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HALL'S
DIFFERENTIAL DIAGNOSIS.

DIFFERENTIAL DIAGNOSIS:
A MANUAL OF THE
COMPARATIVE SEMEIOLOGY
OF THE
MORE IMPORTANT DISEASES.

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SECOND AMERICAN EDITION.
EXTENSIVE ADDITIONS.

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PUBLISHER'S NOTE

TO THE

SECOND AMERICAN EDITION.

The present work is founded upon Dr. F. DE HAVILLAND HALL'S *Synopsis of the Diseases of the Larynx, Lungs, and Heart*. The plan adopted by DR. HALL has, however, been extended to embrace all the more frequent and important diseases.

In the preface to the first American edition the editor stated that he had held especially in view (1) the *early* and often overlooked signs of the presence of disease; (2) the collection of whatever symptoms are alleged on good authority to be *pathognomonic* of pathological conditions; (3) any peculiar features which diseases have been found to present in this country. "Preference has been given to American over European authorities, as every year adds confirmation to the opinion, now widely received, that diseased conditions assume very different aspects under different climatic and sociological surroundings."

As the editor of the first edition could not revise the present one, the publisher was fortunate enough to complete an arrangement with DR. FRANK WOODBURY to give it a thorough revision, and to add to it what was lacking to make it a complete work within the limits which it aims to cover.

INTRODUCTORY.

The nomenclature of diseases adopted by the best authorities divide them into two great classes—General Diseases and Local Diseases. General diseases may be said to comprehend those which pervade the whole system, and in which any local affection may be regarded as accidental; while local diseases are those which particularly affect certain organs, and in which the involvement of any other part of the body is but a sequel of the primary lesion.

This classification, having much to recommend it, from a clinical standpoint, is the one most practically useful to the physician. The first question he should put to himself on examining a patient is, Have we here a general or a local disease? He reaches the answer by excluding those organs whose form and functions present nothing abnormal, and by distinguishing among such as are implicated those which indicate primary and essential lesions, from those which are affected accidentally or secondarily. Where no primary lesions are discoverable, he may conclude that he has to do with a general disease.

For the purpose of diagnosis, General Diseases are best divided into the two classes of (1) Fevers and (2) Diseases of the Blood. These also are each divisible into two or more classes marked by one or two leading and prominent symptoms, which are the guides to the diagnostician. Thus,

The Essential Fevers are usually acute in their course, and either characterized by an eruption of a well-defined character (the Exanthemata); or by a recurrent marked diminution (remission) or total cessation (intermission) of the symptoms (Periodic fevers); or else by a persistent course not manifesting either of these phenomena (Continued fever).

Blood Changes are rarely acute, and are either constitutional (the

Dyscrasiæ); or else characterized by definite organic lesions (Rheumatism, Gout); or by a physical and generally recognizable change in the blood itself (Anæmia, Leukæmia, Scurvy and Purpura).

Local Diseases are more conveniently classified with reference to the physiological than the anatomical divisions of the body. The functions of life are carried on by the Nervous, Respiratory, Circulatory, Digestive and Urinary systems; and the impairments of each of these form classes of diseases which are broadly discriminated by signs easy of recognition. The niceties of diagnosis are needed rather to distinguish between the varied diseases peculiar to each of these systems than to locate the disturbance in one or the other of them.

PART I.

GENERAL DISEASES.

CHAPTER I.

THE FEVERS.

CONTENTS.—*The Febrile State—Inflammatory, or Symptomatic, and Essential Fevers—The Exanthematous or Eruptive Fevers—Typhoid and Typhus Fevers—Typhoid and Malarial Fevers—The Typhoid State—Malarial Fevers—Cerebro-spinal Meningitis—Acute Tubercular Meningitis—Yellow Fever—Relapsing Fever.*

THE FEBRILE STATE.

The most common of all forms of disease is that which is presented by the Febrile State. The chief objective symptoms which it offers are found in

- I. The temperature.
- II. The pulse.
- III. The tongue and uvula.
- IV. The urine.
- V. The state of the skin.

I. The *temperature* is one of the most prominent of the phenomena in fever, and by many is regarded as the essential feature of the febrile condition; yet its correct appreciation was never understood previous to the labors of WUNDERLICH. Now, the clinical thermometer is considered as indispensable to the practitioner, as the lancet used to be.

In using the clinical thermometer, Dr. SYDNEY RINGER, of London, lays down the rule that in order to insure correctness in the observations, the following conditions must be fulfilled:—

1st. That the patient should be in bed, otherwise the temperature of the surface will be much below that of the internal organs.

2d. That the patient be in bed at least one hour before the observations are made, since that time is necessary for the surface of the body to regain the heat lost by previous exposure.

3d. The position of the person examined should be such that the anterior and posterior edges of the axilla are relaxed, for otherwise a cup-shaped cavity is formed, in which the thermometer moves freely, without being in contact with its walls. This occurs especially in emaciated persons.

4th. The temperature should be taken twice daily, say at eight in the morning and eight in the evening. If but one observation is possible, then the evening should be preferred, since the morning temperature, abnormal though it may be, rises in the evening.

5th. The thermometer should remain in the axilla at least five minutes.

As regards the peripheral parts, such as the hands and feet, according to Dr. H. WEGSCHEIDER,* the following propositions should be received in reference to the distribution of temperature in febrile diseases:—

1st. There is no constant relation between the internal temperature, as measured in the axilla, and the general temperature of the surface.

2d. Two completely symmetrical parts of the skin, as between the toes, show no proportionate course in their temperature; not only do they differ by not rising or falling to the same level, but one may rise while the other remains stationary or falls, or *vice versa*.

3d. There is greater variation in the temperature curves in the same part of the skin in the same person in fever than in health; but in fever there is a striking fall of temperature, notably lower than in the healthy state. However, in those people who suffer from cold feet the temperature is often as low, or somewhat lower.

4th. It follows, from the last, that there is a greater difference, in fever, between the temperature of the axilla and that of the periphery than any changes of local temperature which may occur in health.

Although the axilla is generally selected, on account of convenience, the temperature is often taken with the thermometer in the mouth, rectum or vagina, and it is believed that such observations give more correct indications of the heat of the body than those taken on the surface.

The temperature fluctuations in the various zymotic diseases have now

* *Archiv. für Pathologie*, Feb. 1877.

been carefully studied by many clinical observers, who have deduced observations which are of great service in diagnosis, as some of them are characteristic.

A pretty constant increase and decrease of temperature exists in the several specific fevers, a close observation of which, in accordance with the foregoing rules, will often serve as a valuable aid both in diagnosis and in prognosis. Dr. WUNDERLICH, in his work, gives useful tables for this purpose, and we subjoin a valuable comparative table of the pulse as well as the temperature in seven of the more frequent febrile diseases, drawn from recent English observations.

COMPARATIVE TABLE OF THE TEMPERATURE AND PULSE IN THE LEADING FEBRILE DISEASES.

Day.	Typhus Fever.		Typhoid Fever.		Measles.		Scarlet Fever.		Febricula.		Rheumatic Fever.		Pneumonia.	
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
1st.....													102.8	123
2d.....							104.2	144	103.	99			102.3	120
3d.....	104.8	108	103.1	98			104.	148	103.7	103			103.6	122
4th.....	103.6	113	103.4	110	102.3	130	103.	134	104.	105	101.8	105	104.	126
5th.....	103.	114	102.7	107	103.	124	101.2	122	102.6	99	102.	114	103.	122
6th.....	103.2	122	103.2	104	100.2	112	100.6	108	98.4	99	102.	116	102.8	122
7th.....	104.2	124	103.7	107	98.	102	100.	106			103.	120	100.	114
8th.....	103.8	122	102.5	108	98.	98	100.	110			100.	90	99.	94
9th.....	103.	113	103.	108	98.	80	99.8	108			100.	96	98.	78
10th.....	102.7	117	102.6	111			99.	100			99.4	86		
11th.....	102.4	119	103.	111			98.6	104			101.	104		
12th.....	102.2	108	102.5	112			98.	84			101.	102		
13th.....	100.5	106	102.2	108							102.	100		
14th.....	100.	100	102.4	109							100.9	100		
15th.....	99.4	98	101.8	107							100.	88		
16th.....	98.7	92	102.	100							98.	90		
17th.....	98.4	90	101.4	100							99.	94		
18th.....	98.2	85	98.8	98							102.	96		
19th.....			101.4	105							103.	102		
20th.....			102.2	100							101.6	100		
21st.....			98.8	98							101.7	104		

The above table, prepared from a series of observations, by Dr. J. S. WARTER,* illustrates the normal and average contrasts of pulse and temperature in the course of the diseases specified, when their tendency is to recovery.

* *St. Bartholomew's Hospital Reports*, vol. ii, p. 78.

II. The *pulse* is increased in frequency, and may be either hard, full and bounding, or tense, small and contracted. The former condition is more common in active inflammation of the organs above the diaphragm; the latter, in many inflammations below the diaphragm and in idiopathic fevers. In fevers of a typhoid form, an unusually slow pulse is sometimes encountered, as also a pulse with apparently a double beat, the "dierotic" pulse. In the later stages the pulse may be soft, gaseous or thready, indicating febrile changes in the walls of the vessels and the heart.

III. The *tongue* in the beginning of the febrile state is usually whiter and dryer than usual, and more or less coated with a "fur" or viscid covering, from the more rapid evaporation of the watery secretions. Later on, in the progress of severe fever, the tongue becomes dry, and the exsiccated mucus and epithelium form a brownish or blackish crust, while the papillæ shrink, so that on this crust becoming detached, the surface of the organ looks glazed and smooth. The peculiar appearance of the tongue in certain diseases will be described in connection with these diseases.

IV. The *urine* in fever is scanty and high colored. Its alteration from the healthy average is chiefly in the much larger quantity of urea and urates which it contains, and the diminution of its chlorides. According to the researches of Dr. J. BURDON SANDERSON, in the early stage of fever a patient excretes about three times as much urea as he would do on the same diet if he were in health, the difference between the healthy and the fevered body consisting chiefly in this, that whereas the former discharges a quantity of nitrogen equal to that taken in, the latter wastes the store of nitrogen contained in its own tissues. That this disorder of nutrition is an essential constituent of the febrile process is indicated by the fact that it not only accompanies the other phenomena of fever during their whole course, but precedes the earliest symptoms and follows the latest. That it anticipates the beginning of fever was first demonstrated by Dr. SIDNEY RINGER in his investigation of the relation between temperature and the discharge of urea, in ague. That the same condition continues after the crisis has passed, *i.e.*, the temperature has begun to sink, was shown by Dr. SQUAREY.

There are various methods of determining the rate of secretion and

the amount of urea. Its relative excess may be inferred when the urine has a deep yellow color, a high specific gravity, and a strong urinous odor. If a small quantity of it be allowed to evaporate to a mucilaginous consistence, and nitric acid be added, drop by drop, crystals of nitrate of urea are found after a few hours. They are of a pearly white lustre, and their proportion roughly indicates the quantity present. When the urea is in great excess, the crystals will form on the addition of nitric acid to the urine, without the preliminary evaporation, by merely allowing the test tube to stand for a short time.

V. The *skin*, in common with the other emunctories, has its functions much influenced by the fever process. Apart from the changes in temperature, considered above, there are alterations in the appearance of the skin, and in the character of its secretions, which accompany fevers; certain eruptions also appear, that are more or less characteristic; such as the smallpox pustule, the chicken-pox vesicle, the urticarial wheal, and the scarlatinal acute desquamative dermatitis. The parasitic diseases of the skin, although they may accidentally be associated with pyrexia, exist entirely independently, and the fever bears no causal relationship to them.

The skin during fever has for its typical appearance a color which if not decidedly dull and sallow, is at least less clear than in health; in typhus it may be quite dusky; in bilious remittent and yellow fever it becomes jaundiced. In typhoid the surface is more nearly that of health, but the cheeks are flushed and there are rose-spots on the chest and abdomen. In acute rheumatism and in the third stage of intermittent, the skin is covered by a profuse perspiration, which in the former case has a sour smell. Exhausting sweats also occur in pyæmia and phthisis. But the dull-colored, dry and harsh skin is the characteristic appearance, and is due to deficient action of the perspiratory and other glands.

INFLAMMATORY (SYMPTOMATIC) AND ESSENTIAL FEVERS.

The group of symptoms, collectively known as a *fever*, often accompanies strictly local maladies and injuries. In such cases it is distinguished as Inflammatory, or Symptomatic, Fever, and it is of the first importance to distinguish it from Essential, or Idiopathic, Fever, under which general

term all true fevers are included. The development of this distinction has been one of the most prominent achievements of the modern methods of diagnosis. "It is astonishing," remarks a recent writer, "with the progress of medicine, how many affections have been passed over from the domain of fevers to the narrower circle of inflammation of individual organs." Hence it is of prime importance to determine promptly in the beginning of a case whether the febrile symptoms are a feature of a local disease or the commencement of a general one.

Inflammatory or Symptomatic Fever.

Is usually preceded by some local lesions or symptoms.

Pulse frequent, full and generally tense.

Is accompanied by marked and definite local disturbance.

Course is indefinite, dependent upon the progress of the local lesion.

Anatomical lesions definite and invariable.

Prognosis mainly depends upon the progress of local lesion.

Essential or Idiopathic Fever.

Has no definite antecedent local symptoms.

Pulse frequent, full or small, but rarely tense. (DA COSTA.)

Local disturbances vary, and are not prominent, or but temporarily so.

Runs a definite course, with a strong tendency to spontaneous termination at a given time.

Generally characterized by relatively unimportant, or entirely absent anatomical lesions.

Local manifestations of less importance in estimating the prognosis.

Dr. WILLIAM STOKES* divides the local symptoms of essential fever into three groups: (1) Functional or nervous; (2) those dependent on special anatomical changes; (3) those arising from re-active inflammation.

Examples of functional symptoms are delirium, carphologia, cough, diarrhœa, epigastric tenderness, and the like; of the second group, the alterations which occur in the brain, heart, lungs, spleen or intestinal glands; and of the third, the swelling and infiltration of organs. What he calls "the grand rule of diagnosis" in fever is *not to apply to these local symptoms in essential fever the rules of diagnosis of local diseases*, as this would lead to a false appreciation of the disease, and to erroneous treatment. For example, a typhus patient may exhibit the marked symptoms

* "Lectures on Fever," London, 1874.

of inflammation of the brain; but if he is treated with active antiphlogistic treatment, and with ice to the head, and leeches, he forthwith sinks and dies.

Of hardly less importance is the distinction between *organic* and *functional* (or neurotic) *changes* in fevers. Delirium, pain, coma, convulsions, cough, etc., may all appear as phenomena of the evolution of the poison which produces a general fever, without signifying any definite anatomical lesion. In other words, essential fever produces local symptoms without organic change. "It is," remarks the author just quoted, "because this proposition has not been sufficiently accepted, sufficiently engraved upon the minds of medical men, that so much mischief has been done in the erroneous treatment of fever."

THE DIAGNOSIS OF THE ESSENTIAL OR ERUPTIVE FEVERS (EXANTHEMATA).

This group includes smallpox, varioloid, scarlet fever, measles, roseola, and also those more indefinite forms, varicella and r otheln. They have many points of similarity. "They are all characterized by a period of incubation, during which the poison lies dormant in the system; by a fever of more or less intensity preceding the eruption; by an eruption which presents a distinct aspect in each disease, and which pursues a definite, clearly defined course until it, and with it the febrile malady, disappears. Moreover, they are all very prone to occasion serious sequel e; are all, in the main, disorders of childhood; rarely attack the same person twice; are contagious, and have not as yet been brought under specific treatment." (DA COSTA.)

It is of great credit to the practitioner, and often of the utmost utility to others, for him to make an *early* diagnosis between these diseases. This is not always possible to accomplish. But a close observer will find several indications which will guide him to a correct opinion before the appearance of the rash. One of the principal of these is

The condition of the throat. This region is affected at a very early stage in nearly all cases. In simple scarlatina the very earliest symptom is a more or less uniform redness of the middle of the soft palate, the uvula alone, or the uvula, anterior pillars of the fauces, and tonsils; never the posterior wall of the pharynx alone. On the other hand, in

smallpox, the part first affected is the posterior wall of the pharynx; while in measles the posterior walls of the fauces and neighboring parts of the pharynx are always redder than the anterior pillars and soft palate (Dr. ALOIS MONTI). In r otheln and measles the tonsils are red and swollen early in the disease; but in simple scarlet fever, for the first twelve hours there is generally very little swelling of the affected parts, and children seldom complain of pain in the neck or in swallowing. After twelve or twenty-four hours the swelling commences, and the redness becomes less uniform, and more punctiform. This peculiar punctiform appearance may be noted often ten or twelve hours before the rash on the skin is visible.

If in malignant scarlatinal sore throat, however, there is, from the first, parenchymatous inflammation of the tonsils and the submucous connective tissue, and this condition is associated with well-marked nervous symptoms, a severe case with ulceration of the fauces may be confidently predicted.

