochard analyzed one hundred and five cases
ileus from gall-stones in which there was a mortality of fifty per
t. There was rarely a dilatation of the common duct, showing
t the calculus did not reach the intestine through that channel,
ulcerated through the wall of the gall-bladder. The course of
cases was chronic, the patient living many days after the obstruc-
n. This is accounted for by the fact that necrosis of the bowel
ly takes place from gall-stones, as they are propelled from place
lace and do not remain long enough in one position to produce
foration. The patients have the symptoms of obturation ileus.

The symptoms of perforation of the gall-tracts into the intestine
seldom sufficiently distinctive to lead to the *diagnosis*. Riedel and
her succeeded in diagnosticating two fistulæ of this character, one
ling to the colon and the other to the duodenum, and proved the
e by operation. During operations for gall-stones a number of
ulæ have frequently been observed.

Perforation into the Urinary Tract.

Perforation into the urinary tract admits of an early diagnosis.
The absence of icterus, large quantities of bile, cholesterin, or
atic calculi in the urine are pathognomonic of this lesion. The
ion of the urinary tract involved or the method of invasion of the
t is not so easily ascertained, that is, in many cases we are un-
to say whether the passage is through the kidney, the ureter,
patulous or pathologically dilated urachus (Hahn and Krönlein),
stula opening into the urinary bladder. Fistulæ leading into the
na are very rare; only three cases have been collected by Cour-
ier and some doubt exist as to these.

Perforation into the Thoracic Cavity.

Invasion of the thoracic cavity may occur in one of several ways.
t, abscess of the liver resulting from an infection of gall-tracts
wed by subphrenic suppuration which finally perforates through

the diaphragm and pleura into the pleural cavity or lung. Second, through perforation of the gall-bladder, usually at the fundus, and the formation of an abscess with burrowing of pus into the subphrenic space without involvement of the liver and the rupture of the subphrenic abscess into the pleura or lung. Third, the formation of an abscess in the mediastinum from perforation of the gall-tracts and the subsequent perforation of the pleural cavity or lung. The tract of admission of bile is often very circuituous, and the history of the case extends over a long period and is of an indefinite character.

The *symptoms* of perforation into the pleura are those of a septic pleurisy. The diagnosis is verified by puncture of the pleura and the analysis of the fluid. When the perforation is into the bronchus the biliary expectoration is pathognomonic of the lesion. The quantity of bile varies greatly in these cases; in some it is enormous, in others it amounts to a few ounces daily.

The termination of these cases is usually favorable. When rupture into the pleura occurs, immediate and continued drainage is indicated; when into the lungs the treatment is expectant.

Perforation Externally.

External perforations of the gall-tracts comprise about forty per cent. of all perforations recorded. The position of the perforation is most frequently at the margin of the ribs in the hypochondrium, next in frequency at the umbilicus or in close proximity to it; occasionally it occurs in the right iliac region or in the loin. External fistula may, however, open on the opposite side of the body, as in Patheret's case in which a biliary fistula opened into the left flank. The autopsy showed that the sinus extended from the hilum of the liver, between the layers of the lesser omentum behind the stomach, over the left kidney posterior to the descending colon, finally forming an abscess and fistula in the left flank. The cause of the fistula in the great majority of cases has been gall-stones; other obstructions of the duct, as carcinomata and intestinal parasites, rarely cause it.

The duration of the fistula will depend largely upon the condition of the gall-tracts as well as the cause of the fistula, *i.e.*, if we have an occlusion of the cysticus or the choledochus it will continue as long as the occlusion exists. In post-operative fistula when the gall-bladder has been sutured to the skin, the fistula may continue indefinitely with patulous ducts. When the gall-bladder has been sutured to the peritoneum the fistula closes, as a rule, in from six to ten weeks if the ducts are patulous. Calculi in large numbers and of varying size escape through these openings as well as pus, mucus, and bile. The quantity of bile that may be discharged from a biliary

stula will depend upon the quantity of bile secreted and the resistance offered to its passage into the intestinal tract. When the common and hepatic ducts are patulous, slight pressure on the opening of the fistula will cause the bile to pass through the normal channels to the intestine. When there is an occlusion of the common duct the entire quantity of bile will pass through the external fistula. A mucous fistula is an evidence that there is an occlusion of the cystic, which may be due either to a cicatricial stenosis, resulting from necrosis of the mucosa, or to a stone impacted in the cystic duct. It is not uncommon to have a calculus, which has occluded the cystic duct for weeks or months, present at the fistulous opening, having been expelled from within outwards in the direction of least resistance. In two cases observed by the writer calculi which obstructed the common duct were forced back through the cystic duct into the gall-bladder and extracted through the fistula.

When there is a complete occlusion of the common duct and the entire quantity of bile escapes through the fistula, the patient becomes emaciated and finally dies (Courvoisier). The profession is changing its opinion as to the importance of bile in the intestinal tract. It was believed for a long time that it was absolutely necessary for the maintenance of life. Mayo Robson does not consider that external fistula with a discharge of the entire quantity of bile is a grave condition. He believes that bile is an excretion, although it probably aids absorption in the intestine, but that its presence is not essential to the digestion of food or necessary for the stimulation of peristalsis, and that it has feeble or very feeble antiseptic properties. He found that the so-called cholagogues had no effect in increasing the quantity of bile from such a fistula, but that carbonated mineral waters increased the quantity without a subsequent diminution. Investigation did not show whether the increase of the elimination of the solids took place or merely an increase in the quantity of fluid.

Treatment.—Internal medication is useless. The surgical treatment will depend upon the pathology of the fistula as well as upon the pathological conditions of the tracts. If a permanent occlusion of the common duct exists from either calculus, neoplasm, or cicatrix, or suppuration of the gall-bladder has ceased a cholecystenterostomy is the operation *par excellence*, as it allows the bile to enter the intestine, relieves the biliary pressure, and allows the fistula to close. In occlusion of the common duct, when cholecystenterostomy is impossible, an effort must be made to remove the cause of the obstruction. After the patient has been relieved of the cholæmia, and the suppuration in the gall-bladder, if it existed, has ceased, operative measures should be instituted.

NEOPLASMS.

Carcinoma.—Cancer of the gall-bladder has been recognized for many years. The relation of carcinoma to gall-stones has been the subject of much controversy. Many authors claim that gall-stones are sequences of biliary retention produced by the growth, while others contend that the calculi are the cause of the neoplasm. The latter may be accepted as the correct deduction, as in 169 cases of carcinoma gall-stones were present in 89 per cent., and the history of the existence of gall-stones in the gall-bladder for a number of years prior to the carcinoma was noted in a large majority of the cases. Delano Ames places the percentage much higher (91–95 per cent.). Siegert claims that they are present in 90 per cent. of the cases of primary, and only in about 15 per cent. of the cases of secondary carcinoma of the gall-bladder. Ulceration is a predisposing cause; the irritation from gall-stones produces a loss of balance in the epithelial reproduction and a tendency to excessive cellular proliferation and cancer (Zinker). Calculi are present in a very much smaller percentage of the cases of primary carcinoma of the ducts, but this is no proof that they did not cause the carcinoma in their passage some time previously. May recently reported a case of primary carcinoma of the common duct with a gall-stone present.

The carcinomata are of many varieties, the most frequent being the scirrhus, next the cylinder-celled, colloid, medullary, etc. They occur most frequently as primary growths; a secondary cancer is invariably a continuation of the process from the neighboring organs, pylorus, pancreas, duodenum, etc. It is never metastatic.

The shape and size of the carcinoma vary greatly with the histology of the tumor in the individual case. It involves the fundus more frequently than any other portion of the gall-bladder. This is the position in which the gall-stones rest and where they would be likely to produce slight irritation over an extended period of time.

Metastases occur in the liver in about fifty per cent. of the cases. They are embolic, occurring through the veins of the gall-bladder and the lymph channels. It is uncertain, as to the metastases in other organs, whether they are primarily from the gall-bladder or arise from the secondary carcinomata of the liver.

The *symptoms* of carcinoma of the gall-bladder are the symptoms common to carcinoma of structures not of vital importance to the economy. They are pain, emaciation, cachexia, anorexia, and reflex gastric symptoms, and these, following a history of gall-stones, are the only symptoms present. Sensitiveness in the neighborhood of

ıe gall-bladder is less pronounced than in chronic cholecystitis. he irregular nodular tumor present in about one-half of the cases ınnot be differentiated from the irregular nodular tumor produced y pericystic infections from inflammatory diseases of the gall-blad- ɐr. Musser estimates that pain is present in sixty-two per cent., terus in sixty-nine per cent., and tumor in sixty-eight per cent. of ıe cases. Death occurs in from three to six months after the evi- ɐnce is sufficiently clear to admit of diagnosis. This must not be ken as an estimate of the time at which death occurs after the onset the disease.

A case of villous cancer of the gall-bladder was cured by Mosetig- oorhof by curetting and by daily inserting pencils of methylene ue and administering nine and one-quarter grains of the same drug ternally. The one important symptom present in a large percent- ɐe of the non-malignant irregular tumors in the right hypochon- ium is an elevation of temperature. This is absent in carcinoma. ften after an exploratory opening has been made it is not easy to fferentiate inflammatory induration from carcinomatous tissue. any cases are reported in recent literature in which a diagnosis of rcinoma had been made, and the abdomen was closed, but the pa- ɐnt made a complete recovery, proving that carcinoma had not isted. Enlargement of the liver is not present unless it is second- ily involved, in which case it rapidly increases to an enormous ze. When the ducts are included in the carcinoma, we have an en- ɐgement produced by bile retention. Ascites and fever do not ac- mpany an uncomplicated carcinoma of the gall-bladder. Pleuritic usions are often present, not as a primary manifestation, but sec- dary to involvement of the liver, the carcinoma penetrating the ıphragm and appearing on its pleural side.