In general terms, it may be said that when the soft palate has a diffused red hue, "similar," as TROUSSEAU remarks, "to, but deeper than, that of the skin," while the tonsils are not involved; when with this is a very hot skin, a very quick pulse, vomiting, a tongue with thick, creamy fur, red borders, and prominent papul e; and with these symptoms, exposure to the presence of a scarlatinal epidemic, the physician need not hesitate in pronouncing it scarlet fever.

A very early symptom of scarlet fever has been insisted upon as strictly pathognomonic by an Irish physician, Dr. JOSEPH DUGGAN.* It is that the eye assumes a peculiar brilliant and glistening stare, very different from the liquid, tender, watery eye of measles, and which, once carefully noted, remains impressed on the observer's memory.

The character of the preliminary fever often differs. In scarlatina it is marked and high, which distinguishes it broadly from diphtheria, which is not marked at the outset; in measles it is of a catarrhal form; while in smallpox it is often associated with very severe pains in the back and loins, not observed in the other exanthemata. This spine-ache is central in its position, and is less affected by change of posture than is the pain of lumbago, and is not confined to one side nor to the *erector*

* *Medical Press and Circular*, Feb., 1869.

spine muscles. It is stated by some authors that this pain is increased in proportion with the severity of the attack, and thus forms an important element in the prognosis; but this statement should be confined in its application to adults, as in children the rachialgia is rarely intense. Dr. WILKS observes that the most virulent cases of variola are almost apyretic and devoid of feverish symptoms.

In all the exanthemata the eruption makes its appearance in the throat or the mouth, from *twelve to twenty-four hours* (and, in many instances longer) before it appears on the cutaneous surface. In *smallpox*, in *scarlet fever*, in *measles*, in all their grades, the eruption may be looked for, with confidence, in this region long before it can be detected at any other point, and as the eruption is often the last link in the chain of evidence necessary to decide a question of diagnosis, the knowledge of this fact will often relieve both physician and patient.

The eruption may, in smallpox, often be *felt* before it can be seen; the sensation imparted to the finger being like little shot underneath the skin. Its first appearance is as a simple red point or pimple, soon changing to a papule. The red blush of scarlatina disappears on pressure, but is immediately restored when the pressure is removed. It has no decided prominence to sight or feeling. Previous to the general boiled-lobster appearance of scarlatina, there is generally a punctate eruption and the skin appears to be rough, upon passing the hand over it, something like a nutmeg-grater.

Dr. OSLER, of Montreal, has called attention to and described a number of *initial rashes*, which precede, by twelve to twenty-four hours, the appearance of the variolous eruption. They are principally noticeable on the upper part of the trunk, and generally have the similitude of a deep, suffused flush.

The *pulse* in variola is asserted, by some, to be pathognomonic, and significant so early in the disease that the malady can be positively diagnosed many hours before the eruption appears. But no definite descriptions nor tracings of this pulse have been given.*

Having thus defined the special indications for a diagnosis of these diseases in their earliest stage, we give in the following table a synopsis of their comparative clinical history:—

* See Dr. A. S. Payne, *Va. Med. Monthly*, March, 1878; J. S. Conrad, *Trans. of the Med. and Chirurg. Faculty of Md.*, 1874.

RÖTHELN.

SCARLET FEVER.

INCUBATION.

Period of incubation from one to two weeks.

Very uncertain; from a day to several weeks; on an average about twelve days.

INVASION.

Langor; shiverings; nausea and vomiting; perhaps sore throat, but not severe.

Shiverings; nausea; vomiting; throat very much inflamed; sneezing and discharge from the nose; convulsions occasionally in children.

Premonitory fever of short duration; relieved by the eruption.

Great heat of skin and very frequent pulse; not relieved by the eruption.

ERUPTION.

Appears early and almost simultaneously over the *whole body*—is *sudden and general*—is less marked on the limbs than on the trunk, and especially on the chest; may first appear upon the back, upon the chest or neck, upon the cheek or upon the forehead; travels downward.

On second day; first on neck, and face, and body; spreads rapidly to limbs.

At first minute dots, which rapidly assume the appearance of large, irregular-shaped patches, somewhat like measles, but less distinct in color and form, varying from three-cent piece to twenty-five-cent piece in size.

The skin feels harsh and rough (like a nutmeg-grater), minute points of redness next appear, soon surrounded by deep rosy areola (like a boiled lobster).

These patches are raised above the surrounding skin, especially toward the middle, and are of a darker red color at the centres.

The eruption is uniform, or in very large patches, of a scarlet hue, with interspersed raised spots and perhaps a few vesicles; the rash is followed, after the seventh day of its appearance, by complete desquamation.

Fades in about four days; desquamation, when it occurs, is fine and bran-like.

The disease is communicated by these epithelial scales.

MEASLES.

SMALLPOX.

INCUBATION.

Generally from seven to fourteen days.

Generally about ten days, but varies from five to twenty days.

INVASION.

Lassitude, shivering, catarrh; sneezing, discharge from nose; harsh cough; rarely vomiting.

Shivering, severe pains in the back; nausea. There may be a marked chill followed by vomiting.

Fever, with hot skin and frequent pulse; rather increased by the eruption.

Fever often very violent, with bounding pulse and pain in the loins; great relief from occurrence of the eruption.

ERUPTION.

Appears on fourth day, first on face, spreads gradually in forty-eight hours to the rest of body.

Eruption at end of third or on fourth day; first on lips, palate and forehead.

Comes out in small circular dots, like flea-bites. These dots run together and form blotches, of a raspberry color, and the latter are very prone to assume a crescentic or horse-shoe shape, being slightly elevated above surrounding skin.

Eruption is first papular; after about a day becomes vesicular, then pustular; on the eighth day of the eruption, the pustules mature; about the twelfth the scabs begin to fall. The danger of contagion does not cease until desquamation is completed.

Eruption is sometimes diffused over the whole body in a confluent form, and is of a dull, deep red color, offering a contrast to the crimson or scarlet redness of scarlet fever.

Lasts five days; followed by incomplete desquamation.

RÖTHELN.

SCARLET FEVER.

ACCOMPANIMENTS.

Only moderate sore throat, with hoarseness; swelling of the neck; and rarely coryza.

Cerebral symptoms absent.

General systemic disturbance mild.

Sore throat; coryza or bronchitis rare.

Tongue red; raspberry character. Cerebral symptoms frequent and grave.

Marked systemic disturbance.

THERMOMETRY.

“The temperature always highest on first day of attack, not exceeding 102°, next day falling to 100°, and getting normal on the fifth day.”

(*Fox.*)

“The temperature nearly always *sub-febrile* (99.5° to 100.4°)—sometimes *febrile* (101.3° to 102.2°).”

(*Wunderlich.*)

No secondary fever.

Temperature may reach 105.6°, or even a higher point. It usually remains continuously high during the eruption, and it is thus “well distinguished from those affections with which, on account of other symptoms, it is most easily confounded, and more particularly *measles and rötheln.*” (*Wunderlich.*) It begins to subside about the tenth day.

No secondary fever.

DESQUAMATION.

Minute particles of cuticle, like scales of *fine bran*.

Always begins toward centre of the eruptive patch, and gradually extends to the circumference.

Comes off in branny scales and in large patches. Occasionally epidermis of the hands is detached entire, and may be slipped off like a glove. This is true also of the feet.

Sometimes several successive desquamations occur.

Is frequently accompanied with itching, which in some cases is excessive.

COMPLICATIONS.

Pneumonia rare.

Pneumonia rare; pleurisy more frequent. Endocarditis exceptional.

SEQUELÆ.

Dropsy rarely; swelling and sup-puration of the cervical glands not uncommon.

Epidemic, moderately contagious.

Bright's disease, dropsy, conjunctivitis, deafness, phthisis, chronic diarrhœa, glandular enlargement.

Very contagious.

MEASLES.

SMALLPOX.

ACCOMPANIMENTS.

Bronchitis, coryza and redness of the eyes, very constant ; sore throat rare.	Sore throat and dry cough ; bronchitis rare.
Tongue coated, or red at edges.	Tongue coated and swollen, or red at edges.
Cerebral symptoms very rare and not severe.	Cerebral symptoms, especially convulsions in children, frequent.

THERMOMETRY.

Temperature during the preliminary fever may reach 105° – 106° . Within twelve to twenty-four hours from appearance of rash it sinks speedily to the normal. Protracted defervescence indicates a complication.	Temperature during the preliminary fever high, often 106° ; falls rapidly to about 100° after eruption. Rises again during the secondary fever and falls slowly ; a slight rise during desiccation.
No secondary fever, though the fever may increase slightly before eruption leaves.	Secondary fever well marked in all cases.

DESQUAMATION.

Always in branny scales, not in patches or flakes.	In scabs, crusts and thick scales.
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COMPLICATIONS.

Catarrhal pneumonia is very frequent, especially in adults.	Pneumonia not very frequent.
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SEQUELÆ.

Chronic bronchitis, phthisis, conjunctivitis.	Chronic diarrhœa, glandular enlargement, various diseases of the eyeballs and eyelids.
Contagion almost exclusively limited to children.	Very communicable ; mild cases may cause severe or malignant ones. Chiefly adults.

TYPHOID AND TYPHUS FEVERS.

Until within comparatively few years these two fevers were confounded; and although now, in this country at least, they are distinctly recognized as wholly different diseased conditions, yet there are numerous instances where the clinical features of cases assimilate them to one or the other of these conditions, and yet fail to answer satisfactorily their currently received definitions. Such are the numerous gastric, nervous, simple continued, synochal, mixed, entero-miasmatic, typho-malarial, etc., types which are so often referred to in medical literature. There are, in fact, wide variations in the local features of this group of diseases, and it is the exception to find the classical portraits of one of the group, drawn in the hospital wards of great cities, correspond precisely with the case as seen, modified by the numerous special conditions of particular regions. We shall cite some of these modified types, after having considered the early symptoms and broad distinctions of typhoid and typhus.

Typhoid fever is peculiarly a disease of slow and insidious approach. For days, and sometimes for weeks, the patient is ailing; and as this gradual onset is known to the public, the physician is often called upon to pronounce an opinion as to the probability of the threatenings being of typhoid long before any positive sign is present.

The general symptoms are a sense of weakness and fatigue, loss of appetite, muscular soreness, headache, generally dull, sometimes severe, disturbed sleep, poor appetite, low spirits. A characteristic and often early symptom is epistaxis. Frequently there is a bronchitis, with shallow and rather frequent breathing, with some sonorous râles over the chest. A skilled auscultator can often pronounce from the character of the râle as to the presence of typhoid, as they yield a peculiar, dry, ringing sound. In one of his clinical lectures, Dr. DAcOSTA remarks on this: "I should be loath to rest upon this symptom alone, but there is something about it that often makes the diagnosis of typhoid special and specific."* This point is worthy of further investigation.

The pathognomonic symptoms of typhoid are those connected with the abdomen. The belly is swollen and tympanitic; there is diarrhœa with

* *Medical and Surgical Reporter*, Vol. xxviii, p. 11.

perhaps abdominal pains; rumbling and tenderness near the ilio-cæcal valve and about the right iliac fossa. The tongue is tender, and sometimes moved with pain; the teeth show accumulations of dried mucus (sordes); thirst is rarely excessive; vomiting is rare; the mind is dull, and the delirium is usually low and muttering. The peculiar eruption appears on the chest and belly, most frequent between the nipple and navel, about the sixth or eighth (never before the fifth) day of the fever. It is in scattered, small, reddish, delible spots, resembling flea-bites. Later they become rose-colored, and are surrounded by an area of erythema, shading off into the surrounding skin. They are not elevated, or very slightly so, and they disappear entirely on firm pressure, but promptly re-appear. They give no feeling of hardness to the finger passed over them. These spots are, however, often wholly absent; and their presence, number and size do not seem to bear any relation to the severity of the attack.

The prodromal symptoms above mentioned are, however, often varied. Dr. A. LARRABEE, of Louisville, remarks that the characteristics of the prodromal stage, the lassitude, epistaxis, and even the susceptibility of the bowels to purgatives, which are valuable aids to early diagnosis in more northern latitudes, are not so important in the malarial regions of the southern and southwestern United States;* and Dr. JURGENSEN, of Kiel, Prussia, has given the history of a number of cases, with the anatomical characteristics of typhoid fever, when the attacks were sudden, with a well-marked chill, a high temperature (104° Fah.) and quick pulse, swelling of spleen and little or no diarrhœa.† Such a course is extremely rare in this country.

In *typhus* the eruption usually appears as small, discrete spots, slightly elevated, of a dingy red color and not completely fading on pressure. In a short time the spots cease to be elevated, and fade less on pressure, and a purple mottling appears in the interjacent portions of skin. At a still later period—say on the eighth, ninth or tenth day—the spots become entirely petechial, not being at all affected by pressure. The eruption begins to fade about the ninth or tenth day, and disappears about the fourteenth, and, if there be no local complication and the patient has not

* *Trans. Kentucky State Med. Soc.*, 1876, p. 123.

† *Med. Times and Gazette*, 1874.

been very greatly protracted, convalescence is established between that day and the twenty-first. In slighter cases, however, the serious illness may continue for only about a week, the eruption may never be very marked, and the patient may become convalescent from the tenth to the fourteenth day; while, in very severe cases the rash may become petechial at an early period, and may continue on the skin till near the end of the third week, and the convalescence may be very greatly protracted. Generally, in a simple uncomplicated case of typhus, the pulse and temperature fall below the normal standard at the earlier period of convalescence, and again rise when the patient takes more food and is capable of some little muscular exertion. Usually the bowels are confined during the course of the disease, but such is not always the case; sometimes towards the height of the fever and when there is great prostration of strength, the bowels are relaxed, apparently from want of power in the sphincter to retain the fecal matter; but in some cases there is profuse diarrhoea during the whole course of the disease, and this independently of any medicine. It is impossible to base the diagnosis between typhus and typhoid upon the confined state of the bowels in the former disease and the occurrence of relaxation in the latter, for it is not uncommon for the bowels to be confined in typhoid. So, also, though as a rule the cerebral disturbance is more marked in cases of typhus than in typhoid, it sometimes happens that a patient will pass through a marked or even severe attack of typhus without much delirium, and retaining his intelligence to such an extent as to be able to answer simple questions put to him, without any apparent difficulty. In such cases, however, the patient, on recovery, has no recollection of anything that has occurred from a very early period of his illness till convalescence is far advanced.

In the following table, the leading phenomena of the two diseases, as given by the best authorities, are contrasted, for the purpose of establishing their clinical distinction. The non-identity of the two affections is now everywhere acknowledged. (In Germany typhoid is often called abdominal typhus; an unfortunate title.)

TYPHOID.

Age generally from eighteen to thirty-five.

Not directly contagious; often sporadic.

Attack generally insidious.

Duration fully three weeks; often much longer.

Death hardly ever before end of second week; more generally in or after third week.

Cerebral symptoms come on gradually; last longer.

Great emaciation.

Face pale, or flush confined to cheeks.

Skin hot, sometimes covered with acid perspiration.

Abdominal symptoms, such as diarrhoea, tympanites; intestinal hemorrhage not unusual.

Epistaxis common.

Bronchitis and sometimes pleurisy.

Eruption light red and not on extremities.

Autopsy shows morbid state of Peyer's patches and solitary glands; enlargement of mesenteric glands; ulceration of mucous coats of intestines; enlargement and softening of spleen; ulceration of the pharynx.

TYPHUS.

At all ages, often in persons beyond middle life.

Highly contagious, generally epidemic.

Attack generally sudden; no lengthened prodromata.

Duration somewhat shorter; often not prolonged beyond second week.

Death not unfrequently at end of first week, and often before conclusion of second.

Delirium or decided stupor comes on soon, sometimes almost from the onset; headache has appeared and disappeared by about the tenth day.

Less emaciation; greater prostration.

Face deeply flushed, of dusky hue; eye injected.

Skin of pungent heat, sometimes emitting an ammoniacal odor.

No abdominal symptoms; bowels constipated; meteorism rare; no intestinal hemorrhage; sometimes acute dysentery during convalescence.

No epistaxis.

Pneumonia, or marked congestion of the lungs, and bronchitis of finer tubes.

Eruption darker color and all over the body.

No constant *post-mortem* appearances; most common are dark-colored, liquid blood, and enlargement of the spleen; softening of heart more common than in typhoid; no intestinal lesions.

COMPARATIVE THERMOMETRY (DR. J. W. MILLER).

TYPHOID.

TYPHUS.

The duration of elevated temperature is very rarely less than twenty-one days ; it is generally longer, and may be protracted to thirty-five days or even more.

The evening temperature is almost constantly higher than that of the morning.

The difference between the morning and evening temperature is generally, throughout the case, greater than in typhus ; and toward the end of the fever there occurs the very characteristic oscillation of temperature, during which the difference is frequently five, six, or even seven degrees, and which may continue from a few days to a week or more.

A high temperature is frequently accompanied by a pulse but slightly accelerated, and occasionally by a pulse slower than normal, and not infrequently dicrotic, especially during convalescence.

The duration of elevated temperature is very rarely beyond eighteen days ; it is generally shorter by several days, and may be even so short as nine days.

The evening temperature is frequently lower than that of the morning.

The difference between the morning and evening temperature, during the height of the fever, or from about the third to the tenth or eleventh day, is comparatively seldom above one degree, and although about the period of defervescence the difference is sometimes much greater ; the oscillation is not continued over more than one or two days.

A high temperature is, as a rule, accompanied by a high pulse.

The varieties of fever called *gastric* and *nervous* have not been recognized as distinct types by the most recent writers. Yet there is no doubt that many cases of continuing fever present gastric rather than abdominal symptoms, and various other perceptible variants from the type of a mild typhoid. The following semeiological table, drawn from Dr. COPLAND'S work, will illustrate this :—

FORMS OF TYPHOID.

SIMPLE CONTINUED TYPE.

100-120, small, weak, irregular; intermittent when a dangerous attack.

Heat of surface generally rises over 100°.

White, foul, loaded or furred; again red at its sides, and point loaded with dirty yellow fur.

Tenderness at epigastrium; looseness or diarrhœa of an ochery hue; vomiting early.

Pain in head, throbbing of arteries, brilliant expression of eyes, marked acuteness of senses, watchfulness and restlessness, moaning and incoherent muttering, dilated pupils, and coma.

A common and early complication, either to bronchial surface or congestion of substance.

Sore-throat or inflammation of fauces sometimes accompany.

By subsidence of the prominent morbid actions indicative of a gradual decline.

NERVOUS TYPE.

Pulse. Soft, feeble, and quick; about eleventh day very quick and unequal.

Temperature. Heat of skin not much elevated; it may even seem natural or diminished.

Tongue. Loaded or covered with a dirty mucus, afterward brown or black, incrustated or fissured.

Gastric Symptoms. Fœtor of the breath and of the discharges, an irregular relaxed state of the bowels, pain at the epigastrium, nausea and vomiting.

Head Symptoms. Countenance pallid or transiently flushed, head heavy, continual restlessness, want of sleep, tremor, hearing dull, unconscious evacuations, low delirium, early stupor and coma.

Lung Symptoms. The bronchial surface is the part chiefly affected; substance of the lungs sometimes complicated.

Affection of Throat. Sore-throat, occasionally so severe as to resemble an attack of anginose maligna.

Recovery. Often announced by a true crisis.

Gastric fever, which is not to be confounded with gastritis (although this is more properly a fever of gastric origin), is recognized by NIEMEYER and other competent authorities as a separate type. It commences with loss of appetite, headache and languor, followed by a slight chill, with marked gastric irritability, great nausea, frequent vomiting, and constipation. There is considerable tenderness on pressure over the stomach, a low pulse (60 to 70 per minute), and a temperature at first rising slightly to (100° Fahr.), then falling below the normal as the disease advances (to 95° and even lower). A grave symptom is double vision or total loss of sight. There are no tympanites, diarrhœa, delirium, subsultus tendinum, spots, iliac tenderness, nor sordes, as in typhoid. Women are more liable to it than men, old persons than those of middle age or youth. Its outbreaks indicate it to be a zymotic disease, and the mor-

tality is even higher than in typhoid. The pathognomonic symptom of the disease is the peculiar sweetish odor of the breath; it is likened by some to the odor arising from hot water poured on garlic, having a slightly alliaceous odor; or, according to others, it resembles a faint aroma of valerianic acid.*

Typhlitis can readily be distinguished from typhoid fever by the pathognomonic sign of a dense tumor in the iliac fossa, increasing, and exceedingly tender on pressure.

TYPHOID AND MALARIAL FEVERS.

TYPHO-MALARIAL (WOODWARD), ENTERO-MIASMATIC (WOOD), OR
REMITTO-TYPHUS (DRAKE).

In order to bring into relief the broad distinctions between the typhoid and malarial fevers when in their typical forms, the following comparative table has been prepared, which is chiefly that of Dr. E. M. HUME.†

TYPHOID.		MALARIAL.
Decomposing animal and vegetable matter, especially human excrement.	Cause.	Emanations from marshes, damp, low or new soil; always vegetable, never animal.
Old soil; may be high and dry and long settled, especially where saturated with sewage.	Locality.	New land, moist, low and swampy.
Epidemic of typhoid fever.	Circumstantial Evidence.	Prevalence of malarial disease.
Seldom after forty.	Age.	All ages.
Continued without intermission or decided remissions.	Periodicity.	There is either intermission or remission.
Lasts three or four weeks; cannot be cut short.	Duration.	Can be interrupted and cured in a few days.
Great nervous disturbance and prostration; dull, heavy, throbbing, persistent frontal headache; twitching of muscles; tickling of throat; ringing in ears; deafness; mind stupid.	Nervous implication.	None, although there is sometimes severe headache, simulating meningitis.
Asthenic, not wild.	Delirium.	Sthenic.
Frequent.	Epistaxis.	None.
Catarrhal bronchitis with sometimes tough, tenacious sputa.	Lungs.	Congested, when affected at all.

* Dr. G. B. Bullard, *Trans. of the Vt. Med. Soc.* 1877, pp. 52-56.

† *Peninsular Journal of Medicine*, Feb., 1875.

TYPHOID.

From 70 to 140 beats per minute, small, irregular or dicrotic.

Hot, even when moist; emits a peculiar, musty odor, pathognomonic of this fever.

Indicates an increase of temperature from morning to evening of about 2 deg., and a decrease of 1 deg. from night to morning; commences first day 98.5 deg., reaches its maximum of 104 deg. on the morning of the fourth day; from this time the evening temperature ranges between 103 deg. and 104 deg., morning 1 deg. lower, until end of second week, when it gradually declines in the same regular manner, always lower in the morning than in the evening, except when there is a complication.

Protrudes tremblingly; is covered with a whitish yellow coat, which subsequently disappears and is replaced by a dry, pale brown one, with red, glazed tip and edges; teeth covered with dark-brown sordes.

Pale, livid, muddy, or may be clear, with cheeks flushed.

Foaming, light color, free from sediment; frequently contains albumen; has typhoid odor like body.

Diarrhœa, except in mildest cases; stools offensive, pea soup, bright yellow or brown; devoid of mucus, but sometimes contains whitish flocculi.

Tympanites occurs, giving tub shape to abdomen; pressure over cæcum produces pain and gurgling sound; tenderness over spleen.

Stomach not involved; no severe pain anywhere, except when peritonitis occurs.

MALARIAL.

Pulse. More frequently high, full and bounding.

Skin. Varies; sometimes dry and hot; odor acid and swampy; at other times may be normal.

Thermometer. Rises rapidly to 105 deg. or more first day or two, and falls suddenly; is not so uniform; may rise and fall seven degrees in one day.

Tongue. Coated all over with a heavy, dark yellow coat. No sordes.

Complexion Sallow; eyes yellow.

Urine. Dark color, turbid, no albumen, except in malarial hemorrhagic fever.

Excretions from Bowels. Bowels costive; dark, hard, dry, bilious stools.

Abdomen, shape, etc. No tympanites or tenderness of abdomen.

Pain. Gastric disturbance and vomiting of bile; pain in stomach and elsewhere very intense; may be throughout the entire body.

TYPHOID.

Occurs during second week ; from one to twenty small rose-colored spots, size of pin head, appear on abdomen, chest or back ; do not extend to extremities ; present no distinct elevation to the touch, disappearing upon pressure, but reappearing upon its removal ; last about three days ; fade away and a fresh crop appears. This eruption is claimed to be "peculiarly and absolutely diagnostic of typhoid fever." Later in the disease sudamina appear.

Great—averages one in five.

Inflammation and ulceration of Peyer's, solitary and Brunner's glands ; sometimes perforation of bowels with peritonitis, and fatal hemorrhage ; inflammation and enlargement of mesenteric glands and the spleen (which sometimes bursts) ; the brain, stomach, liver and lungs sometimes inflamed.

We shall now consider the character of a disease presenting in its different stages symptoms both of malarial and typhoid fever.

The experience of numerous observers has proven that there is a complex form of fever prevalent in malarious districts, in which the typhoid and miasmatic elements are combined. It has been proposed by Dr. J. J. WOODWARD to call this "typho-malarial fever," a term which he explains to be applied "not to a specific or distinct type of disease, but to the compound forms of fever which result from the combined influence of the causes of the malarious fevers and of typhoid fever."*

The name *Remitto-Typhus* was given to it by Dr. D. DRAKE, who also spoke of it as "the typhoid stage of remittent or autumnal fever." He does not consider it a distinct disease, but a genuine hybrid of typhoid and remittent fevers. He remarks that in many cases the stage of invasion is nearly the same length in both ; both attack males more than females ; and that when remittent terminates fatally ; subsultus tendinum, a dry tongue and intestinal hemorrhage are sometimes present. He has,

**Transactions of the International Medical Congress, 1876, p. 340.*

MALARIAL.

Eruption.

Eruptions of different kinds sometimes occur, but are so different in shape, feel, duration, number, extent and place, that they need never be mistaken for the typhoid eruption.

Mortality.

Very slight—not one fatal case in a hundred.

Lesions.

Hemorrhage from congestion of bowels rare ; congestion of stomach, lungs, liver and spleen, the two latter sometimes become enlarged.

however, never seen a decided intermittent pass into typhoid ; nor well-marked typhoid terminate in an intermittent.*

During and since the war, typho-malarial fever has attracted much attention, and its traits have been thus distinguished from simple typhoid :—

TYPHOID.

Occurs in all localities, most common in the north.

Invasion gradual and without remittance.

Daily exacerbation and remission slight.

Diarrhœa the rule. Tympanites common. Abdominal tenderness considerable ; epigastric and hepatic tenderness slight.

Temperature comparatively low. Delirium low and muttering.

Spleen not involved to the same extent.

Sordes on the teeth the rule.

Peyer's glands always involved.

Rose-colored eruption present.

Pigment deposits absent.

TYPHO-MALARIAL.

Only in miasmatic localities ; most common in the south.

Often begins as simple intermittent or remittent.

Decidedly marked.

Constipation the rule. Tympanites rare. Abdominal tenderness slight ; epigastric and hepatic tenderness considerable.

Temperature high, especially at outset. Delirium active.

Tumefaction of spleen very marked.

Sordes rare.

Rarely involved.

Generally entirely absent.

Pigment deposits in various tissues and organs very common.

THE TYPHOID STATE.

It is a common error to confound the typhoid condition which occurs in many diseases with typhoid fever, properly so-called. This typhoid state may be developed in typhus and other fevers, in acute pneumonia, rheumatism, tuberculosis, pyæmia, and various renal diseases, especially the granular or gouty kidney, and Bright's disease. The exciting cause in all these cases, it is believed, is the accumulation in the blood of the nitrogenous products of disintegration of the tissues.

The so-called "typhoid symptoms" are a quick, soft pulse ; a dry,

* " Diseases of the Interior Valley of North America," p. 556.

brown tongue; the phenomena and physical signs of hypostatic congestion of the lungs; impairment of the mental faculties; stupor passing into coma; delirium, which is at one time acute and noisy, at another low and muttering, and not unfrequently associated with muscular tremor; involuntary discharges. The skin is dusky, moist and often emitting a fetid odor. There is little thirst and often difficulty in swallowing. The temperature and urine vary considerably. The respirations are shallow and somewhat accelerated. The bowels are sometimes constipated, but often relaxed with offensive evacuations.

The difference between this condition, as it supervenes in the above named diseases, and true typhoid, or continued fever, may be thus presented:—

THE TYPHOID STATE.

Arises in the course of an antecedent local disease.

Is always traceable to blood-poisoning from deficient elimination.

Abdominal symptoms generally absent.

Occasionally there may be spots of diffused rosiness, from dilatation of superficial capillaries, but nothing like the *tâches rouges*.

Intestinal hemorrhage not to be expected.

Ague-cake not usual, except in malarious cachexia.

Urine may show albumen or pus.

TYPHOID FEVER.

Begins without any history of preceding disease.

Can often be traced to an external zymotic or septic influence.

Diarrhoea, tympanites, epistaxis, tenderness over intestinal glands, pain in iliac fossa.

Eruption of rose-colored spots.

Intestinal hemorrhage not infrequent.

Enlargement of spleen very constant.

Albumen and pus not present.

MALARIAL FEVERS.

The characteristic symptom of all malarial diseases is *periodicity*. It is not, however, pathognomonic; for hectic and syphilitic fevers, neuralgia and many other diseases, simulate this trait very closely. The diagnosis, however, in most instances is facile.

Intermittent begins with a chill, cold extremities, pale face, chattering

teeth and pulse feeble ; followed by a decided fever, the face flushed, the skin hot, the pulse full and rapid ; and ends with a profuse perspiration, soft, moderate pulse, and restoration of the secretions. This recurs at definite intervals, with complete intermissions between.

In *remittent fever* we find the same development of the phenomena, the chill, the fever, the perspiration, but without complete abatement of the febrile symptoms in the interval. They continue, though lessened, and usually have daily exacerbations. It is generally preceded by intermittent.

Between these two most common forms there are the differences that in intermittent the patient is well between the paroxysms ; in remittent he continues ailing ; in intermittent a distinct chill precedes each attack ; in remittent the chill is slight or absent ; in intermittent the appetite is good between the invasions ; in remittent nausea and anorexia are present. Dr. DANIEL DRAKE says : “ If we suppose an ague shake to be reduced to a mere chill, but the subsequent hot stage aggravated and prolonged, we shall form a just conception of the relations, in symptomatology, between intermittent and remittent fever.”*

The more intense cases of malarial poisoning develop *algid pernicious or congestive chills, malignant remittent fever, and malarial hemorrhagic fever.*

In *congestive chill* the symptoms of an ordinary intermittent are present, but in an exaggerated form. The chill is intense, the skin and even the breath seem cold ; the face is cadaveric ; the respiration is sighing ; the pulse scarcely distinguishable ; the shivering shakes the bed. When the stage of fever comes on the pulse is full and so quick that it can scarcely be counted ; the skin of the body is hot while the feet and hands are cold ; delirium is active ; thirst intense ; the stomach is irritable. The perspiration that follows brings no relief ; the patient lies prostrate and sometimes unconscious. When the congestion affects the lung there is a sense of smothering, difficult breathing and bloody expectoration ; when it attacks the stomach and bowels there are violent spells of vomiting, foaming or soap-like white discharges, and great epigastric tenderness. In these cases the mind is usually clear ; but when it is the brain which is involved, there is intense headache, the

* “ Diseases of the Interior Valley of North America,” p. 95.

mind is dull or delirious, and coma is apt to supervene. Patients rarely survive the third chill of this intensity.

The diagnosis of *malignant remittent* has been carefully set forth by Dr. DANIEL DRAKE as follows:—

1. The pulse does not rise in fullness and force during the exacerbation, as in other forms of remittent fever, but is generally small, frequent, weak and variable. When the remission begins it generally improves slightly, but to a much less extent than in mild remittents.

2. The feeling of abdominal oppression, and the anxiety, restlessness and gastric irritability are deeper in this than in other forms of remittent fever; and these symptoms never entirely cease during the remission.

3. A coldness in the hands and feet, or of the ends of the toes and fingers only, continues through the hot stage, while the trunk of the body and the head are in high fever heat. With the arrival of the remission this coldness, in milder cases, is replaced by a natural temperature; but in the more malignant it continues. Many experienced physicians regard this as the most characteristic sign of malignant remittent.

4. There is no time when the fever is absent; and whatever irritations or congestions are formed in the cold stage, and whatever inflammations are set up in the hot stage, remain, though moderated in degree, throughout the remission.*

Hemorrhagic malarial fever commences with a chill of the congestive type; and during the first paroxysms the symptoms which distinguish this from all other fevers usually make their appearance. These are jaundiced skin, and vomiting, apparently without any effort, of a dark fluid; the feces dark, offensive, and tawny looking; the color of the skin yellowish or bronzed, and the *urine colored with blood*. The last mentioned is pathognomonic. Sometimes the urine is profuse, though mixed with blood, which is a favorable symptom. Most of such cases recover; but when the urine grows scanty, and suppression ensues, the result is said to be always fatal.† The remissions are irregular and often ill-defined; and after the hot stage there is no perspiration.‡ Pain in the back is severe and incessant; the stomach is irritable, and the mental powers often obscured.

* *Loc. cit.*

† Dr. GREENSVILLE DOWELL, "Yellow Fever and Malarial Fever," p. 213.

‡ Dr. THACKER, *Cincinnati Medical News*, 1872.