The *diagnosis* can be more readily made when jaundice is absent when cachexia and emaciation precede the jaundice. Occasion- y we are assisted by an early secondary carcinoma of the um- icus. This was the first evidence in a case observed by the iter in which the symptoms of hepatic calculi extended over a riod of twelve years. The case terminated fatally in five weeks th secondary carcinoma of the liver without emaciation or cachexia. ıere were numerous gall-stones in the gall-bladder with primary ɐcinoma, also secondary cancer of the liver and ligaments. The ration of the disease after the symptoms of gall-bladder carcinoma ɐ manifested is extremely short, according to Courvoisier's statis- s, only three months.

Carcinoma of the cystic duct has been reported only twice. Sev- ıl cases of carcinoma of the choledochus have been reported; the

disease affected the intestinal portion most frequently, the hepatic next, and the middle least.

Treatment.—As the disease is generally recognized but a short time before death it can be seen how hopeless an operation would be. The success of Hochenegg and others illustrates the importance of an earlier diagnosis and operation in order to obtain the best results. Further, post-mortem examinations show that metastases are absent in fifty per cent. of the cases, emphasizing the probability of permanent cure if the primary seat of the disease had been removed. In no other organ are metastases absent in fifty per cent. of the cases at the time of death from carcinoma. For this reason operation in these cases should be encouraged and urged beyond that of operations for carcinoma in any other part of the body. This does not include epitheliomata. Cholecystectomy for carcinoma is neither a difficult nor a dangerous procedure.

Benign Neoplasms.

Fibroids and fatty tumors have been reported as occurring in the common duct and have been considered under the head of obstructions of the choledochus. W. W. Keen excised an adenoma of the bile-tract successfully. Echinococcus cysts of the gall-bladder are of rare occurrence and have no special symptoms. The diagnosis is not possible until the contents of the gall-bladder are examined. Cysts of the gall-bladder occur. Three are reported by Adler, one containing cholesterin crystals.

INJURIES OF THE GALL-TRACTS.

Injuries of the gall-tracts may be divided into three classes: (1) Penetrating wound of the gall-tracts; (2) subcutaneous injuries of the gall-tracts; (3) penetrating wounds from within outwards (operative)

Penetrating Wounds of the Gall-Tracts.

Penetrating wounds of the gall-tracts have been recorded for a number of years, still the number of cases reported is extremely small. They may be divided into gunshot and stab wounds, and the cases reported are equally divided between the two. This is somewhat surprising, as the percentage of perforations of the intestine in gunshot wounds so enormously exceeds the stab perforations in an equal number of penetrating wounds of the abdominal parietes, for the intestine appears to escape from the point of the knife and is rarely involved, but the gall-bladder, being firmly anchored to the liver, is readily penetrated. The course of the case after the perforation depends not so much upon the size of the opening in the gall-bladder

s upon the presence or absence of infection. The mere penetration f the gall-tract with the escape of bile into the peritoneal cavity is ot in itself a grave condition. This has been shown clinically as ell as experimentally, that is, the escape of bile into the peritoneal avity without the admission of infectious material does not as a rule rminate fatally. The wound in the gall-bladder, if there be no in-ction, contracts, cicatrizes, and closes, shutting off the communi-ation between the peritoneum and the gall-bladder and the bile at has escaped into the peritoneal cavity is absorbed. Numerous xperiments support this statement, as also do two of the chole-ystectomies of Courvoisier. Aseptic bile does not produce peri-nitis. Deaths following penetrating wounds of the gall-tracts are aused by the extent of the injury, shock, laceration of the liver and rrounding tissues, and hemorrhage, and not by the admission of bile to the peritoneal cavity. In the deaths from infection the sepsis from without and not from within, unless previous disease of the all-tract existed.

Injuries to the ducts are usually associated with injuries to the ssels—the portal or hepatic veins or the hepatic artery. If the ortal vein is wounded it must not be ligated; the opening must be tured. Ligature of the portal vein terminated fatally in all of Lé-ne's experiments. President Carnot's death was the result of a emorrhage from a wound in the portal vein. Biffett reports a case fatal hæmatemesis from ulceration of the gall-bladder into the duo-enum, and Schmidt a fatal hemorrhage from an aneurysm of the he-atic artery produced by cholelithiasis. Bullet wound in the apex as been recorded in the practice of Hans Kehr, and a rupture of the all-bladder with secondary drainage and removal in the practice of hiersch. When the ducts have ruptured into the lesser peritoneal vity, the accumulation may be drained by making an opening rough the posterior wall of the abdomen over the apex of the right dney, as described by Rutherford Morrison.

Subcutaneous Injuries of the Gall-Tracts.

The following are among the most frequent causes: falling flat on the abdomen, being forcibly driven against a sharp object, kicks the right hypochondrium, being run over by heavily loaded wagons, d forcible contraction of the abdominal muscles as in parturition. he most frequent seat of rupture is the bladder, and then, in the der mentioned, the choledochus, hepaticus, and cysticus. Rupture favored by the presence of gall-stones.

The immediate *symptoms* of rupture of the gall-bladder are only ose common to severe contusions of the abdomen, namely, collapse,

blanched countenance, feeble pulse, sighing respiration, nausea and vomiting, cold and clammy skin, and pale mucosæ. If the patient survives the immediate effects of the traumatism, icterus may appear, its presence or absence depending upon the quantity of bile discharged into the peritoneal cavity and the rapidity of its absorption. It is not a constant symptom. Encapsulation may occur, and if it does absorption will be slow and icterus of a pronounced character will not be present. The presence or absence of fever and septic symptoms depends upon the presence or absence of infection, for aseptic bile in the peritoneal cavity does not produce a marked elevation of temperature. The constant discharge of bile into the peritoneal cavity, that is, without limiting adhesions, will in time produce cholæmia unless the channel to the intestine is intact. These patients live many weeks and even months with the free escape of bile into the peritoneal cavity, but they finally emaciate, become cachectic, and succumb. Acholic stools are present only when the entire quantity of bile is excluded from the intestinal tract.

It is difficult to make a *diagnosis* of subcutaneous rupture of the gall-tracts. A circumscribed area of superficial dulness extending down in the direction of the right iliac fossa, local pain, and very marked reflex cardiac symptoms (angina) are suggestive of this condition. A positive diagnosis can be made only by aspiration or an exploratory incision.

The *prognosis* in rupture of the gall-tracts is very grave, as the injury producing it in a large number of cases involves other organs, and collapse is a frequent cause of death. When the patients survive the collapse there is danger of peritonitis, cholæmia, and marasmus. About sixty-five per cent. of the cases terminate fatally.

Penetrating Wounds from Within.

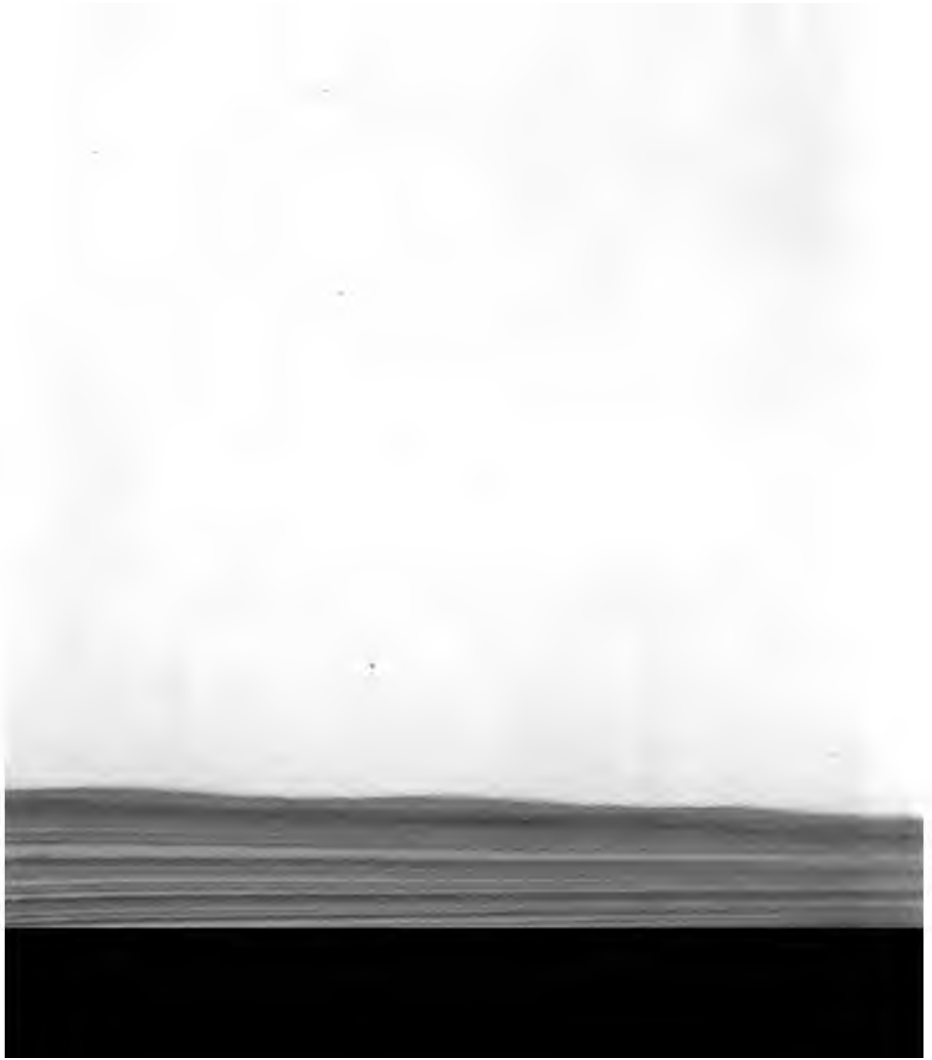
Since operations upon the gall-tracts have become common a number of accidental perforations of the gall-bladder and cystic and common ducts have occurred as the result. Great care should therefore be exercised, during the second part of a cholecystostomy, in extirpating large calculi impacted in a contracted gall-bladder, as well as in the extraction of calculi from the cystic or common duct. Cholelithotripsy is a frequent cause of primary or secondary perforation. Care must be exercised in exploring the gall-tracts after the gall-stones have been removed, as in many cases the wall is weakened by a pressure atrophy produced by the stones. There is danger of tearing the adhesions that secure the gall-bladder to the abdominal wall in the second sitting of cholecystostomy. When rupture occurs under these circumstances we have the same dangers that are present with per-

orations of the gall-tracts, that is, escape of bile and development of epsis.

Treatment of Injuries to the Gall-Tracts.

In contused as well as in penetrating wounds of the abdomen, exploratory laparotomy is now much more frequently performed than in former years. So multiple are the injuries and so fatal the sequences of severe abdominal contusions as well as penetrations, that it is rapidly becoming the practice in the great hospitals to make an immediate exploratory incision in the former, as it is now the accepted practice in the latter. We shall therefore, in the future, have better results in the immediate treatment of punctures and ruptures of the gall-tracts. If the gall-bladder be ruptured the indication is immediate suture; the same is true of the ducts. This suture should always be accompanied by drainage as a precautionary measure. Should union fail to take place in consequence of defective coaptation or of sepsis, the patient is protected by the drainage. When the cystic duct has been completely severed cholecystectomy is indicated and invagination with a Czerny-Lembert suture with drainage should be made as the operation for cholecystostomy under other conditions. If the common duct be completely divided the ends should be closed and immediate cholecystenterostomy performed. When the gall-bladder has been ruptured for several days and circumscribed encapsulation and accumulations of bile are found, the primary indication is drainage without any attempt to locate the seat of rupture. Clinical experience shows that small openings in the tracts close without any assistance from the surgeon, therefore drainage is all that is indicated. If a quantity of bile continues to escape through the external fistula, an operation to divert it into the intestinal tract must be performed. It is preferable to conduct it into the duodenum, although it is compatible with life if it escapes into the colon. If gall-stones be encountered they should be removed at the time of operation. Abuthnot Lane recently reported a case in which a wagon passed over the abdomen, and the gall-bladder was ruptured. Drainage was made and the patient recovered.

In the preparation of this article I have adhered closely to the classification of Courvoisier, as it is based upon the pathological conditions, and his masterly dissertation "Pathologie und Chirurgie der Gallenwege" has been freely used. Naunyn's excellent classification of gall-stones has been followed. I am also indebted to the works of McFadden Gaston, Riedel, Führbringer, Mayo Robson, Martig, and Brockbank.



MOVABLE KIDNEY.

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MOVABLE KIDNEY.

WITHIN quite recent years the medical mind has slowly but certainly become convinced of two facts: first, that mobility of the kidneys is of very frequent occurrence, and second, that it cannot be looked upon as merely a pathological curiosity, but that it is responsible for groups of symptoms which may cause, on the one hand, the most trivial inconvenience or, on the other hand, may be of such intensity as to make life "nothing but an endless torture."[1] Professor Keen writes:[2] "The discomforts are very great and the pain may be so excessively severe and prolonged as to interfere with all occupation, and practically to make life almost unendurable. The disorder may pass beyond the realm of bearable evils into serious and actual danger to life itself, so that in considering the slight mortality from nephrorraphy, we must also bear in mind that there is a mortality attending the expectant treatment as well."

FREQUENCY.

It has long been admitted that a kidney may be displaced or dislocated, but such an occurrence was looked upon as very exceptional. For many years a diagnosis of wandering kidney was treated with scepticism, and the sceptics justified their unbelief on the ground that a movable kidney was very rarely seen in the dissecting-room or in the pathological laboratory. In 11,000 autopsies in three hospitals, quoted by Newman,[3] only 11 cases were found, or but 1 in 1000. In the clinic of Oppolzer, given in Rollet's statistics, there were 22 cases in 5,500 autopsies, or 1 in 250; Ebstein states that in 1658 post-mortem examinations in the Charité at Berlin, a movable kidney was found only five times, or 1 in 731 autopsies.

The infrequency with which mobility of the kidney is discovered after death is mainly due to the more or less post-mortem fixation which takes place. The kidney in health is surrounded by a fatty capsule. In very spare people, or those emaciated by disease, this capsule may be of the scantiest proportions; while in those on whom nature has bestowed large quantities of adipose tissue, the fatty covering of the kidney is abundant in proportion. It is very

generally believed that this fatty envelope contributes largely to the fixation of the kidney; but this is not the case, at least not to any very appreciable extent. The postperitoneal space in the loin in which the kidney lies may contain in well-nourished people a considerable packet of fat which acts like a cushion round the kidney. This cushion of fat by its presence increases the size of the space behind the peritoneum. Any cause which brings about emaciation of the patient will remove the fat, leaving the cavity undiminished in size. It is obvious that this will materially aid in bringing about mobility of the organ, if other conditions are at the same time favorable. In subjects who have always been spare the cavity is normally sufficient to contain the kidney, and the kidney alone, and such subjects are not more liable in my experience of renal mobility than the well-nourished.

It is important to bear in mind, however, that the fat in the body is of a very different consistence during life than it is after death. In health it is more of a semi-fluid, almost jelly-like consistence, and can act to a very slight extent, if at all, in restraining a kidney which is mobile. But after death the fat solidifies, and will serve to fix a kidney which during life was movable. A patient generally occupies the recumbent position for days, weeks, or months previous to death. If the kidney happen to be movable, it naturally gravitates in the dorsal decubitus into its normal situation in the loin, and becomes fixed there by the solidifying fat. It thus happens that in the dissecting-room, or during post-mortem investigations, the evidence of ante-mortem mobility of the kidney is very easily overlooked, especially if the investigator is not particularly interested in the question of mobility of the renal organs.

Of recent years the frequency with which a wandering kidney has been found during an abdominal operation, or the diagnosis of such has been confirmed by an incision in the loin, has convinced medical men generally that renal mobility is a fact of the very greatest importance. In consequence of this, the presence of a train of symptoms, which formerly were erroneously referred to the stomach, the intestines, the gall-bladder, or the uterus and its appendages, has led to a more frequent examination of the loin, and the existence of a wandering kidney has been discovered so often as to prove clearly that its occurrence is much more common than it was formerly supposed to be. Skorczewsky states that in 1,422 patients whom he examined during life, 32 females out of 1,030 and 3 males out of 392 were the subjects of mobility of the kidney. Among the poor of Austria, according to Oser of Vienna, 10 per cent. of the women who have borne children are affected. These statistics show that in from 3 to 10 per cent. of wo-

nen the kidney is found mobile. It is otherwise in men. In a table of 249 cases compiled by A. W. Foot,[4] there were 215 women to 34 men affected, a proportion of about 6 women to 1 man. In 290 cases collected by Newman the sex is given. Of these, 252 were women and 38 men, a proportion of 6.6 to 1. In Keen's table of cases of nephrorrhaphy for movable kidney,[2] the sex is reported in 99 cases. Of these, 93 were women and 6 men—a proportion of 15.5 to 1. The excessive proportion in Keen's table would seem to indicate that women suffer more from a movable kidney than men, or that they are more ready to submit to an operation for relief.

In considering the relative frequency of mobility of the two kidneys, it is remarkable how much more common it is on the right than on the left side. Ebstein gives 4.5 to 1 as the ratio; but this seems too small. In Landau's tables[5] we find movable kidney 151 times on the right to 13 on the left—a proportion of 11.6 to 1. In fourteen cases Landau found it bilateral. In Keen's tables of nephrorrhaphy the side is reported in 87 cases; of these 76 were right and 7 left, a proportion of very nearly 11 to 1. It was found on both sides in four cases.

We may summarize thus: 1. Mobility of the kidney is more frequent than is generally supposed (from 3 to 10 per cent. of adult women); 2. It is found more frequently in women than in men (about 6 to 1); 3. It is more common on the right than on the left side (about 11 to 1).

DEFINITION.

There are two distinct forms of wandering kidney, the congenital and the acquired. The congenital is properly termed a *floating* kidney. It is a kidney which is freely movable within the cavity of the abdomen, is completely surrounded by peritoneum, and is possessed of a mesonephron. This is a very rare condition. Thus Sir William Jenner says in his clinical lectures on the diagnosis of extra-pelvic tumors of the abdomen: "A movable kidney is one thing, a floating kidney is another. We very rarely see or feel a floating kidney. I have never met with one after death, though I have felt in a patient what has been supposed to be one." Mr. Lawson Tait is more dogmatic. In the *British Medical Journal* for November, 1882, in reference to a case "pronounced to be a 'floating kidney' by several distinguished authorities," but which proved at the time of operation to be a distended gall-bladder containing a large number of gall-stones, he says: "I put the floating kidney altogether on one side; besides I have never seen such a thing, either in life or in a museum, nor have I met any one who has. In fact, I have no belief in its existence as a

pathological incident." Such a case, however, came under my own observation in October, 1892, and was subsequently published in the *Dublin Journal of Medical Science.*[6] The abdomen had been opened in the right linea semilunaris. The right kidney "appeared a little more crimpled than usual, but otherwise normal, except for its surroundings. It was totally devoid of its usual fatty capsule, and instead of this was accurately covered all round with peritoneum. The peritoneum was reflected at the hilum on to the vessels and ureter until it reached the right side of the spine, where it became continuous with the posterior parietal layer."

The true floating kidney is a pathological curiosity. Its rarity makes it of little clinical importance. Diagnostically it is practically indistinguishable from the movable kidney, which is an acquired condition.