The *tongue* presents in malarious diseases some peculiar appearances. One of these has been described as follows by Dr. WM. A. LOVE, of Atlanta :*—

“While the appearance of the tongue indicative of physiological and pathological conditions of the alimentary mucous membrane presents itself on the upper papillated surface, the *border* and *outer edges* present the peculiarity indicative of malarial toxæmia. It consists in a peculiar *pectiniforme* appearance of the edges of the tongue, as though these edges had been under the pressure of the sides of the teeth of a comb—just as, in certain “languid and flabby” states of the primæ viæ, we find the edges presenting a crenated appearance, produced by the indentations resulting from the pressure of the teeth in the oral cavity—just within this *pectiniforme edge*, making the outer border of the upper surface of greater or less width, in different cases; or in different degrees of malarial toxæmia there appears a *smooth margin*, both the *pectiniforme edge* and the *smooth margin* presenting a *cleaner appearance* and a *brighter hue* than the other portions of the surface of the organ.”

A characteristic color of the tongue in malarial poisoning has been observed by Professor CHARLES O. CURTMAN, M.D., of St. Louis. He describes it as almost uniformly present. The color of the dorsum of the tongue as far back as the circumvallate papillæ is of a bluish gray tinge, somewhat resembling that of old sheet zinc or lead. It occurs in various degrees of intensity, giving the impression of a coloring of greater or less thickness, superimposed upon the epithelial surface, sometimes quite thin and transparent, at other times quite opaque. In some cases this hue is observed without any other pronounced symptoms of malaria; but in all such the distinct malarial symptoms follow. The disappearance of this color serves as a valuable index of the perfect restoration to health.†

The symptoms of malarial poisoning are multiform, and are frequently so masked and disguised that the closest observation fails to detect their origin. This is the condition of *malarial toxæmia*. It is broadly characterized by a tendency to cerebral, thoracic and abdominal congestion, obstinate to ordinary remedies, and often slightly periodic in

* “*Transactions of the Medical Association of Georgia*, 1878.”

† *St. Louis Medical and Surgical Journal*, 1869.

exacerbations. Bronchitis, diarrhœa, simple fever, toothache, neuralgia, ophthalmia, urticaria and other skin diseases, even hæmoptysis, hysteria and rheumatism, may all be caused, instituted, or simulated by this subtle poison.

Careful examination will sometimes disclose evidence of periodicity in an increase of suffering at regular periods; sometimes at intervals of several days, or even weeks, apart; or they may be regularly aggravated at morning, noon or night. Sometimes subordinated to the prominent symptoms, and apt to be overlooked by the patient unless particular inquiry is made, are slight recurrent headaches, intolerance of light, shiverings, or a sense of cold, or alternating heat and cold, or perspirations. A trace of blood in the urine, especially in the tropics, is a common indication of malaria. Nausea or vomiting, or a copious watery discharge from the bowels at periodic intervals, are often observed, especially in children.* The skin is harsh, dry, and presents a muddy or else a greenish-yellow hue, which is most noticeable on the face, neck and arms. The appetite is capricious, the strength easily exhausted, the temper irritable, the mind readily depressed, and the energies diminished. On careful percussion the spleen is nearly always found to be decidedly, and the liver slightly, larger than in health.

The condition of the blood in malarial poisoning has been studied with definite results. Dr. A. KELSCH has found that the white corpuscles diminish during an attack to one-half or one-third of their normal number, and continue less than usual so long as there is splenic enlargement.†

Dr. E. L. MOSS, staff-surgeon in the British Navy, has constantly found, after the lapse of forty-eight or more hours, organisms in the blood of intermittent fever which he was unable to find in fresh blood. The organisms consist of bacteria, singly or in pairs, in active movement, sometimes stationary in zoöglea groups, occasionally in chains of four or more. Dr. MOSS's method would appear to exclude every possibility of infection. His apparatus consists of a series of small glass bulbs connected by capillary tubes, so that one bulb and its contents

*See an article on "Infantile Malarial Toxæmia," by Dr. JOEL C. HALL, in the *Medical and Surgical Reporter*, Vol. xxxi, p. 147.

† *Archives de Physiologie*, October, 1876.

can be separated from the balance by the blowpipe. The tubes and bulbs are so arranged that the entire apparatus can be heated in a water or paraffin bath. One end of the series is left open, packed with baked wool and connected with an aspirator; the other end is drawn to a fine point and sealed. The sealed point is connected with a fine hypodermic needle by a piece of rubber tubing; the needle is protected by a glass sheath. The apparatus is repeatedly heated in a water bath before using. To use the apparatus the sheath is removed from the needle and the latter is plunged into any suitable vein, the sealed point inside the rubber connection is broken and the blood flows gently through the bulbs, drawn on by the aspirator. When sufficient blood has entered, the tube next to the needle is instantly closed by the blowpipe, and then the end near the wool plug.

CEREBRO-SPINAL FEVER (EPIDEMIC MENINGITIS, OR SPOTTED FEVER).

The onset of this disease is usually sudden, often beginning with a chill, vomiting and intense headache, and an elevation of pulse and temperature. The pathognomonic symptom is that *the head is drawn backward and downward, and the muscles at the back of the neck are rigidly contracted, and very painful on motion.* The pupils are also contracted.

At an early period herpes may appear on the face or limbs, the skin is hyperæsthetic, and the patient cannot bear handling. After about four days convulsions may set in, or tetanic contractions make their appearance, and stupor follows, passing into a coma, preceding dissolution. The bowels are persistently constipated, and the urine passes involuntarily.

In cases tending toward recovery the acute symptoms gradually subside, and, after a week or two, convalescence takes place, attended by more or less headache and muscular contraction.

In regard to differential diagnosis, it may be simulated by typhus or masked variola. The absence of tetanic spasms of the post-cervical muscles in these diseases will aid in recognizing them. The protracted cases, where this symptom is not prominent, may resemble typhoid fever. In both there is an eruption, some similar cerebral symptoms,

and occasionally intercurrent diarrhœa. But the invasion of cerebro-spinal meningitis is more sudden, the headache more violent, and there is vomiting and constipation; while later the spinal pain, the herpes, the tetanic spasms and the continued headache, are broad distinctions.

True tetanus is distinguished by the absence of epidemic prevalence, by the clearness of the mental powers, and by the history of the case pointing to some injury.

Certain forms of malignant malarial fever counterfeit cerebro-spinal meningitis, especially during convalescence, when the affection presents periodical intermissions of the febrile state. The points of difference may be summed up as follows (HAMILTON):—

CEREBRO-SPINAL MENINGITIS.	CONGESTIVE PERNICIOUS MALARIAL FEVER.
Inceptive chill not marked.	Chill quite marked.
Disease epidemic, and chiefly among children.	Epidemic and common to all ages.
Muscular spasms the rule.	Muscular spasms very rare.
Bowels constipated.	Not usually so.
Pulse and temperature do not suffer rapid variations.	Both subject to great variations, feeble and irregular.
Temperature does not undergo periodical changes.	Undergoes decided periodical changes.
Face flushed; eruption before fourth day.	Complexion sallow; no eruption.
Delirium and coma not affected by large doses of quinine.	All symptoms modified usually by large doses of quinine.
Increase of fibrin and rapid coagulation of blood when drawn.	

In distinguishing it from other head affections it should be observed that, while pain in the head, vomiting, epileptiform attacks, disease of the optic discs, emaciation, eruptions, involuntary micturition, are symptoms found in many of them, the sudden onset of symptoms, pain in the back of the neck, the stiffness of the muscles of the neck, and retraction of the head, are sufficient to separate cerebro-spinal

meningitis from *hydrocephalus acquisitus*, *basilar meningitis*, and *tumor of the brain*, diseases to which, in its symptoms, it is nearly allied.

It may also be noted that Dr. HAYDEN, of Dublin, a competent authority, states that he never saw a case of cerebro-spinal meningitis unattended by *pains in the calves of the legs*, and he should make a presumptive diagnosis from the presence of that symptom alone.

Dr. DOWSE, of London, has insisted on the importance of distinguishing *sporadic* from *epidemic* cerebro-spinal meningitis. He maintains that in its epidemic form the sensorium is more or less affected from the first, and that the membranes over the superior cerebral convolutions, cerebellum, and posterior columns of the cord, including the nerve substance, are primarily, if not wholly, the seats of lesion. In the sporadic form, on the contrary, the sensorium and special senses are only slightly influenced, and the inflammation centres itself upon the meninges at the base of the brain and the anterior columns of the cord. He therefore gives to the latter affection the name of *occipito*, or *basic cerebro-spinal meningitis*, in contradistinction to the former well-known disease. He draws his conclusions and diagnosis from signs and symptoms, as evidenced in the following table:—

EPIDEMIC CEREBRO-SPINAL MENINGITIS.	SPORADIC OR BASIC CEREBRO-SPINAL MENINGITIS.
Attack sudden, without any special predisposing cause.	Attack commences gradually and resembles an onset of acute rheumatism.
Apparently of a contagious or infectious origin.	Usually arises from exposure to cold, exhaustion, and privation.
Sensorium affected from the first.	Sensorium never affected until the last stage.
Excito-motor spasms of a tonic character in groups or groupings of muscles, with marked loss of cutaneous and muscular sense.	Incoördination of movement with cutaneous formication, partial anæsthesia, muscular hyperalgia, but no tetanic spasms.
Reflex movements common.	Reflex movements rare.
Vomiting urgent and uncontrollable.	Vomiting not so severe.
Temperature rarely exceeds 100°.	Temperature often rises to 105°.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.	SPORADIC OR BASIC CEREBRO- SPINAL MENINGITIS.
Purpuric maculæ diffuse and general.	Maculæ never seen in the desudate form.
Death usually takes place from coma.	Death usually takes place from apnoea.
Prognosis grave.	Prognosis hopeful.
Post-mortem appearances reveal the membranes over the superior cerebral convolutions and posterior columns of the cord as the seat of lesion.	Post-mortem appearances reveal the membranes over the base of the brain and over the anterior columns of the cord as the prime seat of lesion.

This distinction has, however, not been wholly accepted by American authorities. Dr. DA COSTA questions the main point of difference—the temperature; and Dr. ALFRED STILLÉ writes: “The whole medical literature does not contain a single case of *sporadic* idiopathic cerebro-spinal meningitis with the characteristic *sudden onset* of the epidemic disease.” From that writer’s admirable monograph* we extract the following exhaustive comparison of meningitis and typhus, with which latter it has often been confounded:

EPIDEMIC CEREBRO-SPINAL MENINGITIS.	TYPHUS FEVER.
A <i>pandemic</i> disease; occurs in places remote from one another and without intercommunication.	Essentially an <i>endemic</i> disease. Always due to local causes. Spreads by intercommunication only.
Attacks all classes of society. Is never primarily developed by squalor and deficient ventilation.	Attacks primarily the poor, filthy and crowded alone.
Is not contagious.	Contagious in a high degree.
More males than females attacked.	The two sexes equally affected.
More young persons than adults attacked.	More adults than young persons.
Generally occurs in winter.	Epidemics irrespective of season.

* “Epidemic Meningitis,” pp. 107, 117.

MENINGITIS.

Eruptions are wanting in at least half the cases; they occur within the first day or two.

The eruptions are very various, including erythema, roseola, urticaria, herpes, etc. Ecchymoses are common.

Headache acute, agonizing, tense.

Delirium often absent; often hysterical, sometimes vivacious, sometimes maniacal. Generally begins on the first or second day.

Pulse very often not above the natural standard; often preternaturally frequent or unfrequent. Is subject to sudden and great variations.

The temperature is lower than that recorded in any other typhoid or inflammatory disease. It is also very fluctuating.

The body emits no peculiar smell.

The tongue is generally moist and soft; sordes of the teeth, etc., is rare.

Vomiting, generally of bilious matter, is an almost constant and urgent symptom, especially in the first stage.

Pains in the spine and limbs of a sharp and lancinating character are usual, and evidently neuralgic.

Tetanic spasms in a very large proportion of cases, and within the first two or three days.

TYPHUS.

The eruption is rarely absent, and appears between the fourth and the seventh day.

The eruption is uniformly roseolous, and then petechial. Ecchymoses are rare.

Headache dull and heavy.

Rarely absent; usually muttering. Rarely begins before the end of the first week.

A slow pulse exceedingly rare; its rate pretty constantly between 90° and 120° .

The temperature is always more or less elevated, and it does not fall until the close of the disease. The skin is hot, burning, and pungent to the touch.

The mouse-like odor of typhus is characteristic.

The tongue is generally dry, hard and brown, and the teeth and gums fuliginous.

Vomiting is rare and not urgent.

Pains are dull, heavy, and apparently muscular.

Tetanic spasms are unknown in typhus. Convulsions sometimes occur, due to pyæmia.

MENINGITIS.

Cutaneous hyperæsthesia a prominent symptom.

Strabismus common. The eye, if injected, has a light red or pinkish color. The pupils are often unequal.

Deafness is often complete and permanent.

Duration very indefinite; but generally from four to seven days.

The blood is often highly fibrinous.

The lesions, unless in the most rapid cases, consist of a fibrinous or purulent exudation in the meshes of the cerebro-spinal pia mater.

Mortality from 20 to 75 per cent.

TYPHUS.

The sensibility of the skin is generally blunted.

Strabismus rare. The blood in the conjunctival vessels has a dark hue; the pupils are always equal.

Deafness is hardly ever permanent, or attended with signs of disorganization of the ear.

Duration from twelve to fourteen days.

Blood never fibrinous.

There are no inflammatory lesions whatever.

Mortality from 8 to 40 per cent.

ACUTE TUBERCULAR (GRANULAR) MENINGITIS.

This serious disease is apt to be confounded, especially in the adult, with typhoid or typhus fever, the exanthemata and pneumonia. The following characteristics of the disease, as given by Drs. REGINALD SOUTHEY and HAMILTON, will serve to distinguish it:—

1. The prodromal symptoms of this form of meningitis are well marked. The history of the case usually records an illness that has endured some two or four weeks, but one which has not attracted much attention until distracting headache, with some delirium at night, has supervened.

2. Vomiting is generally the first and most important symptom. Headache is invariably present.

3. After two or three days there is a marked rise of temperature, say from 101° to 105°, with greatly increased pulse.

4. The bowels are constipated and not tender to firm pressure. Very little nourishment is voluntarily taken. The abdomen becomes retracted, and the aspect of the patient, with half-open eyelids, or some slight paralysis of these, becomes highly diagnostic.

5. There is no characteristic rash. The so-called *tâche cérébrale* of this form of meningitis is not a true eruption, but is produced by pressure or contact. When the finger is drawn across the skin of the forehead it leaves a vivid red mark, which has been considered a pathognomonic sign of the disease.

6. The skin is hyperæsthetic, the delirium slight and transitory, the temper irritable, obstinate and unaccommodating.

7. There are general muscular pains, followed first by stiffness, and then by slight paralysis, as shown in the imperfect coördination of the muscular movements, in tremblings and in twitchings. The muscular pain and stiffness are often first complained of in the nape of the neck, and then in the muscles of the back.

8. Slight epileptiform convulsions are observed, followed by paralysis of motion in the limbs or parts convulsed; this paralysis being most usually of a transitory or temporary kind. Among the paralyzes most characteristic are those affecting the optic commissure and oculo-motor tracts, causing a slight internal squint, with dilated inactive pupil of one eye, with drooping of the same eyelid, and paralysis of the facial nerve upon one side. The paralysis of the limbs, although usually hemiplegic, is seldom one that invades the body upon one side in its entirety. Further, its mode of attack is gradual; usually, the arm and leg are affected upon the same side, even when the facial muscles are not involved.

YELLOW FEVER.

The name *Yellow Fever* is misleading, as the coloration of the skin to which it refers is not an invariable nor even a common sign of the disease. According to Dr. GREENSVILLE DOWELL,* the skin does not turn yellow in more than one case in six, and many die before there is the least appearance of yellowness even in the eyes. Of those who die after the black vomit has set in, not more than one in three presents the yellowness.

The pathognomonic sign of the disease is the *black vomit*. It is brownish black, semi-fluid, with a glistening reflection and acid reaction, and varies in quantity from a mere stain on a handkerchief to many pints in the twenty-four hours. It, however, is not thrown up in more than one in three fatal cases.

* "Yellow Fever and Malarial Diseases."

The usual course of the disease as witnessed in the southern and southwestern States is as follows:—

1. Onset with a chilly feeling along the spine, passing into actual rigor.
2. Pain in the head, severe in proportion to the malignancy of the disease.
3. Fever slight, tending to perspiration.
4. Remission after a period varying from twenty-four hours to five days.
5. The secondary fever, commencing usually without a chill; it runs an indefinite course.

The discoloration begins at the white of the eye, and extends over the skin of the forehead, chest, abdomen, and extremities. The urine is high-colored and stains linen, and in some cases the perspiration gives the same yellowish stain.