In movable kidney the organ is mobile behind the peritoneum, either within its adipose capsule or in a sac formed between the peritoneum in front and the muscular wall of the abdomen behind. The area of mobility may be very great; the peritoneum may be detached over a large surface, and be so flaccid as to afford as great a freedom of movement to the kidney as though this organ possessed a long and loose mesonephron. In the report of the committee of the Pathological Society of London,[7] Dr. Bindley's case is mentioned, in which the kidney was able to move under the peritoneum over a space described as a circle, having a diameter of eight or nine inches. Thus a movable kidney may fall as low as the brim of the true pelvis, or may lie immediately beneath the anterior abdominal wall, or may cross the spinal column to the opposite side.

When we speak of a movable kidney, we must bear in mind that normally the kidney enjoys a certain amount of mobility—that is, it can and does move within certain limits; thus it moves downwards on inspiration and rises again on expiration, and this normal movement may sometimes be so free and be so easily felt as to raise a doubt as to whether the movement is normal or abnormal. This kind of mobility we may call *physiological mobility* of the kidney. A kidney *pathologically movable* exhibits generally a much wider range of motion; but we may find every degree of mobility, from the most extreme, when the organ may fall into the pelvis, to the slightest, when its motion may with difficulty be detected. We shall presently see that there are anatomical conditions which mark a very distinct line between the physiological and the pathological mobility of the kidney. For clinical purposes we may consider the condition pathological when by a little dexterity the fingers can be insinuated between the eleventh and twelfth ribs above, and the upper margin

f the kidney below. The easiest and best method of detecting a athologically mobile kidney is to place the patient in the dorsal po-ition, with the head and shoulders slightly raised and supported on illows. The examiner standing on the right side, the four fingers f his left hand are passed underneath the hollow of the loin below ıe twelfth rib. The thumb in front encircles the abdomen just below ıe costal arch, but without exercising any pressure. The patient is ıen directed to draw a full breath. Immediately before expiration egins the thumb is pressed upwards beneath the costal arch, and is lowed to sink in as deeply as possible, following the liver as it re-ides during expiration, while the fingers behind press the loin for-ards. If now the right hand can feel the kidney lying entirely below e grasp of the left hand, that kidney is pathologically movable. If e right hand presses up the tumor so felt, while the left hand re-xes its grasp gradually, the tumor can be felt to slip between the ıgers of the left hand and to disappear upwards into the position rmally occupied by the kidney. This sensation is pathognomonic a movable kidney. Sometimes the kidney does not leave its bed adily by this method. I have then found the following manœuvre eful: The patient is asked to sit up in bed; the elbows, held firmly the sides, are grasped and the patient's body is raised about a ot from the bed; the body is brought down again with a jolt. ıis will generally dislocate a movable kidney, which can then be sily felt in the loin or in the iliac fossa.

ETIOLOGY.

A great many theories have from time to time been promulgated explain the causation of movable kidney. Before, however, these ories can be discussed, we should consider the means which nature provided for keeping these organs in their normal position. We the kidney surrounded on all sides by a fatty capsule and by nective tissue. We have already discussed the importance of the y capsule as a means of keeping the kidney in its place, which has n very much exaggerated. Of more importance is the connective ue which binds the kidney to the loin behind and to the various lominal viscera above, below, and in front. In the latter position s assisted in no inconsiderable degree by the peritoneum reflected r its anterior surface. In probably a majority of cases these con-tive-tissue adhesions and the disposition of the peritoneum are icient to maintain the kidney in its place for a time, even when ses operate which would dislocate an organ with less firm connec-ıs. These connections are not in themselves sufficient to with-

stand alone the weight of the kidney, but they are important to bear in mind from another point of view, for, though they may not keep a kidney in its place when once it has become dislocated, they are responsible for many of the symptoms which accompany movable kidney and which may make life unendurable.

Fig. 52. Fig. 53.

Fig. 52.—Anterior Surface of the Right Kidney. S.S., Impression for suprarenal capsule; S.H., hepatic impression; S.C., colic impression.

Fig. 53.—Anterior Surface of the Left Kidney. Lettering same as in Fig. 54. (From a photograph by Professor Cunningham.)

What, then, is the main factor which keeps the kidney in its proper place?

The researches of His and of Cunningham have clearly shown that the form of the kidney itself and the influences exerted upon it during life, or rather in the unopened body, vary very materially from what is usually observed in the dissecting-room or on the post-mortem table. The method employed lately by Professor Cunningham with the view of obtaining an accurate knowledge of the true form of the solid viscera of the abdomen was briefly this: Having selected two well-formed subjects, he proceeded to harden them by the injection method, employing Müller's fluid, followed by graduated spirit injections. In the case of one of the subjects, the injections were repeated almost daily for a period of two weeks, while in the other the hardening process was continued for fully six weeks. By this method all the internal organs were hardened *in situ* before any disturbing influence had been introduced by opening the abdomen.

As regards the kidney, and with the kidney we are now alone concerned, the results have been most remarkable. The kidneys present many slight changes in form, according to the amount and the kind of pressure which is exerted upon them by contiguous viscera; and these changes undoubtedly are continually taking place in the same individual. We should fix our attention, however, on the more constant conditions of forms which the accompanying illustrations clearly show.

Professor Cunningham thus describes them:* "In every case and on both sides there is on the anterior surface a point of maximum convexity, a place where the kidney substance is raised in the form

f a marked prominence or bulging, which may slowly rise from all ides to a blunt summit, as is usually the case in the left kidney, or hich may extend across the anterior surface in the form of a rounded idge, as happens more commonly in the case of the right kidney. bove and below this eminence the anterior surface falls away wards each extremity in the form of an inclined or sloping plane of reater or less obliquity (see Fig. 52). These impressed districts in-icate pressure exercised on the anterior surface of the kidney in two irections, and the intervening eminence is the result of this counter-ressure. Upon the upper inclined plane of the anterior surface of ie left kidney are placed the suprarenal capsule, the stomach, and ie spleen (see Fig. 53). These exercise a downward and a back-ard pressure. Upon the inferior inclined surface of the left kidney ie counter-pressure is produced by the intestinal canal, which presses a rule upwards and backwards (see Figs. 53 and 54). On the ght side the upper inclined surface is occupied by the liver, while contact with the lower inclined area is the colon. In many cases e colic or inferior sloping surface esents a high degree of obli-uity" (see Fig. 52).

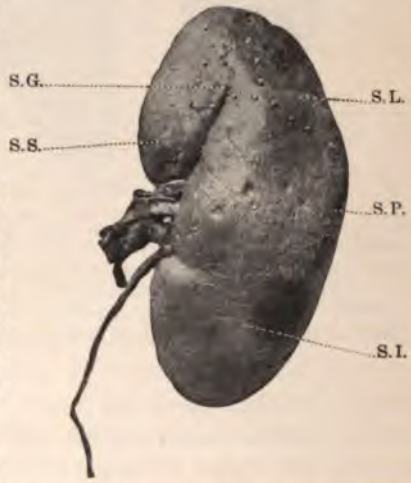


Fig. 54.—Anterior Surface of the Left Kidney. S.S., Impression for suprarenal capsule; S.G., area in direct contact with stomach; S.L., impression for the spleen; S.P., impression for the pancreas; S.I., inferior inclined surface, in contact with intestine. (From a photograph by Professor Cunningham.)

We see, then, that on the right le we have two forces acting on e kidney: one, acting from above, esses the kidney downwards and ckwards; the other, acting from low, presses the organ upwards d backwards. The resultant of ese forces will be a force acting nost directly backwards, wedg-g, as it were, the kidney into e position it usually occupies. w, the pressure from above practically constant. It varies newhat with the position of the ly, and also during respira-n. But these variations do not lly affect the question, for the er, kidney, and colon move ether under the circumstances, l the relative position of the forces is practically unaffected.

The pressure from below varies to a much greater degree with the ying conditions of the intestine itself. It diminishes when the vels are empty; it increases with flatulent distention. Still the

general direction of the pressure, upwards and backwards, on the inferior inclined plane of the kidney is maintained. The effect of these normal variations of the two forces is shown in the illustrations. In one kidney we find the transverse ridge on the anterior surfaces near the middle of the organ, in another we find the ridge is below the junction of the middle and lower third. Slight alterations in shape appear to be the main results of these frequent variations between the upper and lower compressing forces.

The effect of the two pressures, the one acting upwards and backwards and the other acting downwards and backwards, upon the shape of the kidney is very well illustrated in Fig. 55, which shows the appearance the kidneys present when placed edgeways, that is with the convex outer borders facing us, the hilum away from us.

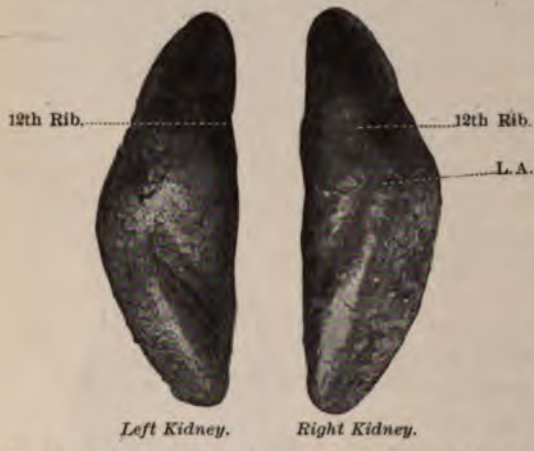


FIG. 55.—Outer Convex Borders of the Right and Left Kidneys, showing the wedge-shaped outline of the kidneys when viewed from this aspect; also the central prominence and the two inclined surfaces on the anterior aspect of each. L.A., Lig. arcuatum externum groove on the right kidney. (From a photograph by Professor Cunningham.)

I have seen this wedge-shaped appearance of the kidney in an ordinary dissecting-room subject, in which no special precautions had been taken to harden the organs *in situ*. The abdominal viscera had all been removed with the exception of the left kidney, which had not been disturbed. The superior and inferior inclined planes were marked in a high degree, and the appearance of the central prominence was accentuated by the disposition of the peritoneum; for, as the latter crossed over this eminence to be reflected on to the internal aspect of the ribs, it was thrown into a distinct fold, almost looking as if a string lay stretched beneath it from the kidney to the ribs.