The shades which separate the symptoms of one fever from those of another, in warm climates, are sometimes of such gentle gradation that *primâ facie* they may seem to belong to one and the same disease; this more especially refers to the yellow and remittent type of fevers, between which so slight is sometimes the distinction, that bilious remittent has frequently been considered and classified as true yellow fever; for in the prominent symptoms which appear in both yellow and remittent fever a great similarity obtains; both take their origin in paludal soils; both in their course offer symptoms of so seemingly similar a nature that the shades of difference are so slight as to frequently escape even a good observer and cause him to fall into error. But this apparent similarity vanishes on close and continuous inspection, for then essential and distinctive marks are observed, which stamp each with an individuality, and which characterize each as a separate disease, distinct in its essence and differing signally the one from the other. These differences may be summarized as follows (J. J. L. DONNET, DACOSTA, DOWELL, and others):

YELLOW FEVER.

Is essentially of an infectious nature, and found in cities.

Chiefly vigorous and young constitutions fall victims to it. Colored population *less* liable than white.

BILIOUS REMITTENT.

Is not of an infectious nature, and usually found in the country.

All ages and constitutions are liable, and the weakest most so. Colored population liable.

YELLOW FEVER.

Restricted chiefly to the yellow fever zone.

Is of a continued type; remissions not marked.

Temperature in bad cases very high.

Usually attacks at night.

Severe nausea and vomiting throughout. Epigastric tenderness early and decided black vomit. Headache occipital.

Hemorrhages from the gums and various parts of the body.

Tongue clean or but slightly coated; pulse variable, becoming slow in the last stages.

Eye highly injected and humid; expression often fierce or anxious.

Pain in the back very severe; also pain in the calves and over the eyes.

Delirium rare; mind generally clear and cheerful.

Urine generally albuminous; suppression common.

Muscular prostration slight; convalescence rapid; no sequelæ.

Liver affected and slightly enlarged.

Spleen not affected.

One attack affords an almost certain immunity.

Mortality very high.

Peculiar smell often perceptible.

BILIOUS REMITTENT.

Is to be found in all parts of the world where marshy soils prevail.

Remissions observed in the morning.

Temperature not extraordinarily high.

Usually attacks in daytime.

Nausea and vomiting moderate. Epigastric tenderness slight. Headache frontal.

No hemorrhagic tendency.

Tongue heavily coated; pulse varies little, remaining quick until convalescence sets in.

Eye and physiognomy not peculiar.

Rachialgia slight or absent; headache moderate.

Delirium frequent; mind always dull.

Albuminous urine rare; suppression also rare.

Much muscular prostration; convalescence slow; sequelæ various and tedious.

Liver not affected.

Spleen invariably affected.

One attack seems rather to predispose to others.

Mortality slight.

No peculiar smell observed.

YELLOW FEVER.

Never merges into intermittent.

Treatment unsatisfactory; quinine useless.

Autopsies show great congestion, inflammation, ulceration and softening of the stomach. Liver enlarged, fatty, yellowish in color, its secreting cells filled with oil globules. Heart often exhibits disintegration of the muscular fibres.

BILIOUS REMITTENT.

Often merges into intermittent.

Quite amenable to treatment; antagonistic power of quinine beyond question.

Autopsies show congestion of the stomach, but rarely inflammation. Liver of an olive or bronze hue, not fatty. Spleen enlarged.

RELAPSING FEVER.

Of late years epidemics of this disease have appeared at various points in this country. It is eminently contagious in character, and a physician should be prepared to recognize it early. The invasion is sudden, the fever soon developed and high, the pulse very rapid, the skin often jaundiced, and the temperature elevated (106° – 107°). Toward the close of the first week the symptoms rapidly subside, and convalescence seems at hand; but after about another week the symptoms all return with as much violence as ever, to again disappear, as a rule, after four or five days.

The epidemic prevalence of the disease, its sudden invasion, the persistence without remission of the high febrile symptoms, and the a-febrile interval, give it a peculiar physiognomy.

The characteristic feature of the disease, asserted by some to be truly pathognomonic, is the presence of *spirillum in the blood*. The following method of demonstrating them is that recommended by Dr. R. ALBRECHT, of St. Petersburg:*

Spread out a drop of blood on a slide, not too thin; let it dry; treat it with a drop of acetic acid, and repeat it in a few seconds. By this means all the fibrin and blood-corpuscles will be destroyed and dissolved, and after careful washing away of the acid with distilled water, and final drying, the preparation is ready for use. With a little care in washing, which must not be in a stream, the spirilla are not lost, especially if the preparation has been dried for six to twelve hours before being treated

* St. Petersburg *Med. Wochenschrift*, June, 1878.

with acetic acid. The glass slide then looks quite transparent, and, at the place where the drop of blood was, it looks a little dusty. Under the microscope the nuclei and nucleoli of the white blood corpuscles are visible, and between these the spirilla lie in great numbers and in the most distinct arrangement and position, showing up very beautifully and distinctly. They give the impression of being thicker than they generally are, probably because they are no longer imbedded in a highly refracting substance—plasma.

Relapsing fever is liable to be mistaken for one of the forms of continued fever. Its epidemic prevalence will naturally put the physician on his guard. It is, moreover, especially a disease of the lower classes, who suffer from insufficient food and filthy surroundings. In most cases it is associated with jaundice, which is a rare complication in typhoid. When the disease rapidly abates, and this cessation is followed by the characteristic relapse, no reasonable doubt as to its nature can be entertained. The main distinctions between relapsing and typhoid may be thrown into a comparative view as follows:—

RELAPSING FEVER.	TYPHOID FEVER.
Invasion sudden.	Invasion gradual, with epistaxis; no chill.
Bowels generally constipated	Generally diarrhœa.
Conjunctivitis.	No conjunctivitis; eyes bright and clear.
Liver engorged, skin yellow, tenderness over epigastrium.	No yellowness; tenderness over right iliac region.
Temperature high, 105°-107°.	Temperature during first week rarely above 104°.
Fever abates in three or four days, with critical sweats; diminution or cessation of the febrile symptoms, with subsequent relapse.	These phenomena absent; symptoms continuous for three or four weeks.
Spirilla in the blood.	No spirilla.
Splenic enlargement.	Spleen not materially enlarged.
No characteristic eruption.	"Rose spots," inflammation of Peyer's glands.

CHAPTER II.

DISEASES OF THE BLOOD.

CONTENTS.—*The Dyscrasiæ—The Arthritic, Dartrous, or Rheumic Dyscrasia—The Scrofulous or Strumous Dyscrasia—The Syphilitic Dyscrasia—The Tuberculous Dyscrasia—Rheumatism—Chronic Rheumatism—Gout—Rheumatic Arthritis—Pernicious Anæmia and Leukæmia.*

THE DYSCRASIAE.

As is justly remarked by Professor THEODOR BILLROTH, in his *Surgical Pathology*, while it is true that there are some objections to the employment of the term *dyscrasia*, as committing one to a humoral pathology, these are overbalanced by the fact that there are certain well-defined, long-recognized, inherited physical peculiarities, which render the person possessing them unusually prone to certain diseases and complications, and which lend a complexion of their own to very many affections seemingly remote in form and pathology.

These constitutional tendencies may as well be known by the term *Dyscrasiæ* as by any other, since their existence cannot well be denied.

The principal dyscrasiæ are: 1. The *arthritic*, sometimes called *dartrous* or *rheumic*, believed to be pathologically akin to *gout* and *rheumatism*; 2. The *strumous*, or *scrofulous*; 3. The *syphilitic*; and 4. The *tuberculous*, or *phthisical*; the last three mentioned, in the opinion of some, being derived from a common ancestral taint.

I. THE ARTHRITIC, DARTROUS, OR RHEUMIC DYSCRASIA.

This form of blood poisoning has been aptly termed, by Mr. JONATHAN HUTCHINSON, "the basis-diathesis on which both *gout* and *rheumatic arthritis* are built, and which to a large extent is indifferent and common to both." When a man with such a diathesis becomes affected with a renal disease, *gout* develops itself; otherwise he will probably have

rheumatism. In many families it is observed that the males have gout, the females rheumatism. The explanation is not far to seek.* In another lecture Mr. Hutchinson describes gout as “chronic rheumatism *plus* a dietetic derangement.”

Many skin diseases, nervous affections (so-called), “cramp colic,” headaches, sciatica, vertigoes, palpitation, and obstinate dyspepsia are really latent gout. In such cases there is usually a history of antecedent or hereditary rheumatic diathesis, frequent acid eructations, the emission of pale, limpid, acid urine, of low specific gravity, and with traces of sugar or albumen or both; some varicosity of the veins; the nails are brittle; and there is slight redness around the eye, indicative of mild chronic conjunctivitis (Dr. J. RUSSELL REYNOLDS).

The following are the signs as stated by Professor HARDY, of Paris: Persons who have this diathesis appear to enjoy good health, but their skin is habitually dry and their perspiration scanty. They often experience a lively itching without eruption. The appetite is generally well developed, and they are apt to eat a much greater quantity of food (especially animal food) than others in analogous conditions. Another important peculiarity is the extreme sensibility of the skin and the facility with which it is influenced by the lightest and most fugitive impressions. Sometimes general excitement, alcoholic excess, watching, use of coffee, of certain kinds of food; sometimes a local excitement, irritating frictions, or the application of a plaster, will give rise to an eruption, often ephemeral, which reveals a peculiar predisposition of the economy, and the existence of a latent vice which needs but a favorable occasion to manifest itself.

To this diathesis HARDY ascribes eczema, lichen, psoriasis and pityriasis, among diseases of the skin.†

Mr. PRESCOTT HEWETT adds that when a patient complains of dyspepsia, more or less troublesome, frequent deposits of lithates in the urine, slight eczematous eruptions on the skin from time to time, anomalous wandering pains in various muscles, sharp, deep-seated pains in the tongue, continuing for two or three days, and then disappearing altogether for a while, crackling about the cervical spine on slight move-

* *Medical Times and Gazette*, June, 1876.

† *Maladies de la Peau*, Paris, 1860.

ments, some, it may be very slight, knottiness about the smaller joints of the fingers—we may be very certain that he has the arthritic diathesis.

Sir JAMES PAGET adds to the above: Small (chalky) nodules in the cartilages of the ears (*tophi*); nodular enlargement of the knuckles; thickening of the cutis, with subcutaneous bursæ over the knuckles, chiefly between the first and second phalanges of the fingers; thickening of the palmar fascia, adhering to the cutis, and producing contraction of the fingers; spontaneous pain in the tendo-Achillis; pain in the heel; frequent and persistent erections at night, not connected with any sexual feelings; “burning soles” and “burning palms;” sensations of heat; tingling and burning patches of the skin of the thighs, without external appearances of redness or eruption; patches of “dry eczema.”

In such patients, an injury may be followed by a well-marked attack of gout; or the trouble may linger, with pain and occasional swelling, and with constantly increasing distrust of surgery and the surgeon, till some one suspects the existing taint of the arthritic diathesis, and acting on the suspicion, addresses his remedies to it, and promptly cures the local trouble.

(The points of diagnosis between the gouty diathesis and chronic rheumatism, as summed up by FOTHERGILL, are given on page 64, in the section devoted to “Diseases Likely to be Confounded with Rheumatism.”)

II. THE SCROFULOUS OR STRUMOUS DYSCRASIA.

Sir JAMES PAGET defines the principal signs of scrofulous constitution to be slowly progressive and long abiding inflammation, provoked by less causes than would excite inflammation in healthy persons, the inflammatory process tending to the production of “cheesy” matter; the middle permanent incisors, with their borders barred, crenated, thin and brittle; the mucous membrane of the lower turbinated bone swollen, puffed and congested; a long abiding ozæna in early life, with frequent or daily discharge of scabs; general swelling, with glandular enlargement of the whole naso-palatine mucous membrane; a granular pharynx, with its lining membrane more or less thickly scattered with prominent glands; the perforating ulcer of the nasal septum—these are some of the minor signs. Still more positive are enlarged and suppurating lymph glands

discharging curdy pus, and slowly healing with red-banded and barred scars; pustules by the edge of the cornea; frequent impetigo with swollen glands; periosteal swellings of the phalanges; chronic thickenings of synovial membranes; obstinate otorrhœa. If a patient is found to have or to have had any few of these, he may justly be pronounced scrofulous, and scrofula may be suspected in any localized morbid process in him. Or, if these diseases are known to have occurred singly or together in many members of a family, we should look out for scrofula as an element of whatever disease may appear in any member of that family.

Dr. FRANCIS DELAFIELD, of New York city, observes* that practitioners in this country see comparatively so little of scrofula that it is difficult for them to appreciate the prominent place it holds in the minds of physicians in European countries. It is a condition which is hardly susceptible of a definition, and yet it is not hard to understand what is meant by the term.

It means this: When an individual acquires an inflammation of a mucous membrane, of the skin, of the joints, of the bones, of the genito-urinary apparatus, or of almost any part of the body, such an inflammation usually runs an acute course and terminates in resolution, or in suppuration, or in the formation of organized new tissue. But, if the inflammation, instead of doing this, simply reaches a certain point and stays there, and then, instead of resolving or suppurating merely, goes through a succession of degenerative changes, such an inflammation is said to be scrofulous.

The scrofulous inflammations have several well-marked characteristics. They are very slow in their progress; they are very rebellious to treatment; they are accompanied by an extensive cellular infiltration of the inflamed parts, so that when the degenerative changes ensue there is large destruction of tissue. The degeneration which occurs in the products of such a scrofulous inflammation is peculiar in its nature; it is commonly called cheesy degeneration, and consists in the transformation of the products of inflammation into a dry, yellow mass, composed of amorphous granular matter. Examples of this form of inflammation will at once suggest themselves. Caries of the vertebra, hip-joint disease, white swelling of the knee-joint, scrofulous orchitis, and enlarged lymphatic glands, are all of frequent occurrence.

* *N. Y. Medical Record*, Vol. x, p. 338.

III. THE SYPHILITIC DYSCRASIA.

Apart from the special recognition of constitutional syphilis, it is of the utmost importance for the physician to be on the alert to recognize and meet the syphilitic dyscrasia as it exists, (1) in the infantile period of life by inheritance, and (2) in advanced years developed, or in the condition of *latency*.

Mr. JONATHAN HUTCHINSON (*loc. cit.*), states that in the infantile period we recognize syphilis by the peculiarity of certain single symptoms, or else by the peculiar grouping of several different symptoms.

The *rash* on the skin is one of the commonest evidences. It is usually erythematous or papular, of a peculiar red or coppery tint, in abruptly-margined patches. Pustular, vesicular and bullous rashes and condylo-mata about the anal orifice are also frequent.

The *snuffles*, a peculiar, obstinate coryza, is almost always present.

Iritis and a tendency to deep-seated inflammation of the eyes are often met with.

At or about the age of one year, if the child survives, these symptoms usually all disappear, and the disease enters upon its stage of *latency*.

To detect its presence in the system at this period, we must first look to the evidences of past disease.

A sunken bridge of nose, caused by the long continued swelling of the nasal mucous membrane when the bones are soft; a skin marked by little pits and linear scars, especially near the angles of the mouth; the relics of an ulcerating eruption in early life; a protuberant forehead consequent upon infantile arachnitis; clouds in the cornea from past iritis—are all signs pointing to the constitutional taint.

The *teeth* furnish valuable aid in diagnosis. The upper central incisors are narrow and short, and notched in the centre in a half-moon shape, a shallow furrow running from this notch to the gum; the canines are narrow, rounded and peg-like; there are usually interspaces between the teeth. This may be considered an almost absolute sign of the taint.

The general growth is not often retarded, but the *complexion* is an important indication. It is exceedingly rare to meet a florid, good complexion in a young adult who is the subject of this taint. It is almost always pale.

Such persons, seemingly in full youth and vigor, generally have little

spontaneous physical energy ; they do not seek athletic exercise nor the trials of strength ; and are languid in motion.

Other signs which may be mentioned are : a patch upon the choroid, an optic irregular neuritis ; a faint interstitial keratitis ; an unequal thickening of the vocal cords, with cicatrices of old ulceration ; and last, but not least—and especially where syphilis is associated with a gouty habit of body—psoriasis upon the sides of the tongue, as well as an indurated irregular thickening of the lower bowel.

IV. THE TUBERCULOUS DYSCRASIA.

There are families in which the children, while apparently healthy during their development, perish early in adult life with tubercular manifestations, especially in the lungs. This indicates a peculiar inheritance, which may be called the tubercular dyscrasia. More frequently the children of decidedly strumous parents die in infancy, with tubercular meningitis, which furnishes ground for the belief that in many instances tubercular disease is brought about by the strumous dyscrasia ; and, indeed, it is by many identified with it. The physical characteristics of scrofulous subjects belong also to the majority of consumptives, in a greater or less degree. Others are predisposed to the disease through defective oxygenation caused by unfavorable form of the thoracic walls. But the researches on this subject are still incomplete, and it is well to bear in mind the words of Dr. A. T. H. WATERS :—

“There is no temperament which does not furnish victims to consumption ; nor can we say that there is any conformation of the body which is characteristic of the phthisical. I have seen men and women with the best developed frames and the most ample chests attacked with phthisis. You must not, therefore, be misled, by the existence of these conditions, by the appearance of robustness in your patients, into imagining that they cannot possibly become the subjects of this disease.”