Now if the balance between these forces be lost, so that their resultant acts in a more or less downward direction, it is easy to conceive how the kidney itself, aided by this downward force and by its own weight, would gradually sink, making a way for itself behind the peritoneum which lines the loin below it.

There are several ways in which this balance of pressure may be

destroyed. An obvious example is after parturition. We know that normally the pressure within the abdomen is positive, but immediately, and for some days at any rate, after parturition the pressure becomes negative. If during this period the patient assume the erect posture, the pressure from above acts downwards and backwards unopposed, and unless the connective tissue round the kidney is sufficiently and unusually firm, the kidney must gradually yield to the combined forces, and slip from its place. Each act of parturition, with shortened respites in bed, will increase this tendency, and it is difficult to understand how the kidney, having once slipped from the control of the colon, could ever again regain accurately its relative position, so as to be wedged between it and the liver, as we have seen it normally is. Probably a movable kidney, when it slips into place in the recumbent position, lies behind the colon and below the liver, its anterior surface being flattened, so that the wedging forces can no longer act. Another method by which the balance of power may be destroyed is by traumatism. If by means of a violent shock, such as a fall from a height on to the feet, or by means of extreme pressure, as the passage of a cart wheel across the body, the kidney be forced downwards out of its bed, the equilibrium of forces is disturbed, not to be again restored.

A very interesting example was laid before the Medical Society of the Royal College of Physicians in Ireland, in 1874, by Dr. W. B. Peebles.[9] A young married lady had a phaeton turned over upon her by a runaway horse. She was found underneath it, on her right side, insensible. She was kept in the horizontal position for a month. Two days after she was allowed up she was seized with symptoms of acute nephritis. Shortly afterwards she discovered what she called a "gizzard" loose in the abdomen, which followed the movements of the body. When it was near the stomach food passed in seemed to strike against it. When the upright position was assumed it fell downwards. It was tossed about by the movements of flatus in the bowels. There was "great agony" accompanying all these movements. The diagnosis made by those who saw her was a displaced kidney. It seemed probable that the edge of the vehicle, resting directly on the left side of the loin, had contused the deep parts against the spine, while a good deal of clothing and the cushion had saved the skin; that the kidney was then loosened in its bed, and that it dropped out afterwards when the upright position had been assumed. The diagnosis was confirmed a month later when suppuration occurred in the tumor, and pus was discharged with the urine per urethram. Recovery was subsequently complete.

At the same meeting of the Medical Society, Dr. Gerald Yeo re-

lated the case of a laboring man, aged forty-five, who had recovered from serious injuries caused by a cart passing over his loins. A year and a half after the accident his left kidney could be displaced by manipulation over the margin of the quadratus muscle, and could be seized through the abdominal wall as it lay under the ribs.

Morris[10] quotes from Henoch the case of a military officer who fell upon his feet from a horse, and suffered a violent concussion of his entire body; subsequently both his kidneys became movable.

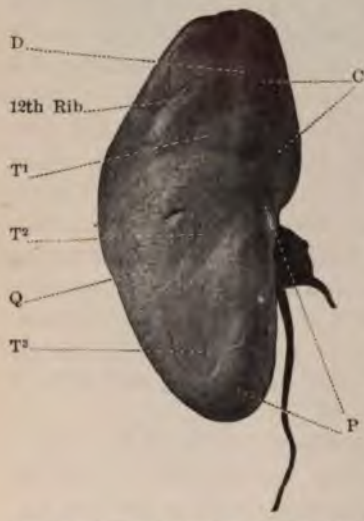


FIG. 56.—Posterior Surface of the Left Kidney. (Note the vertical ridge which separates the surface of the kidney into an internal and external district.) D., Surface in contact with the diaphragm; P., impression for the psoas; C., impression for the crus of the diaphragm; Q., impression for the quadratus lumborum; T^1, T^2, T^3, dimples corresponding to the tips of the transverse processes of the first, second, and third lumbar vertebræ.

It is not, however, always necessary that the traumatism should be severe or the shock violent. In some cases a comparatively slight jolt or fall seems to have been sufficient to induce the kidney to leave its bed and to pass from the control of the wedging forces. It is not always easy to get a history of such an accident, but such a history should be carefully sought for in the case of young women who have never borne children, and in whom no other cause can be discovered. I have met with several cases of this sort, and have generally been able to discover a forgotten trauma.

Besides the ridge and planes on the anterior surface there are other markings on the kidney which may be shortly alluded to. The duodenum almost invariably rests upon the anterior surface of the kidney, and it gives rise to a very evident duodenal impression. The outer convex border of the kidney of both sides presents a marked thickening opposite the eminence on the anterior surface. In the neighborhood of this thickening a faint groove passing upwards indicates the place where the border of the kidney is "clasped" by the last rib (see Figs. 55 and 56). This is worthy of notice, as it supports those who advocate fixing a movable kidney to the last rib. The posterior surface of the kidney exhibits three well-marked areas, which correspond respectively to the psoas internally, to the quadratus lumborum externally, and to the diaphragm above. Between the areas marked by the psoas and the quadratus lumborum muscles dimples correspond-

ing to the transverse processes of the lumbar vertebræ may sometimes be observed in spare subjects (see Fig. 56).

I have hitherto referred to the right kidney because dislocation and mobility are much more frequent on the right than on the left side. The proportion is about eleven to one. It will be understood that the arguments which have been adduced regarding the right kidney apply also to the left, taking into consideration the different relations on the left side.

It will be interesting to review the various theories which have from time to time been promulgated to explain the occurrence of movable kidney. Cruveilhier attributed this condition to the pressure exerted upon the liver by stays, this transmitted pressure driving the kidney out of its normal situation. This theory has met with considerable support, even from writers of the present day; but it will not explain the occurrence in men, nor in that large class of women, especially among the poor in whom mobility of the kidney is so common, who do not wear tight stays. Moreover, if we consider the position of greatest pressure in tight-lacing, we shall find it to be at the waist, that is, below the ribs. It is evident that if a kidney is sufficiently mobile to fall below the waist, tight-lacing will help to push it and to keep it down; but if the kidney occupies its normal position, its upper extremity embedded in the under surface of the liver, the stay pressure is exerted mainly below it, and will tend rather to keep it up in its place than to aid its dislocation.

Lancereaux explains its occurrence by the physiological and pathological relation between the kidneys and the generative organs. Guttmann,[11] following this up, calls attention to the congestion of the kidneys which accompanies the menstrual epochs. These organs, he says, tumefy, become heavier, and have a tendency to leave their normal position and to descend little by little into the abdomen. But even in women this will not always explain the occurrence of renal mobility—as, for instance, in the case reported by Guyon,[12] in which a movable kidney, accompanied by acute pain, appeared for the first time in a woman, aged fifty-four, a year after the menopause.

Newman offers the following explanation: "The posterior surface of the right kidney is practically in contact with the crura of the diaphragm and the great lumbar muscles, while interposed between its anterior surface and the anterior abdominal wall there is a large solid organ—the liver. Now suppose the patient strains the abdominal muscles, as during delivery or in lifting a heavy weight, both the anterior and posterior abdominal muscles are brought into powerful action, the liver being pressed backwards by the anterior muscles,

the kidney pressed downwards by the crura of the diaphragm, and forwards by the posterior abdominal muscles. Now the resultant of these forces will act in a manner similar to what takes place when one presses a bean between the finger and thumb—that is to say, the kidney will be pressed downwards."

This ingenious theory must be rejected on two grounds. In the first place, although women experience great abdominal strain during delivery, yet in lifting heavy weights and in work involving severe strains on the abdominal muscles, men are much more liable to injury than women. If this were the cause of mobility in the kidney we should find it much more commonly in men than we do, and perhaps even more frequently in men than in women. In the second place, the anatomical arrangement of the muscles, as shown by the impressions seen on the kidney when the organ is hardened *in situ*, disproves this theory. If we examine the posterior surface of the kidney (Fig. 56) we see in the lower half of the kidney the impression for the psoas muscle on its inner side runs downwards and outwards, while the impression for the quadratus lumborum runs downwards and inwards; these two impressions practically meet at the inferior apex and divaricate upwards. Now, when these muscles and the abdominal muscles in front are thrown into violent action, they may, as Newman says, "act in a manner similar to what takes place when one presses a bean between the finger and thumb"—that is to say, they may force the kidney upwards, and not downwards, as supposed by Newman. The real effect of this muscular contraction is, I believe, quite different. In the living body the liver is not the solid organ which it appears to be on the dissecting-room table, but partakes much more of the quality of a sponge; and in a hollow, on its under surface, well seen in the liver hardened *in situ*, lies the kidney. Strong muscular contraction serves in reality to embed the kidney the deeper into the substance of the liver.

Albarran[13] has recently propounded another theory, namely, that movable kidney is an evidence of nervous degeneration, and he quotes a number of cases of mobility of the renal organs associated with neurasthenia, hypochondriasis, perversion of the sexual instinct, imbecility, etc. It is of course possible that the presence of an abdominal tumor, or continuous suffering caused by it may so affect the patient as to render her neurasthenic, but the neurotic symptoms will be the effect and not the cause. In my own experience the large majority of the cases I have met have been singularly free from any such symptoms. We need not go so far afield in search of a cause upon which the more recent anatomical researches have thrown so much light.

The right kidney, as we have seen, is much more liable to dislocation than the left. The proportion is eleven times on the right to one on the left. This must be explained on purely anatomical grounds. Landau attributes the different behavior of the right and left kidneys to the difference in their mode of attachment, to the difference between the hepatic and the splenic flexures of the colon, to the position of the left renal vessels above the horizontal part of the duodenum, to the shortness of the left renal artery, and to the extra support the left kidney gets from the attachment of its vessels to the pancreas.

More important still is the position of the tail of the pancreas itself. If we examine Figs. 53 and 54, we shall see that the pancreas forms a depression on the anterior surface of the left kidney, and this depression lies, as is well seen in Fig. 53, just below the eminence on this surface of the kidney. This position of the pancreas and its firm attachments have no doubt a powerful influence in keeping the left kidney in its place. Moreover, we must bear in mind that the pressure exerted by the stomach in a downward direction is not comparable with that exerted by the liver (see also Fig. 57).

Symptoms.

The symptoms produced by movable kidney are many and various. They may practically be *nil*, and some accident alone reveals the presence of a movable tumor within the abdomen; or, on the other hand, to quote Le Dentu,[1] "the suffering is sometimes so acute that life becomes nothing but an endless torture." The multiplicity and variety of these symptoms require that they should be grouped and reduced to some sort of order if we would rightly interpret them. For convenience they may be divided into three groups: 1. Those symptoms which are common to both right and left movable kidney; 2. Those symptoms which belong exclusively to mobility of the right kidney; 3. Those symptoms which may be found when the left kidney is mobile.

In the first group are comprised those symptoms which indicate the effect produced upon its vessels and nerves, the ureter, or itself by the falling of the kidney, whether right or left. There can be no doubt that one of the results of mobility of the kidney is elongation of the vessels and nerves which supply it. Every surgeon accustomed to operate for movable kidney must have observed the facility with which the kidney can be brought, not only up to the wound, but sometimes outside the body altogether; this is a good indication of the abnormal length of the renal pedicle. This lengthening of the pedicle cannot

take place without a prolonged "dragging" upon the vessels and nerves. The nervous supply of the kidney is very great, and any traction upon or interference with it must give rise to much pain and to some reflex troubles. While admitting this, we must be careful not to attribute all the phenomena of movable kidney to this one cause and to hold the already overburdened "nervous system" responsible for symptoms which we shall presently see are referable to other causes altogether. As results of this dragging upon the renal vessels and nerves we may enumerate a dragging pain in the abdomen and a sense of pain in one or the other side, neuralgic pains in the loin or shooting down the thigh, fatigue on the slightest exertion, and general debility which makes any effort a "weariness to the flesh."

In addition to the symptoms referable to the renal plexus of nerves, we have in the same group symptoms due to torsion of the blood-vessels and consequent congestion of the kidney; these symptoms are much more severe and serious. When this accident occurs we find severe pain in the loin, generally coming on suddenly and recurring in paroxysms; suppression of urine without dilatation of the pelvis of the kidney, showing that the urine is not secreted, or secreted in small quantities; what urine escapes is of a high specific gravity and contains albumin and tube casts. When the torsion of the vessels is relieved the symptoms subside, the albumin and tube casts disappearing from the urine. It is clear that such a train of symptoms cannot be attributed to traction on the nerves, or to any reflex consequences of such traction. This accident may sometimes result from some effort, or from driving in a jolting vehicle or over a rough road, or it may result from the mild exercise of walking. It is ushered in by violent pain in the flank, shooting in various directions, accompanied by vomiting, and a pinched, agonized expression of face, in fact symptoms closely simulating those of nephritic colic.

Symptoms equally severe and in many respects closely resembling the above indicate another accident which may occur in a case of movable kidney, namely, twisting, kinking, or compression of the ureter. This condition is chiefly characterized by intermittent hydronephrosis. In addition to the other symptoms resembling renal colic, during the paroxysm there is usually scanty urine or even anuria, due to reflex inhibition of the other kidney. The affected kidney distends till a fluctuating tumor can be felt in the abdomen. The cessation of the attack is ushered in by a copious flow of urine, which is followed by a disappearance of the fluctuating tumor. The urine may contain a transitory amount of albumin, but this is not constant. The presence of a distinct amount of albumin, accompanied by tube casts, in a case of intermittent hydronephrosis indicates that in addi-

ion to the kinking of the ureter the renal vessels are at the same ime twisted. It is remarkable that sometimes a kidney is pos-essed of extreme mobility, and yet hydronephrosis does not occur, vhile sometimes with a much less degree of mobility intermitting ttacks of hydronephrosis are continually recurring. The explana-ion of this lies, I believe, in the degree of fixation of the ureter. If he ureter be comparatively fixed, the kidney in its descent cannot lraw the ureter with it, and kinking or compression of the latter eadily takes place, but if the ureter enjoys a certain amount of mo-ility as well as the kidney, the kidney carries its tube with it, and hus kinking is avoided. An interesting case of the former was laid efore the French Surgical Congress by Albarran. He operated on a atient during the attack of ureteral strangulation, and found that the idney was retroverted, causing a well-marked kinking of the ureter.

I have myself observed a very interesting instance of extreme mo-ility of the ureter in a case of movable kidney in which no symp-oms of hydronephrosis or of any urinary trouble were at any period bservable. It was the case of a young lady who had suffered more r less for several years from a right movable kidney. The kidney as very easily felt, lying in general below a horizontal line drawn hrough the umbilicus. She suffered from gastric troubles, indiges-on, and gradually increasing emaciation. She never had hydrone-hrosis or any derangement of the urinary organs. At the opera-on I found the ureter almost completely surrounding the kidney. passed from the hilum transversely across the anterior surface of ie kidney, round the outer convex border and then in an oblique di-ction downwards and inwards behind the kidney. It was unwound id replaced in its normal position, and nephrorrhaphy was per-rmed with most satisfactory results. The ureter, though thus risted round a very movable kidney, was not in any way occluded.

In addition to the symptoms enumerated above, as belonging to e first group there are other symptoms which call for mention—such e the neuralgic pains in the loins, starting down the thigh or along e groin of the affected side. Defontaine has reported the case of a tient in whom pressure over the displaced kidney occasioned an mediate echo at the meatus and a sense of weight in the scrotum; d Le Dentu mentions the case of a lady who complained of a nsation at the neck of the bladder the moment he laid his hand over e site of a dislocated kidney. For diagnostic purposes it is well bear in mind another characteristic of the sufferings induced by vable kidney. In a large number of cases the symptoms become re accentuated and severe immediately before and during the cata-nial periods.

We may now take into consideration the second group of symp-

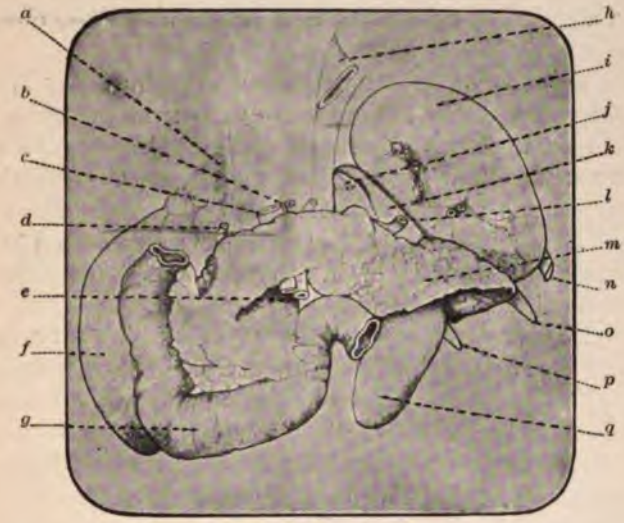
toms, or those which belong exclusively to mobility of the right kidney. This group comprises those symptoms which we recognize in their severer form under the term "the gastric crises." In the milder form the chief complaint is of indigestion, manifested by flatulence and sometimes vomiting. The following points can generally be elicited: The indigestion does not depend on the kind of food taken; one patient told me she had made the round of all sorts of food, and that nothing made any difference. Pain generally comes on about one or two hours after the ingestion of food, and large quantities of gas are frequently belched up. The usual remedies for indigestion and flatulence are of little if any use. I have frequently been told that the only relief to be obtained was by lying down, and one lady informed me that she always found ease by kneading her right side. Such a train of symptoms should always make us suspect a movable kidney on the right side. Now these symptoms can be explained on purely mechanical grounds. In October, 1892, I opened the abdomen of a woman who had what was diagnosed to be a most abnormally movable kidney. The opening was made in the right linea semilunaris, and the kidney was found lying in the right iliac fossa. The cause of all the symptoms was at once obvious. The kidney was attached by strong bands to the descending portion of the duodenum, and when the kidney fell towards the iliac fossa, it so dragged upon the duodenum as to kink it, and it was obvious that in that state nothing could pass out of the stomach. The stomach was in a state of dilatation. The symptoms from which the patient suffered and which she herself described as agonizing, disappeared when the kidney was fixed into its proper position.

Now, if we examine the normal relation of the descending portion of the duodenum to the kidney, we find that it generally lies on the inner portion of the anterior surface; sometimes, as shown in Fig. 57, it covers more than half the surface of the kidney, but this is an unusual amount. Occasionally, though very rarely, it does not touch the kidney at all, but lies on the renal vessels and the commencement of the ureter. The kidney and the vertical portion of the duodenum are bound together by connective tissue, which may sometimes be extremely lax and weak, sometimes may have an appreciable degree of strength. Both organs are retroperitoneal.

Now what occurs is this. In many cases when the kidney slips, its attachment to the duodenum may be so slight and lax, or the surface in contact with the duodenum may be so small, that the kidney disengages itself from the duodenum, and may become freely movable in the abdomen without in any way interfering with the gut. Under such circumstances we have, what is a not uncommon experience, a

reely movable kidney presenting no symptoms—a condition very lifficult to explain on the usual theories.