The diagnosis of these different conditions is made less difficult by bearing in mind their peculiar tendencies and characteristic manifestations, as set forth in the table upon the next page.

SCROFULOSIS.
More particularly limited to childhood.

Affects especially the lymphatic glands (causing abscesses), the mucous membranes (ophthalmia), the skin (obstinate cutaneous diseases, especially the pustular) and bones (caries and necrosis, abscesses indolent (cold abscess). Frequently resulting in phthisis and hydrocephalus.

Generally afebrile.

Temperament phlegmatic; mind and body backward; skin muddy; upper lip thick; nostrils wide and alæ thickened. Abdomen tumid; ends of bones large; shafts thick. Otorrhœa, ozæna, ophthalmia common.

Mercury prohibited.

TUBERCULOSIS.
Not specially limited.

Affects internal organs (phthisis, hydrocephalus, peritonitis, tubercles of mesenteric, or bronchial, glands).

Pyrexia marked in acute cases, irregular in chronic.

Nervous system highly developed; mind and body active; organization delicate and refined. "Thin skin, clear complexion, the surface veins distinct, eyes bright, pupils large, eyelashes long, hair silken, face oval, ends of bones small, shafts thin, limbs straight" (JENNER).

Mercury only for temporary indigestions, etc., used carefully.

INHERITED SYPHILIS.
Manifests itself early, generally before third month (from fourteen days to six weeks).

Prominent symptoms:—
Cachectic appearance, snuffles, condylomata around the anus. Child thin, poorly nourished, muscles flabby. Skin brownish, cracked, thick and rough. Fontanelle open; ossification slow. Posterior cervical glands enlarged. Second set of incisors characteristic (Hutchinson teeth). The central incisors short, narrow and thin, chisel-shaped; edges soon become notched and broken; also striped or ribbed horizontally. Hair thin, and may have alopecia. Eruptions copper-colored and chronic; generally dry, but may be pustular (erythema, lichen, psoriasis and eczema, or impetigo, ecthyma and pemphigus), often seen on palms of hands or soles of feet. Liver enlarged (albuminoid). Ascites common, with tympanites.

Pyrexia only in complications.

Physically and mentally inferior in structure, and slow in movement; inactive, dull and often cachectic looking.

Mercury the sheet-anchor.

They resemble each other in hereditary character, their familiar manifestations being readily induced by defective hygienic conditions.

DISEASES LIKELY TO BE CONFOUNDED WITH RHEUMATISM.

Ordinarily an attack of acute rheumatism is recognized without difficulty by the pains in the joints, their swelling and tenderness, the shifting character of the disorder from joint to joint, and the absence of the symptoms so common in continued fevers, of disturbance of the stomach and brain (if we except the so-called cerebral rheumatism which appears to be associated with a uræmic condition, if its symptoms are not in reality occasioned in this way), as well as of the intermissions or remissions of periodic fevers.

Nevertheless it is true, as remarked by Dr. S. O. HABERSHON,* that while there are many characteristics of true rheumatic disease, few maladies are more easily mistaken, and there is *no* sign which is *uniformly* present. Pain is, perhaps, the most constant indication, with stiffness of one or more joints; but rheumatic pericarditis may, and often does, exist without any pain whatever. The same may be said in reference to febrile symptoms, to increase of temperature, and to changes in the urine; none of these signs is pathognomonic. Many maladies are designated rheumatic which have no connection with that disease.

1. *Diseases of the spine* are often said to *commence* with an attack of rheumatism; but it will generally be found that the pain in the course of the nerves or in the fibrous tissues arises from direct implication of the nerves or their centres.

2. The same remark applies to pain produced by the pressure of *cancerous*, *aneurismal*, or other tumors. Thus cancerous disease of the lumbar glands is often mistaken for lumbago; so also the pain from aneurismal disease of the thoracic and abdominal aorta, when no pulsating tumor can be detected, is referred to rheumatism.

3. During the course of *renal disease*, abnormal irritation arises not only in the serous membranes, producing pericarditis, pleurisy, peritonitis, etc., but a similar change happens with the synovial membranes, and a form of disease is induced which simulates rheumatism.

4. In chronic poisoning by *lead*, vague pains in the fasciæ, as well as in the joints, have been designated "saturnine arthralgia."

* *Half-Yearly Compendium of Medical Science.* III.

5. *Periosteal disease* is occasionally a source of fallacy in the diagnosis of rheumatism.

6. *Shingles* or *herpes zoster* may be found in the course both of the cerebral and spinal nerves; and the severe pain which precedes the eruption of the vesicles, and which also follows their disappearance, closely simulates local rheumatism.

7. A more important disease, and one which is attended with fatal issue, is *pyæmia*. It closely resembles rheumatism; for, with rigor and febrile symptoms, there is fixed pain and swelling in the joints—first one and then another being affected, *though without subsidence of those parts first attacked*. But while there may be some similarity in the symptoms, the prognosis is widely different. The one is generally a curable disease; the other a fatal one.

8. *Acute synovitis* closely resembles rheumatism, having pain and heat in the joint, with distention. But as a rule it affects only one joint; it is never subject to metastasis; and there is little or no effusion into the surrounding tissue. The accumulation of fluid in the joint is greater, but the constitutional symptoms are less prominent.

9. *Milk leg* occurs after fevers, or, in women after confinement. The limb swells throughout, becoming white, firm, hot and shining, and pits but little on pressure. The history of the case and appearance of the limb are usually sufficient to form the diagnosis.

CHRONIC RHEUMATISM.

The most common form of chronic rheumatism is that which affects the muscles, and it is frequently by no means easy to distinguish the pains due to the rheumatic diathesis from those of a wholly diverse etiology.

The principal distinctions are—

1. From *neuralgia*. Neuralgic pains are usually confined to the distribution of one nerve; they are not increased by motion or pressure; they are not attended with diffused soreness; and they are variable in intensity, and are not attended with acid secretion. The tender spots of Valleix may be detected along the trunk of the nerve or in its distribution.

2. From the pains of *organic lesions*. These are usually so clearly

localized as to point to their origin. Nevertheless the pain in the right shoulder, symptomatic of hepatic disease, and especially of an abscess approaching the serous surface of the liver, and the sympathetic pain down the left arm in some cases of heart disease, are often carelessly looked over, and their significance unheeded, by classing them as rheumatic. Intercostal rheumatism has included pleurisy, pleurodynia, broken ribs, herpes, neuralgia, the peculiar pain, generally of the left side, found in women and connected with menorrhagia and leucorrhœa; the pain on either side, which is intimately connected with debility and anæmia; and again is confounded with that condition of pain and soreness of the muscle developed by overwork, and attended with both muscular and cutaneous hyperæsthesia, designated by INMAN "myalgia."

3. *From the osteocopic pains of syphilis.* The history of the case throws some light; but as this often cannot be had, it should be remembered that syphilitic periostitis evinces a decided partiality for the periosteum and shafts of the long bones, and is very generally accompanied by nodes, especially in the anterior surface of the tibia, which are almost pathognomonic. There is often, too, a more marked cachexia than is found along with non-specific rheumatism. The clavicle, humerus, and forearms, are frequent locations of this form of rheumatism. As well as its favorite seats and accompanying nodes, there are evidences of skin and throat affections, a mutilated iris, etc., which will assist in forming a correct diagnosis. Furthermore, the ready response to a specific treatment aids in distinguishing syphilitic pains.

4. *From progressive locomotor ataxia.* Ataxic patients often bitterly complain of supposed rheumatic pains. These pains, in locomotor ataxy, come on in severe pangs—"stabbing, boring, shooting like lightning, flitting from one place to another in a very erratic manner, and recurring in paroxysms lasting from a few minutes to many hours." Their suddenness is their especial characteristic, and should always put the medical observer on his guard to look out for the other indications, as loss of tactile sensibility, etc. These pains may be accompanied by a feeling of coldness, thus closely simulating some forms of rheumatism. The importance of them lies in the prognosis, as the causes of locomotor ataxy are not to be relieved by art, although the pains may be mitigated by anodynes and frictions.

5. The *pains of chronic renal disease* often closely simulate *lumbago*, or muscular rheumatism of the loins. No clinical distinction can be positively drawn, except from examination of the urine; but, in some forms of renal disease albumen is often absent for long periods together. Moreover, the amount of the urine varies, and when great in quantity is usually of low specific gravity, and contains granular casts, which, however, are often few in number and not easily found. An absolute diagnosis is not always attainable. In gouty kidney we may fall back upon the rational symptoms, and the distinguishing characteristics may be found to run in the following directions: Rheumatism is associated with the fibro-serous texture; in lithiasis the poison has more affinity for the true serous surfaces, and is often the cause of pleurisy and peritonitis. Lithiasis more affects the muscles, and rheumatism rather the large joints. Diarrhœa, vomiting and other affections of the mucous membrane, as bronchitis, accompany lithiasis; and in these it differs from rheumatism. Lithiasis is accompanied by headache, especially of the vertex (persistent and recurring vertical headache is almost pathognomonic of lithiasis), or the pain may be frontal. (FOTHERGILL.)

6. A dislocation of the shoulder has been prescribed for as "rheumatism," which shows the necessity of inspection of affected joints.

A typical effect of the acid diathesis of chronic rheumatism is the *rheumatic markings of the teeth*, to which attention has been directed by Dr. L. G. NOEL.*

These markings seldom appear until after middle life is past. They are most frequent upon the crowns of the teeth, though they are sometimes seen upon their buccal and labial surfaces. It is that condition of the teeth treated of in dental works as "spontaneous abrasion."

The abrasion often begins as decay in the fissures on the grinding surface of the molars and bicuspid, but instead of following the tubuli, and dipping deep into the interior of the teeth, these become closed by a calcareous deposit, as fast as laid open, and the decay spreads out into a wide saucer-shape. This cupping out of the teeth is not, however, confined to the molars and bicuspid, but commencing upon the cusps of the canines, and cutting edges of the incisors, as mere mechanical abrasion, asperities disappear, the teeth become square and polished on

* Nashville *Journal of Medicine and Surgery*, Feb., 1875.

the ends, and presently the surfaces begin to assume a concave, instead of their original convex, appearance. This cupping out may go on until the pulp is so nearly reached as to become irritated to the point of inflammation and death; but usually its irritation is only sufficient to cause a deposition of secondary dentine on the interior of its chamber, a part of its substance forming a matrix in which lime-salts are deposited.

GOUT.

The signs of gout have already been in part referred to (page 55). It is not nearly so frequent in the United States as in England, and is apt, therefore, to be mistaken for rheumatism, which it closely resembles.

The following table of differences will facilitate the diagnosis:—

GOUT.	RHEUMATISM.
Generally a hereditary history.	Rarely hereditary.
Occurs usually in males, beyond middle age.	Occurs oftener in females, and before middle age.
Attacks generally periodic, and last about a week.	Attacks dependent on exposure, and last several weeks.
The small joints chiefly affected, especially that of the great toe, or lower extremity.	The large joints are those generally attacked.
Much local pain, redness, œdema, and enlargement of veins.	All these symptoms less marked.
Kidneys generally affected; little fever; no sweating; heart not implicated.	Kidneys not involved; fever often high; sweating profuse; heart often implicated.
Chalk stones in the joints and ears.	Chalk stones never present.
Uric acid always present in the blood in large excess (GARROD).	Uric acid never found in excess.

Dr. GARROD says that the presence of uric acid in the blood can readily be demonstrated by taking a fluidrachm of the serum from a blister, adding to it six minims of acetic acid, and placing a thread in the mixture. The uric acid, if present, will be deposited in fine crystals along the thread.

RHEUMATOID ARTHRITIS (RHEUMATIC GOUT, ARTHRITIS RHEUMATICA DEFORMANS).

This is by no means an infrequent disease in this country, and is a very serious one. It is now acknowledged by the best authorities to be a distinct malady, different in origin, history and treatment from both rheumatism and gout. It is common in women and young persons, and is not produced by alcoholic or other excesses. It implicates joints of all sizes, and in all the extremities. They become permanently affected, stiffened and enlarged, but no deposits of urate of soda are found in them. The disease frequently shows itself without fever; the joints swell by serous effusions into the capsules, and along with this the ends of the bones enlarge. The integument is not inflamed, or but moderately so, and the muscles do not appear to suffer. The result on the joint may be subluxation, relaxation, or ankylosis. The concretions attendant on the disease prove, on analysis, to be of the same composition as bone, with a slight preponderance of lime (DRACHMANN). Phosphoric acid is diminished in the urine and increased in the blood (BÖCHER).

Neither the treatment of gout nor that for acute rheumatism yields its usual results in this disease.

PERNICIOUS ANÆMIA AND LEUKÆMIA.

The positive diagnosis of these conditions can only be secured by a microscopic examination of the blood.

In pernicious anæmia, according to Dr. EICHHORST, the characteristic appearances are: A portion of the red corpuscles are seen to retain their normal size, but are marked by an extreme paleness, with a tendency to crenation and the formation of rouleaux, while others among them attract attention by their small size, which is reduced often to one-fourth the diameter of the well formed corpuscles. These small ones are more deeply colored, and if allowed to roll over under the thin cover-glass, their appearance in profile shows them to have lost to a greater or less extent their bi-concave outline.

For the examination of the blood in such investigations, Dr. GOWERS, of London, recommends the use of the *hæmacytometer*, by which he

measures for the purpose of ascertaining the number of red and white cells in a given volume of blood. The essential part of the apparatus consists of a glass slip, on which is a cell one-fifth of a *millimètre* (.008 inch) deep. The bottom of this cell is divided into one-tenth *millimètre* squares. Upon the top of the cell rests the glass cover, which is kept in its place by the pressure of two springs. In estimating the number of corpuscles, the patient's finger is pricked; then by means of a capillary pipette, five cubic *millimètres* of blood are taken up and well mixed up with 995 cubic *millimètres* of saline solution; a drop of the dilution is then placed in the glass cell, the cover is adjusted, and the slide is placed in the field of a microscope. In a few minutes all the corpuscles have sunk to the bottom of the cell, and are seen lying on the squares; the number of corpuscles in ten squares is then counted, and this, multiplied by 10,000, gives the number in a cubic *millimètre* of blood. The degree of dilution and size of the squares are so proportioned that, with normal blood, two squares contain about 100 corpuscles, and the number in two squares thus expresses the percentage proportion of corpuscles to that of health. The proportion of white corpuscles to red, or their absolute number, may be easily determined during the same observation.

A simpler method is used by Dr. J. G. RICHARDSON, of Philadelphia. He spreads a drop of fresh blood thinly on a glass slide, letting it dry, and then counting the number of white corpuscles. The specimens when thus prepared can be kept dry for any length of time, if preserved from dust and moisture, and by comparing specimens of different persons' blood, prepared similarly, the variations in the number of white corpuscles can be readily observed. By this means he claims to detect leucæmia in its early stages.

Profound anæmia is met with in the following conditions: (1) After great loss of blood or exhausting discharges; (2) where there is inanition (insufficient nourishment); (3) in chlorosis; (4) in cases of malignant disease; (5) in Bright's and Addison's disease, (6) leucocythemia, and (7) chronic metallic poisoning.

The symptoms of the idiopathic or "progressive pernicious" form of anæmia are described by Dr. BYRON BRAMWELL as follows: A profound anæmia, which is associated with marked changes in the microscopical

characters of the blood, and (in most cases) with the presence of retinal hemorrhages. The patient is generally well covered with fat, the skin is smooth and soft, the face looks slightly swollen, and is of a pale yellow or yellowish-green color. All the symptoms of profound anæmia are present, viz., extreme pallor of the mucous membrane, great debility, tendency to fainting, dyspnoea and palpitation on exertion, buzzing in the ears, headache, subcutaneous œdema, etc.; loud blowing murmurs are heard over the heart and great vessels; there is a venous hum in the neck; the pulse is very soft and compressible. Attacks of vomiting and diarrhoea are frequent; irregular elevations in temperature, transient paralyses, hemorrhages from the mucous membranes occasionally occur. The causes of the disease are at present unknown. The disease is said to occur more frequently in women than in men. In the majority of cases the termination is in death, the end being ushered in by profuse diarrhoea, coma, or delirium.

THE ORIGIN OF ZYMOTIC DISEASE, AND THE SO-CALLED GERM THEORY.

The following is an abstract of an instructive and elaborate paper by Dr. D. D. CUNNINGHAM, on the "Development of Certain Microscopic Organisms occurring in the Intestinal Canal,"* which is introduced here because it has a direct bearing upon the so-called germ theory of disease that is now exciting much discussion:—

"At a time when the association of special parasites with morbid states of their host is readily interpreted as evidence in favor of current theories regarding the parasitic origin of disease, any exact information regarding the true significance of the phenomenon in particular cases may serve a useful purpose."