If, on the contrary, the surface of contact between the kidney and luodenum be extensive, when the kidney loses its usual support and begins to descend behind the peritoneum, it gradually drags the duodenum with it, and, as in the instance in which I had the good fortune to observe what occurred, the duodenum kinks, and a temporary condition is induced very similar in its symptoms to what we find in stricture of the pyloric end of the stomach. This view is further



G. 57.—Relations of the Kidneys to the Other Abdominal Organs. *a*, Right suprarenal capsule; *b*, hepatic artery; *c*, portal vein; *d*, bile duct; *e*, superior mesenteric vessel; *f*, right kidney; *g*, duodenum; *h*, œsophagus; *i*, spleen; *j*, left suprarenal capsule; *k*, left kidney; *l*, splenic artery; *m*, pancreas; *n*, tenth rib; *o*, eleventh rib; *p*, twelfth rib; *q*, left kidney. Reproduced from the *Journal of Anatomy and Physiology*, July, 1895.

rengthened by the fact that marked dilatation of the stomach is ound in some of these cases. Ever since I observed this case I ave made a point of examining as carefully as possible, whenever I as engaged in performing the operation of nephrorrhaphy, the atchment of the kidney to the duodenum. When the kidney has been ripped of its fatty capsule, and is being drawn up into the wound, the fingers of the left hand be passed behind it, it is quite easy, as very operator knows, to make out the beginning of the ureter and e renal vessels. With equal facility, in cases of movable kidney ith gastric symptoms, I have found that the duodenal attachment n be made out. A band of more or less dense connective tissue ill be felt extending from the internal border of the kidney, above

the hilum inwards and upwards. By following this band inwards, especially if some traction be made on the kidney, the duodenum can be easily felt, indeed sometimes it can be distinctly seen at the bottom of the wound, and some idea of the extent to which it suffers from traction can be obtained. It should be observed that these gastric symptoms or crises differ very materially from the symptoms usually recognized as evidence of renal colic. The acute symptoms which have already been described, as indicating, in a case of movable kidney, that the vessels have been twisted or the ureter kinked, are very different from the symptoms above described, which point to duodenal obstruction and closely resemble those observed in cases of stricture of the pylorus.

These symptoms are not found when the left kidney is alone at fault. Dislocation of the kidney is very much more uncommon on the left than on the right side; it is difficult, therefore, to speak of the symptoms produced by a kidney mobile on the left side with as much confidence as we can of the symptoms produced on the right side. But this I can affirm: I have never seen the gastric crises, nor anything like them, in any case of left movable kidney which has come under my notice.

In the third group of symptoms are those which may be found when the left kidney is mobile. If we examine the relations on the left side, at first sight there does not appear to be any reason why the left kidney, slipping out of its place, should interfere with any organ in its neighborhood. But I have had reason to change my view regarding this, owing to a circumstance which has still further convinced me that the causation of the symptoms produced by right movable kidney is as I have stated above.

In March, 1894, I saw in consultation a lady who suffered from extreme debility, inability to walk, caused by a feeling of weight in the abdomen, complete exhaustion after any attempt at exertion, distention of the abdomen, and aggravated constipation. She had been ailing since the beginning of 1892. Castor oil and turpentine enemata gave her no relief, and large doses of cascara, or of castor oil very frequently were unavailing. On examination I found she had both right and left kidney movable, the left most marked. An abdominal belt and massage improved her condition considerably, especially as regards the constipation; but as her progress was not altogether satisfactory, I performed nephrorrhaphy on the left side on April 10th, 1895. At the operation I found a very interesting state of affairs. The kidney was firmly fixed to the splenic flexure of the colon and as it fell downwards it carried the colon with it, so that it became kinked, forming a V bend. That this was the cause of the intractable constipation was proved by the sequel. The wound in the loin healed without suppuration, but I kept the patient lying in bed

for six weeks in order to allow the adhesion the kidney had contracted to the loin to become firm. When she began to go about she rapidly regained strength, the constipation entirely disappeared, and she was able to go about as much as she wished.

This case, if unusual, is most instructive, for it indicates that if symptoms other than those comprised in the first group arise, the symptoms will be due to traction on neighboring organs, and the one most likely to suffer is the colon, so that constipation may be included among the symptoms of the third group.

Diagnosis.

The diagnosis of wandering kidney can in the majority of cases be easily arrived at. A tumor of the shape and consistence of a kidney, which can be pushed easily from below upwards, and be made to disappear completely into the lumbar region, is sufficiently characteristic. When its reducibility is, however, not complete, the diagnosis is not so easy, and the tumor may be readily confounded with a tumor of the liver, of the spleen, of the ovary, or more frequently still of the mesentery. A kidney which has been mobile for a length of time may, owing to the pressure or traction which it exerts upon neighboring structures, set up inflammatory action, which leads to the formation of adhesions, and consequently changes the movable into a fixed displaced kidney. In such cases much assistance in the diagnosis may be obtained by a careful attention to the previous history. But even in movable kidney mistakes have been made. T. W. Foot[1] quotes the case of a woman in whom attempts had been made to restrain the motion in a movable kidney by passing a tape seton through the walls of the abdomen and the tumor. "Some hæmaturia was noticed after the operation. After three months the seton broke and came away. Four years after, the kidney was removed successfully, the organ was found in the umbilical region; a deep cicatrix, about two and a half inches long, marked the track of the seton. The patient was an Irish woman, aged thirty-five. She had been eight years affected with a pain and tumor in the right side, and, the report says, she had undergone the usual operation for ovarian tumor without removing the cause of her trouble." Prescott Hewitt relates the case of a woman, aged seventy-three, in whom a hard tumor the size of the fist, lying in the right iliac fossa, had on three separate occasions been detected by medical men. Although the patient had for several years before her death passed bloody urine, alkaline and full of mucus, the diagnosis was made of a fæcal accumulation. At the autopsy, the tumor was

discovered to be a movable kidney, containing one large and two small stones in its pelvis.

In Keen's tables of nephrorrhaphy there are two cases in which a distended gall-bladder was present as well as a movable kidney. In one of them, Mears' case, the movable kidney was found through the lumbar incision and stitched into position, and then cholecystostomy was performed, and the opening in the gall-bladder was stitched below the kidney in the lumbar wound. In the other, Tischendorf's case, the mobile kidney was discovered during the operation for gallstones. Mr. Lawson Tait has published a case of distended gall-bladder upon which he successfully operated, but which had been diagnosticated as a floating kidney "by several distinguished authorities."

McCosh relates two cases in which dilatation of the uterine cavity and removal of the ovaries had been the preliminary and ineffectual methods of relieving a woman of symptoms which entirely disappeared after the performance of nephrorrhaphy.

Quite recently a case was reported in the *British Medical Journal* of a woman who was admitted into an English County Hospital complaining of weakness and indefinite abdominal pains. She was a worn, emaciated woman. Her ill-health had been gradually increasing for eight years. Among a variety of symptoms noted we find sickness frequent at intervals of a few days, occasional attacks of jaundice, constipation, and pains in the right hypochondrium, shooting to the back. She could not sit up without feeling sick. Abdominal examination showed dilatation of the stomach. "A freely movable, rounded mass could be felt in the right hypochondriac and lumbar regions, which slipped away from under the fingers." And yet a diagnosis of movable kidney was not arrived at until an exploratory incision "three inches long was made in the right semilunar line over the tumor. . . . The movable organ was found to be the right kidney. The anterior wound having been closed, the patient was turned over on her side, and the right kidney was exposed by the usual lumbar incision." This case is of interest because it appears to have been a fairly typical case of mobile kidney, and the diagnosis ought to have been made without resorting to an exploratory incision, if the subject of movable kidney had received the consideration which it deserves. Moreover, it is of interest as showing, as the report states, that a most dangerous state of debility had been brought about by the troubles induced by a moving kidney, notably the vomiting. "It may be mentioned that during her long confinement to bed, lasting for years, various remedies had been tried for the vomiting and debility. Improvement commenced for the first time on the kidney being secured in its proper place."

TREATMENT.

The treatment of movable kidney will depend upon the nature of the case we have to deal with. In the large number of cases which present no symptoms, and in which the mobility of the kidney has perhaps been discovered by accident, it will be unnecessary to do more than to advise the patient to wear a binder. In some cases, in which the symptoms are not sufficiently acute to require operative interference, or in which for other reasons an operation is contraindicated, it may be necessary to afford greater support to the kidney. The most serviceable and at the same time the least disagreeable apparatus is a wide belt of strong webbing, which should come down well over the hips, and which should be prevented from slipping upwards by means of two rubber tubes passing from behind forwards between the legs. A firm, circular or triangular pad is placed underneath the belt so as to press into the side immediately below the region of the kidney. A little care is required to fit the belt accurately and to make it comfortable. It will then be found in many cases to effect all that is required.

In some cases, however, the symptoms are very severe, and no relief is obtained by any external support—indeed, sometimes pressure seems to increase the patient's sufferings; or, again, the conditions required to obtain relief may be so elaborate and irksome as to render life miserable; then it becomes the duty of the surgeon to consider what means his art can supply to relieve the distress and to cure the patient. The first attempt to deal radically with this condition was in 1878, when Martin, of Berlin, performed the first *nephrectomy* for a painful movable kidney. Since then the operation has been performed a sufficient number of times to enable us to estimate its value. Lindner[14] has collected 36 cases of excision for movable kidney, of which 9 resulted fatally, a mortality of 25 per cent. Newman[3] gives a table of 30 nephrectomies for this ailment, with 9 deaths, a mortality of 30 per cent. He observes that in 17 cases the excised kidney was normal. These statistics show that, as regards risk to life, nephrectomy possesses a bad record. It is far more dangerous than nephrorrhaphy, as will appear when we consider this operation. Nephrectomy has the disadvantage, moreover, of removing what may be, and very frequently is, a perfectly sound organ; at the same time the other, or fixed, kidney may be disorganized. In certain cases, however, excision of the kidney may be justifiable, and even necessary. If, for instance, the movable kidney is at the same time diseased and perhaps useless, or if it be impossible to push it back into its proper

position in the loin, or if nephrorrhaphy has been attempted and has failed, removal of the organ may be the only resource left to us.

Three years after Martin had first performed nephrectomy for movable kidney, Hahn[15] published his first two cases of *nephrorrhaphy*, or fixing the kidney into its proper position by means of sutures. Since then this operation has been done a number of times, and has almost entirely, except under special conditions, superseded the operation of excision, on account of the successful results obtained, the small death-rate attending it, and because it retains for the patient the use of an important organ.

In order to reach the kidney, with the object of suturing it into its normal position, either of two methods may be adopted. First, it may be reached through the lumbar incision—that is, an incision beginning over the outer edge of the erector spinæ muscle, immediately below the twelfth rib, and extending obliquely downwards and forwards for about four inches. Secondly, the kidney may be approached from the front through a vertical incision in the linea semilunaris, as recommended by Langenbuch. The former is generally to be preferred, as in most cases of mobile kidney the organ can be found and sutured in position without opening the peritoneum. In cases of floating kidney, however, as the kidney possesses a mesentery and lies within the peritoneal cavity, it cannot be dealt with without incising the peritoneum. It matters very little whether this serous cavity is opened in front or behind, but the anterior method has the advantage of allowing a more thorough examination of the affected kidney to be made, and, in case it be diseased, to discover the condition of the other organ as well.

Four methods have been employed for fixing the kidney in the loin.

McCosh [16] thus summarizes them:

1. The sutures may be passed through the adipose capsule alone.
2. They may be passed through the fibrous capsule of the kidney itself.
3. They may be passed through the parenchyma of the kidney.
4. The fibrous capsule may be partially stripped off the kidney in order to obtain a raw surface of renal tissue, by means of which the adhesions, it is believed, would be firmer. The sutures are then passed through the parenchyma and capsule, just inside the border of the raw surface. This is the method recommended by Jordan Lloyd.

Of these various methods it would appear that the best results are obtained when the parenchyma of the kidney is included in the suture, the capsule being partially stripped off.

A great deal has been written and said on the method of suture, and the best materials to employ. Many advise that the kidney should be fixed to the edges of the wound; some that it should be fastened to the last rib. I think it matters little which method we adopt. As a matter of fact I have for a long time been in the habit of suturing it to the twelfth rib. When we see, in the kidneys hardened *in situ*, that the upper margin is marked by a groove where the kidney is clasped by the twelfth rib, this gives us a clear indication as to the proper position the sutured kidney should occupy. The material employed for suture is also a matter of no moment. I always use catgut now because it becomes absorbed. There are two considerations of the first importance: the first is to have plenty of material thrown out to glue the kidney into its place, and, secondly to allow no strain upon this material until it has had time to become organized and sufficiently strong to withstand any reasonable strain put upon it. The best way of insuring the first of these desiderata is to employ the method, first suggested, I believe, by Mr. Jordan Lloyd, namely, to divide the capsule of the kidney at the place where it is proposed to pass the sutures, to partially strip it off, and to suture the capsule and the renal parenchyma to the wound, or to the last rib if preferred.

To secure the second object we should keep the patient lying in bed for at least six weeks, no matter whether the wound has healed by first intention or not. A patient who will submit to an operation in order to be relieved from the inconveniences or perhaps agonies of a movable kidney, will generally consent willingly to a little extra confinement in order to insure that the operation shall not have been in vain.

Tillmanns,[11] who always passes the sutures through the kidney substance itself, employs both silk and catgut. He incises the capsule, and strips it partially off the renal substance. The free edges of the capsule are sutured to the wound in the loin by one catgut and one silk suture, and then a second catgut and a second silk suture penetrate the parenchyma of the kidney and secure it to the wound. These sutures through the parenchyma do not seem to cause any trouble. Occasionally a little blood is noticed in the urine for a few days, but it soon disappears. Tillmanns objects to the use of silkworm gut, as it cuts through the tissues too quickly, and thus after its use a return of mobility is facilitated.

The wound in the loin is generally healed in eight to ten days; but it is advisable to keep the patient in bed in the horizontal position for a much longer period—about six or seven weeks—so as to afford time for the adhesions to become firm.

By these means a complete cure can be obtained, and the kidney may be firmly secured into its normal position. An interesting case in point is published by von Langenbuch.[1] Some months after the performance of nephrorrhaphy for a movable kidney the patient slipped down some steps, and in consequence of this accident the kidney was apparently again displaced. Upon opening up the original incision, von Langenbuch found that the kidney, which had been fixed in position by four silk sutures, was so firmly retained *in situ* that it had not been displaced by the accident, but the tumor which was found was the displaced lobe of the liver.

The mortality attending nephrorrhaphy is exceedingly small. In one hundred and thirty-four cases collected by Keen,[2] there were four deaths, giving a death-rate of 2.98 per cent. The causes of death in these four cases were:

1st. Ceccherelli's case, in which he resected the eleventh and twelfth ribs in order to get more room, and in so doing wounded the pleura. The patient died in forty-five hours of atelectasis and pleural effusion.

2d. Hahn's case, in which death was due to an unrelieved ileus, and not to the operation.

3d. Von Langenbuch's case, in which one of the stitches passed through an old embolic infarct in the kidney, which caused death from septicæmia in three days.

4th. Lawson Tait's case, in which death resulted from suppuration, presumably the result of the operation.

In reference to this last case, Keen writes: "Mr. Tait has operated upon but three cases, one of which, he writes me, ultimately died from the suppuration following the operation, and the others were not benefited in the least; and he declares (*British Medical Journal*, November 16th, 1889) that he will have nothing more to do with the fixation of the kidney."

Tillmanns has operated sixteen times by nephrorrhaphy. Of these one died fifty-two days later of concurrent pulmonary phthisis, and death was in no way due to the operation. All the others recovered.

It thus appears that, as regards risk to life, nephrorrhaphy may be regarded as safe an operation as any in surgery.

As regards the results obtained, Tillmann's cases have the advantage, from a statistical point of view, of having been all performed by the one surgeon. He has been able to follow 12 out of the 16 cases closely. Of these 12, 6 cases were found to have been completely and permanently cured when examined at periods varying from one to three years after the operation. Of the 6 remaining

cases 1 died of phthisis forty-five days after the operation, and at the autopsy the wandering kidney was found to have contracted firm adhesions to the loin. In 3 cases no displacement of the kidney was subsequently discovered, but symptoms of "traumatic neurosis" continued. In 2 cases the kidneys relapsed into their mobile condition. Tillmanns explains these relapses, one being due to the use of silkworm gut, which cut through the tissues too rapidly; the other to the fact that the patient returned to her work too soon, before the adhesions could have become firm.

In Keen's tables of 134 cases, of which sufficient details were given in 121, there were, after three months, 52 per cent. cured, 17.3 per cent. improved, and 15.7 per cent. were failures.

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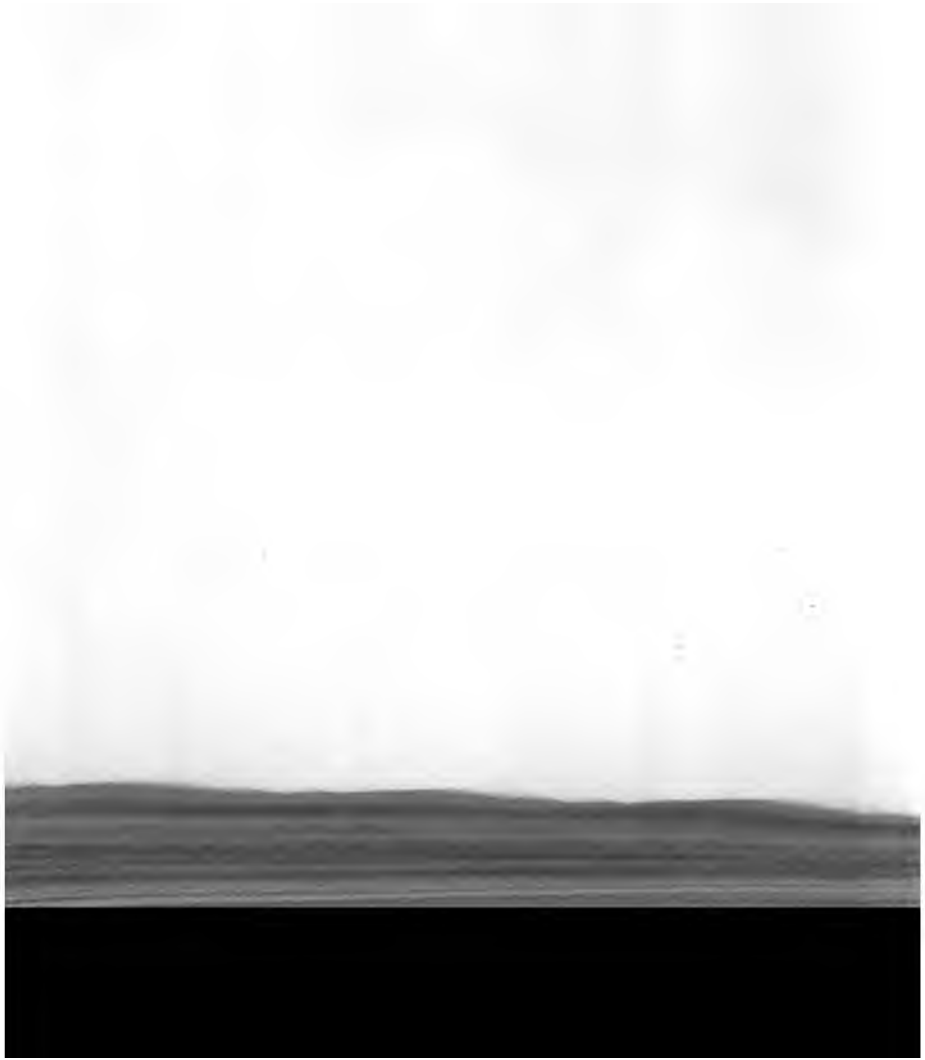

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