Monads and amœbæ in excessive numbers are met with in the intestinal canal in cholera and certain other conditions characterized by special characters of intestinal contents. DAVAINÉ appears to have been the first to observe them, during the cholera epidemic of 1853–54. Similar bodies have been described by others in diarrhoea, typhoid fever, and dysentery. Drs. CUNNINGHAM and LEWIS recorded the occurrence of amœbal organ-

* Fifteenth Annual Report of the Sanitary Commissioner with the Government of India, 1878. Calcutta, 1880. Notice of, in *Medical Herald*, Louisville, July, 1880.

isms in choleraic and other excreta in 1870-71, and LÖSCH, in 1875, assumed that *amoeba coli* was the specific cause of a dysenteric condition of the large intestine.

The monads present in the digestive canal of man in India present characters entitling them equally to a place in two genera—cercomonas and trichomonas. These monads, or zoospores, exhibit no constancy of form, but are continually varying, in consequence of both intrinsic and extrinsic influences. The body in most cases is a mere fragment of naked protoplasm, with no differentiated covering, and with hardly, if any, indications of a differentiation of ectosarc and endosarc. Owing to this and to their minute size it is almost impossible to determine with any certainty many points regarding it when in a state of full activity. Nearly all reagents almost immediately produce destructive changes, leading on rapidly to disintegration and disappearance, and even slight changes in the medium, such as depression of temperature or dilution with water, are sufficient to arrest activity and induce disintegration.

The presence of zoospores is by no means confined to choleraic excreta. The two media best adapted to secure the demonstration of their presence are, first, the alkaline fluid of choleraic excreta; and second, a solution of cow-dung.

The zoospores are not peculiar to any diseased condition. Indeed, certain diseases associated with an acid reaction of intestinal secretions are incompatible with their presence. That they have been rarely detected in Europe is probably due to their ready destruction by very slight reduction of temperature. Other infusorial organisms are prejudicially affected by the initial fermentative changes occurring in the excreta.

Monads and amœbæ abound in the excreta of horses and cows. There are less bacterial elements in vaccine than human discharges.

As bearing, perhaps remotely, on the phenomena of periodicity of disease, there is a very interesting observation by Dr. CUNNINGHAM relating to the periodicity of sporangial development. In cultivation experiments, while at dawn there would be no trace of sporangia, an abundant crop of such bodies appeared within the course of a few hours. The development is regularly limited to the period between dawn and noon, or at least, 1 P.M. If sporangia have not appeared by the latter hour they will not appear until the following morning. At first sight it appeared

not improbable that light conditions were the determinant of this phenomenon, but experiments proved that this was not so, for the development followed the same course, even where all light was carefully and absolutely excluded.

Dr. CUNNINGHAM asserts that the characteristic parasitic zoospores and amœbæ of human and vaccine excreta are identical, and this affords a ready explanation of the extreme frequency of the parasite in the human subject, owing to the presence of a constant source of readily transferred reproductive elements. This transfer is principally effected through the air. The sporangia when thoroughly dried are detached, by the slightest contact, from their points of attachment, and having been so are so light as readily to be carried about by breezes. Desiccated, these sporangia probably resist fairly the action of the gastric juice; active or softened, they might fail, more easily than if dried, to reach unhurt the lower portions of the digestive canal.

Dr. CUNNINGHAM'S conclusions are as follows :—

“1. Special parasitic forms may be specially associated with particular forms of disease without holding any causal relation to them.

“2. The monadic, amœbal, and sporoid bodies, so abundant in many choleraic excreta, are all developmental forms of one species of parasite, which I propose to call *Protomyxomyces coprinarius*.

“3. This parasite appears to be closely related to the organisms included within the Protist groups of Protomonadinae and Myxomycetes, and in certain respects seems to represent a connecting link between them.

“4. It is not confined to choleraic or even to human excreta as a basis, and only attains its full development external to the bodies of the animals within which it occurs.

“5. Its immature forms occur parasitically, as normal inmates of the digestive canal in certain of the lower animals.

“6. In the human subject, both in health and disease, they are very frequently present in varying numbers.

“7. During health their number and activity are limited, due to repressive influences exerted by the normal intestinal contents as a medium.

“8. Their excessive abundance in certain forms of disease is due to abnormal conditions of the intestinal contents, permitting of processes of rapid multiplication.

“9. Normal human excreta do not form a medium in which any further development of the parasitic elements outside the host-body can occur.

“10. On the contrary, the normal series of fermentative changes through which the excreta pass after exit from the body, insures the complete destruction of the parasitic elements.

“11. No such destructive effect, however, is exerted by the changes occurring during the decomposition of the excreta in certain lower animals, especially cows and horses; and here the parasitic elements, on their escape from the body, undergo further processes of development, resulting in the production of reproductive bodies, securing the continuance and diffusion of the species.

“12. Such excretal matters, therefore, serve as a constant source whence parasitic elements may be transformed in the bodies of other animals.

“13. Human excreta which have passed through the initial processes of decomposition, and which have thus become alkaline, allow of the continued existence and multiplication of elements of the parasite which may then obtain access to them, and may thus serve as a second centre of reproduction.

“14. The introduction of the reproductive elements of the parasite into the human body is mainly effected through the medium of the air.

“15. The introduction of the reproductive elements *per se* seems to be quite innocuous.

“16. The special association of the parasite with intestinal disorders appears to be dependent on the abnormal condition of the intestinal contents, allowing of the rapid multiplication of reproductive elements which may obtain access to them.

PART II

LOCAL DISEASES.

CHAPTER I.

DISEASES OF THE NERVOUS SYSTEM.

CONTENTS. — *Cerebral Disorders—Congestion—Anæmia—Apoplexy—Thrombosis—Embolism—Meningitis—Tubercular Meningitis—Rheumatic Meningitis—Acute Cerebritis—The Ophthalmoscope in Nervous Disorders—Headache—Chronic Cerebral Disorders—Hypertrophy—Hydrocephalus—Brain Tumor—White Softening—Abscess—Chronic Meningitis—Thrombosis—Sclerosis—Localization of Brain Disease—Lesions of Cerebral Cortex—Brain Lesions other than Cortical—Tabular View of Paralysis with Seat of Lesion—Spinal Disease—Organic and Functional Paraplegia—Diagram of Spinal, Inflammatory and Degenerative Diseases—Tabular View of Spinal Paralysis, Congestion, Meningeal Apoplexy, Spinal Apoplexy, Acute Primary Myelitis—Comparison of Acute Spinal Disorders—Tumors—Tremors—Chronic Degenerative Diseases of the Cord—Prof. Charcot's Diagnostic Chart of Cerebro-Spinal Affections—Patellar-Tendon Reflex of Westphal—Analysis of Symptoms of Focal Lesions of the Cord (Gowers, Charcot, and Erb)—Comparative Semeiology of Cerebro-Spinal Sclerosis, Paralysis Agitans, and Locomotor Ataxia—Paraplegia from Reflex Irritation and Myelitis Compared—Gowers' Classification of Spinal Lesions—Pseudo-Hypertrophic Paralysis—Lead Palsy—Hysterical Paralysis—General Paralysis of the Insane—Spinal Irritation, and Spinal Weakness—Hysteria and Hystero-Epilepsy—Neuralgia—Insanity, Its Different Forms; their Pathology and Etiology.*

Recent advances in the physiology of the nervous system have thrown much light upon mental and nervous manifestations; and many con-

ditions which had been hitherto regarded as primary have been shown to be in reality symptomatic and secondary to definite morbid changes occurring either in the central nervous system, the trunks of the nerves, or in their peripheral terminations. It is evident that diseases having their origin or seat of lesion in the nervous system will vary in their symptoms with the locality of the morbid process and the function of the part affected. Disorders of intellection and insanity result from involvement of the cerebral hemispheres, with impairment of special senses, and paralysis of parts supplied by cranial nerves, and occurring with or without loss of power in the extremities. Diseases of the spinal cord give rise to paralysis of organs having direct connection with the seat of lesion, and also to disorders of sensation and nutrition. Hemiplegia may be of cerebral origin; paraplegia is generally spinal. Pressure or irritation of nerve trunks may cause local palsy, spasmodic affection, or neuralgia, while myopathic paralysis (such as encountered in lead palsy, pseudo-hypertrophic paralysis, and progressive muscular atrophy) may be due to a peripheral nervous affection. Hysteria, vertigo, neurasthenia and some mental disorders, being of uncertain seat and unknown relations, may be provisionally considered as functional disorders of the nervous system.

The principal symptoms referable to the brain may be considered as being caused by congestion, anæmia, apoplexy, thrombosis, embolism, brain tumor, cerebritis and abscess; by influence upon the brain disease of neighboring structures, such as meningeal inflammation, hemorrhage, effusion or neoplasm, necrosis, disease of the middle ear; and by poisoned conditions of the blood, as in uræmia, alcoholism, and the delirium of fevers. An irregular and abnormal distribution of the blood supply may give rise to night terrors, epilepsy, syncope, temporary (functional?) paralysis, cerebral exhaustion, aphasia, aphemia and agraphia; and irregular motor discharges from the cerebral centres are directly associated with chorea, tremor, and epileptiform convulsions; the higher mental powers being apparently merely held in abeyance in catalepsy, trance and hysterical coma.

Passing to the diagnosis of the principal cerebral disorders, the following points are of importance in distinguishing cerebral congestion and cerebral anæmia, which sometimes are difficult of clinical separation and diagnosis:—

CEREBRAL CONGESTION.

Severe, throbbing and diffused.

May be absent.

Full, throbbing, tortuous and distinct.

Full, tense ; often signs of plethora.

May be rumbling or singing.

Hallucinations ; may have active delirium.

Surface temperature of scalp may be increased.

Contracted.

Not increased ; may contain urates and phosphates (HAMMOND).

Headache.

Vertigo.

Temporal Vessels.

General Circulation.

Tinnitus aurium.

Mental Phenomena.

Temperature.

Pupils.

Urine.

CEREBRAL ANÆMIA.

Less sharp, generally vertical.

Usually a marked symptom.

Not prominent.

Pulse irritable, often anæmic murmur of pulmonary artery.

Noises may be short and high-pitched.

Below normal ; incapacity for mental application.

Surface temperature, if at all affected, is diminished.

Dilated.

Limpid, and may be passed in excess ; decrease of salts.

Cerebral exhaustion is sometimes so marked as to produce coma, and thus form a variety of apoplexy, and its diagnosis is made by excluding hyperæmia, hemorrhage, embolism and thrombosis of basilar artery. Where the latter condition terminates in recovery, it is almost identical in its manifestations ; indeed, "it would be difficult to disprove the assertion that cases of cerebral exhaustion belong in this category" (FLINT).

A sudden attack of coma in a case of albuminuria may be set down as due to *uræmia*, if embolism and apoplexy are excluded (by noting the absence of hemiplegia). Should the coma be associated with epileptiform convulsions this diagnosis is likely to be correct, even if no albumen can be detected in the urine ; since the form of renal disease most likely to give rise to uræmic poisoning is the cirrhotic form (contracted kidney), in which the albumen may be absent from the urine for considerable periods of time.

The early diagnosis of diseases attended by coma is of great importance, with the view of promptly instituting proper treatment.

CEREBRAL APOPLEXY.

Apoplexy is to be distinguished from drunkenness, narcotic poisoning, uræmic poisoning, epilepsy, concussion of the brain, cerebral thrombosis, embolism, and insolation or sunstroke.

Drunkenness. The odor of liquor may excite suspicion. If the patient vomit, the ejecta may be tested for alcohol. Or the urine may be examined by Anstie's test, as follows:—

R. Bichromate of potash,	1 part	
Strong sulphuric acid,	300 parts.	Mix.

To fifteen minims of this add a few drops of the urine, and if the patient has taken a toxic dose of alcohol, the mixture will turn an emerald green. In drunkenness the pulse is generally rapid, the pupils not dilated, the eye injected. The patient can be roused and hiccoughs.

Dr. MACEWEN, of Glasgow, gives the following method of distinguishing alcoholic coma from that of apoplexy, fracture of the skull, and other causes. In alcoholic coma, as long as the patient is undisturbed the pupil is contracted; but if any stimulus not sufficient to arouse the patient be applied to him, such as a shake or a pull of the beard, *the pupil dilates*, only, however, to become contracted again as soon as the person is left at rest.*

Narcotic poisoning. In this condition the outset is gradual; there are often convulsions, but the patient may be roused. In opium poisoning the pupil is contracted; so it is in hemorrhage in the pons. The vomiting, the acrid odor of opium, and the gradual intensification of the coma are diagnostic. There is no hemiplegia.

Uræmic poisoning. Here the coma nearly always comes on gradually and is preceded by convulsions. It is not deep, and at first the patient may be aroused. The stertor of the breathing is more superficial, while there is also frothing at the mouth.

Nearly always, distinctive modifications of the heart sounds will be heard, as reduplication of one or both, intensity of second sound, etc.; while there are elevation of the arterial tension and increased cardiac impulse. Of these cardiac physical signs none seem so constant or remarkable as muffling of the first sound. (Mr. W. WHITTLE.)

There are, moreover, in many cases marked prodromata. The skin has been waxy and œdematous, the eyelids puffed and the legs and feet swollen. The urine may or may not be albuminous (but this may also be present in apoplexy).

Epileptic coma presents a history of convulsions; lasts but for an hour

* *British Medical Journal*, November 16, 1878.

or two; there is frothing at the mouth; and the temperature is elevated.

In *hysteria* and *cataplexy* there is no elevation of temperature and no frothing at the mouth.

In *concussion* or *compression* from injuries to the head the skin is pale, the pupil dilated, and vomiting occurs. The symptoms are usually of short duration and there is a history of injury. Meningeal hemorrhage from injury presents no points of difference from true apoplexy, except that hemiplegia is generally wanting (FLINT).

Syncope is readily distinguished by the feeble pulse, the pale face, the quiet respiration and the brief duration of the unconsciousness; while in *asphyxia* the livid face, distressed breathing and blue lip which precede the coma indicate its distinction.

In regard to *thrombosis* and *embolism* of the larger cerebral vessels the diagnosis is often extremely difficult. The following table of the comparative symptoms is drawn up from the works of BUDUY, GELPKKE, FLINT and HAMILTON:—

CEREBRAL HEMORRHAGE.	CEREBRAL THROMBOSIS.	CEREBRAL EMBOLISM.
Occurs in advanced age, with atheromatous arteries.	In advanced age. May occur in children during scarlet fever and renal disease.	Almost always in early or middle life (FLINT).
Onset generally sudden.	Development of symptoms gradual.	Prodromata absent.
Hypertrophy of left ventricle. Alcoholism or other debilitating habits.	No rheumatic history. Endarteritis deformans of peripheral arteries sometimes present.	Previous articular rheumatism or other disease leading to formation of clots. Often cardiac valvular insufficiency. Coincident embolisms are sometimes present.
Pain in the head.	No headache.	No headache.
Aphasia ataxic, secondary to a loss of consciousness. Intelligence much involved.	Aphasia incomplete and primary, occasionally absent. Intelligence less involved.	Aphasia amnesic. Retention of mental power.
Often coma.	Rarely loss of consciousness.	No coma.
Paralysis very marked; occurs on either side.	Paralysis less marked.	Muscular paralysis extensive; nearly always on the right side (FLINT).

CEREBRAL HEMORRHAGE.	CEREBRAL THROMBOSIS.	CEREBRAL EMBOLISM.
Apoplectic phenomena from the outset. Symptoms of cerebral pressure.	No apoplectic phenomena at onset.	Early apoplectic phenomena, but without loss of consciousness.
Disappearance of the residual disorder after a moderate time. May terminate in chronic abscess.	Recovery slow; more or less hemiplegia may remain.	Very rapid, or else quite imperceptible disappearance of the residual disorder. May be followed by softening.
After a few days pain in the head and increased temperature of the body on the <i>unaffected</i> side (FLINT).	May have œdema more marked on affected side.	One-sided œdema, often in the arm alone.

The high temperature (108° to 113° F.) of cases of sunstroke serves to distinguish such from the coma of apoplexy; although in some cases of insolation the coma is probably due to cerebral exhaustion, the distinguishing features of which have been previously considered. The subjects attacked are generally laboring men, who have been exposed, while at their work, to a continuous high temperature.

ACUTE CEREBRAL INFLAMMATIONS.

Considering the acute inflammatory state of the brain and its coverings, we tabulate their comparative semeiology as follows:—

SIMPLE MENINGITIS. (<i>Lepto-meningitis.</i>)	TUBERCULAR MENINGITIS.	RHEUMATIC MENINGITIS.	ACUTE CEREBRITIS AND CEREBRAL ABSCESS.
Due to disease of the cranial bones, traumatism, exposure to sun. (Very frequently the meningitis of young adults has a syphilitic source.) May be epidemic.	Scrofulous inheritance.	Rheumatic diathesis.	May be due to general causes, such as pyæmia, etc., or to local causes, as traumatism, bone disease, local irritation, extension from meninges, etc.
A disease of both infants and adults, though usually in the latter.	Often children under five years of age.	Adults.	Often in elderly subjects.
Previously healthy; no prodromata.	History of persistent headache and obstinate constipation; wasting.	Often during an attack of joint inflammation.	Rarely occurs in previously healthy persons.
No chest symptoms.	Previous pulmonary trouble.	None.	None.

SIMPLE MENINGITIS. (<i>Lepto-meningitis</i> .)	TUBERCULAR MENINGITIS.	RHEUMATIC MENINGITIS.	ACUTE CEREBRITIS AND CEREBRAL ABSCESS.
Onset sudden.	Takes four or five days to develop; approach insidious.	Rapidly developed.	Slow, and may simulate typhoid.
Headache intense on both sides of head.	Persistent and marked headache, which exacerbates.	Intense pain.	Dull, persistent and localized; less than in meningitis.
Pupils contracted.	Pupils irregularly dilated.	Leads to active delirium.	Mental confusion and impairment of intelligence.
Intelligence clear at first, but may become furiously delirious.	Delirium of low grade at night (stupor in second stage); strabismus, and oscillation of eyeballs.	Not marked.	Vomiting not infrequent.
Vomiting early, frequent.	Vomiting occasionally.	Pulse full and rapid.	Less fever.
Pulse full and rapid.	Irregular and slow pulse.	Temperature may be very high.	No convulsions; but sudden hemiplegia may occur.
High fever.	Fever not intense.	No convulsions.	Course is often chronic.
Convulsions early, contracted pupils, with contractions of flexor muscles of arm or leg.	Convulsions late, with dilated pupils and hemiplegia.	Lasts a few days; death often occurs from continued high temperature.	Prognosis not encouraging.
In fatal cases death generally occurs in a week; recovery is slow.	Lasts from one to three weeks.	Prognosis fair.	
Prognosis favorable under prompt treatment.	Prognosis unfavorable.		

Dr. GEE notes that meningitis of the base of the brain is generally tubercular; and when tubercular meningitis attacks the convexity, there is a constant convulsive condition, moderate force and very variable pulse. (See page 48 for a more detailed account of tubercular meningitis.)

These cerebral diseases may be distinguished from typhoid fever by the history and course of the affection. Typhoid occurs in the spring and fall, and is often endemic; it never appears in children under five, and generally attacks young adults. It is a continued fever, coming on in a hitherto healthy person with malaise, epistaxis and diarrhoea. Chills and vomiting are rare. Convulsions and paralysis are late manifestations,

and due to complications. Delirium of low type, headache dull, moderate deafness, pulse rapid but regular. Abdominal symptoms generally prominent, tympanites, diarrhœa, tenderness and gurgling on pressure in the right iliac fossa, and a discrete rose-colored eruption upon the chest and belly. Convalescence at the beginning of the third week; disease generally continues about four weeks.

THE OPHTHALMOSCOPE IN NERVOUS DISORDERS.

In the diagnosis of intracranial disorders the ophthalmoscope is often of great service, though, perhaps, scarcely to the extent advocated by BOUCHUT. The *discrete tubercles of the choroid* accompanying meningeal deposit, the *choked disc* in cerebral tumors and inflammations, and the *retinitis* and *retinal hemorrhages* of Bright's disease, are of great importance. BOUCHUT declares* that the ophthalmoscope is as indispensable to the physician as to the oculist, and he was among the first to point out the great importance of this aid to practical medicine. We quote his opinions and conclusions:—

“All diseases of the brain and spinal cord, and all the nervous affections termed neuroses, because they are regarded rather as functional than organic, ought to be investigated by its aid. When by its assistance the physician discovers a lesion of the optic nerve, of the retina, or of the choroid, in a case presenting convulsive, choreic, paralytic, or spasmodic nervous phenomena, he may be certain that a cerebro-spinal lesion is the starting-point of these symptoms. Every symptom regarded as nervous, which is accompanied by a lesion of the fundus of the eye, is caused by an organic alteration of the brain, the cord, or the membranes. Thus is it with chorea, considered by many physicians as a simple neurosis; and yet this should, in consequence of the congestive optic neuritis found in its subjects, be regarded as a congestive affection of the anterior spinal columns. So also epilepsy, in a certain number of cases, is the result of cerebro-spinal lesions which at the same time induce changes in the optic nerve or retina. Hysterical paraplegia and paralysis produce no neuro-retinian changes, while symptomatic paraplegia and spinal ataxia produce either simple hyperæmia of the optic nerve or hyperæmia and atrophy. So leucæmic, tubercular, glycosuric, or albuminuric diatheses

* “Revue Cérébroscopique,” in *Gazette des Hôpitaux*, for January, 1874.

are often revealed by optic neuritis, the ophthalmoscopic diagnosis in some of these cases being most striking. It is especially in patients attacked by general acute tuberculosis, accompanied by typhoid symptoms, and which are mistaken for typhoid fever, that cerebroscopy becomes truly remarkable. In an infant in whom the disease had all the appearance of typhus, the ophthalmoscope, by revealing tubercles of the choroid with neuro-retinitis, determined that there were tubercles in the brain, and consequently productions of the same character all over the body—which the autopsy demonstrated to be the fact.

“Can any diagnosis be more exact than this? You see, in the living man, tubercles of an organ which permit you to conclude that they will also be found elsewhere. You see a nerve either healthy or diseased, and this indicates whether its roots are sound or diseased; and you have almost laid bare arteries and nerves which are so afferent to the brain that changes in them, studied with care, represent similar changes in a portion of the nervous centres. It seems almost marvelous; and I do not think that since auscultation there has been anything discovered so useful to semeiology. Henceforth, the physician may divine and often affirm lesions of the brain, cord, or meninges, the diagnosis of which before was impossible or only probable. Thus: 1. From hyperæmia and hyperæmic tumefaction of the optic nerve there results the diagnosis of mechanical or inflammatory hyperæmia of the brain in meningitis, in cerebral hemorrhage, effusions into the brain, and in some cases the diagnosis of ataxic or other spinal diseases. 2. By papillary œdema joined to hyperæmia I recognize œdema of the meninges; or an obstructed cerebral circulation through meningitis, cerebral tumors, ventricular hydrocephalus, cerebral hemorrhage, meningeal effusions, thrombosis of the sinus, etc. 3. By neuro-retinian and choroidean anæmia, I recognize cerebral hemorrhage of *ramollissement*, and if the anæmia be absolute it is fatal. Empty arteries and veins of the eye, and an exsanguineous condition of the choroidean network, indicate arrest of cerebral and cardiac circulation. 4. By exudative and fatty optic neuro-retinitis, I recognize chronic meningo-cephalitis; the encephalitis of cerebral tumors, and the changes in the nervous substance which accompany these tumors. 5. By retinian varices and thromboses, I distinguish meningeal thromboses, or those of the sinuses. 6. By the aneurisms of the retinian arteries we may recog-

nize the miliary aneurisms of the brain. 7. By simple retinian hemorrhages we recognize a compression of the brain by hemorrhagic or other effusions; but if these retinian hemorrhages are accompanied by retinian steatosis, there is also cerebral steatosis, and this is the case in chronic albuminuria, leucocythæmia, and glycosuria. 8. By atrophy of the optic nerve, tumors of the brain and cerebral or spinal sclerosis are discovered. 9. Finally, we never meet with tubercular granulations in the choroid without the existence of similar ones in the lungs or other organs.*

The ophthalmoscope is now frequently employed for diagnostic purposes in ordinary medical practice where there is imperfection of vision, to determine whether it is due to other than nervous lesions, to discriminate between affections of different portions of the eye, and, sometimes, to measure the amount of refraction in cases of hypermetropia and myopia. Even where there is no impairment of sight, there still may occur very decided and characteristic retinal changes and alterations in the optic disc, which are readily detected by ophthalmoscopic examination, as already indicated; so that in obscure cases the routine examination of the eyes has become nearly as imperative as the chemical and microscopical examination of the urine.

HEADACHE.

Some of the most trying cases to treat are those of headache, because this symptom may appear in many and even diverse morbid states, and often indicates serious cerebral disorder. Mr. WM. HENRY DAY, of London,† has made a study of these conditions, and thus summarizes his conclusions:—

Headache usually denotes some functional disturbance of the brain or its membranes, induced (1) by excess of local blood pressure, (2) by absorption into the blood of poisonous matters, (3) or by such a diminution of healthy blood as provokes irritation and suffering. It may be a symptom of organic disease, either of the brain or its membranes, or of the kidneys or stomach, and uterus.

Cerebral anæmia.—A striking symptom is pain at the *top* of the head, which often feels hot and burning, sometimes gnawing and scraping.

* *Medical Times and Gazette*, January 23, 1875.

† *British Medical Journal*, Nov. 16th, 1878.

Irritability of temper. Face livid and cold. Patient easily exhausted. Eyes dull.

Hyperæmia, Active or Passive—Active.—Arterial fullness. Head hot, pain frontal, throbbing and bursting, pulse tense, full. Conjunctivæ reddened. Eyes bright. Photophobia. Mentality dull. Apoplexy may ensue.

Passive.—Venous fullness from obstruction caused by heart disease, bronchocele, etc., pleuritic effusion, defective ventricular action.

Sympathetic headache.—Faulty digestion or ovarian excitement. Stomach sometimes weak and over-sensitive. Catamenial headache. Dyspeptic and bilious headache. *Irritation of the sympathetic reduces the amount of blood in the brain.*

Nervous headache.—Disturbance of brain from overwork, worry and anxiety. Aggravated by some of the circumstances favoring sympathetic headache. In women there is a passage of a large quantity of limpid urine; feet and hands cold. Confusion of ideas. Nausea and sickness, not attributable to errors in diet, may precede the attack. In nervous people constipation may cause headache.

Poor seamstress headache.—Spanæmia. Headache of excessive menstruation, or menorrhagia. Hereditary influence strong.

Neuralgic headache.—From decayed teeth, peripheral irritation, malarial poison. Pain and tenderness along the fifth nerve. Pain intense; not relieved by vomiting.

Toxæmic headache.—Poisoned blood acting on nerve centres, from particular articles of food or drink, and drugs. Or certain specific diseases—gout, rheumatism and syphilis. Headache of vitiated atmosphere.

Organic headache.—Morbid growths; meningitis. When slowly progressing pain is limited to smaller area, and is intense. Periosteal inflammation is accompanied by tenderness upon pressure.

Headache in children, due to accidental injuries, to derangement of alimentary canal, anæmia, exhausting influences, such as bad food and impure air, immoderate intellectual efforts, and sometimes to organic diseases (often tuberculosis).

In strumous and weakly children headache must be carefully watched. A headache of long standing in a child is significant, and requires more serious attention than in the adult.

CHRONIC CEREBRAL DISORDERS.

In children the diagnosis may be required to be made between hypertrophy of the brain and hydrocephalus, which have enlargement of the head as a common sign.

HYPERTROPHY.	HYDROCEPHALUS.
Increase in size most marked above the superciliary ridges.	Increase in size most marked at the temples.
Head square in shape.	Head more rounded.
No yielding of fontanelle on pressure.	Fontanelle elastic.
Eyes at normal distance.	Distance between the eyes increased.
Excessive amount of brain, especially white matter.	Excessive amount of fluid in ventricles, or sub-arachnoid space.
Patient dull, liable to epileptic fits, and suffers from headache.	Mentality feeble; generally can be traced to congenital source; death may occur from convulsions. No marked headache.

The diagnosis of hydrocephalus may be confirmed by tapping the fontanelle with the aspirator, or a hypodermic syringe. In adults brain tumor and sclerosis are among the prominent disorders of slow progress, the symptoms varying in a very marked manner with the location of the lesion. Chronic inflammation of the brain may terminate in insanity or in abscess. In its course it has been mistaken for dyspepsia, but a proper inquiry into the mental condition of the patient will reveal the cerebral mischief, which continues to progress even after any coexisting indigestion has been corrected. There is, moreover, sluggish intelligence, and partial paralysis or rigidity of certain muscles of the extremities. Attacks of delirium or mania finally confirm the diagnosis, and the patient dies in a state of coma.

Intra-cranial disease of a chronic character is often so obscure as to leave even the most experienced in doubt, and the post-mortem examination sometimes produces revelations that disconcert the medical attendant. Due regard to some of the characteristic phenomena in the accompanying table will often serve to clear up the doubts surrounding a difficult case.

BRAIN TUMOR.	SOFTENING (WHITE).	ABSCESS.	CHRONIC MENINGITIS.	THROMBOSIS OF SINUSES OF BRAIN.
Of slow development.	Approach and progress slow. Follows embolism or apoplexy. Non-inflammatory.	Follows injury to the skull or chronic disease of the head.	Caused by syphilis, rheumatism, disease of bones, blows upon the head, etc.	Sudden development of symptoms.
Intellect not disordered at first.	Early affection of intelligence. Marked impairment of memory.	Varies with seat.	Intelligence not affected, except during attacks of delirium.	May be unconsciousness or not. Intelligence subsequently good.
Headache violent, paroxysmal and often localized.	Dull and constant.	Sudden in its development, and general.	Subject to exacerbations, but generally chronic.	No headache; oedema of forehead and eyelids.
Paralysis slow in appearing, and often limited to the muscles of eye or of the face; more rarely hemiplegia.	Motor and sensor phenomena more frequent and prominent. Partial palsies and disturbances of sensibility subsequently.	Course is much more rapid: convulsions, drowsiness, paralysis and coma quickly developed.	In consequence of meningeal exudation may present the clinical signs of a brain tumor.	May be coma; varies greatly, according to part of brain whose vascular supply is disturbed.
Convulsions a common symptom, epileptiform in character. Not followed by palsy or hebetude.	Begins often with apoplectiform attacks, which seldom occur afterward.	Convulsions early; paralysis belongs to developed stage.		Very rare.
Vertigo and tinnitus aurium.	Vertigo.		More vertigo.	Varies.
Vomiting.	Not unfrequently.	Rare.	Frequent vomiting.	No vomiting.

SCLEROSIS.

Sclerosis is a disease of the nerve centres, in which there is increase of connective tissue elements, without primary involvement of the nerve cells. It may exist as diffused cerebral sclerosis, spinal sclerosis (several forms), cerebro-spinal sclerosis (sclérose en plaque), and glosso-labio-laryngeal paralysis. Cerebral sclerosis occurring in children can be distinguished from deficient development by the following characteristics:—

DEFECTIVE DEVELOPMENT OF INTELLIGENCE. DIFFUSED CEREBRAL SCLEROSIS.

Intelligence stationary, instead of progressing with age.

Intelligence retrogressive and more affected. Often terminates in idiocy.

Not connected with disease.

May follow injury to head, zymotic fevers, severe application of body or mind.

Speech restricted to few words, imperfectly pronounced.

Never learns to talk, or speech becomes imperfect or lost after it has been acquired.

DEFECTIVE DEVELOPMENT OF INTELLIGENCE. DIFFUSED CEREBRAL SCLEROSIS.

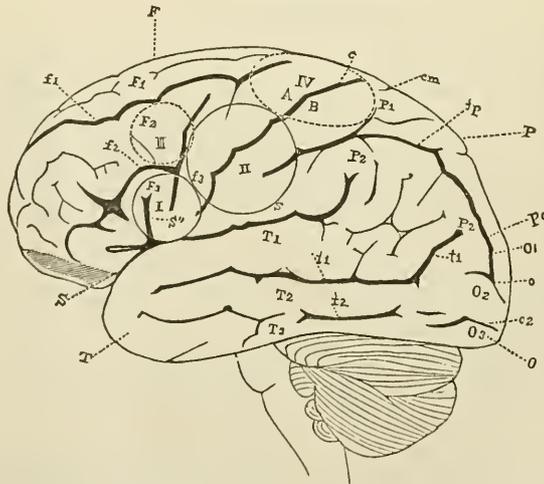
<p>No paralyses.</p> <p>Muscular system in good condition.</p> <p>No convulsions.</p> <p>Improved by training and education.</p>	<p>Usually more or less hemiplegia.</p> <p>Arrest of growth of certain parts of body, with contraction and distortion of affected limbs.</p> <p>Frequent convulsions.</p> <p>Progress very chronic, and may live to advanced age.</p>
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LOCALIZATION OF BRAIN DISEASE.

The localization of diseases of the brain is a subject of great interest. In order that a correct diagnosis should be made the important anatomical and physiological data must ever be borne in mind. We proceed first to the consideration of

LESIONS OF THE CEREBRAL CORTEX.

[The accompanying excellent diagram, or physiological map of the principal cerebral cortical centres, modified from FERRIER and ECKER,



CORTICAL CENTRES OF THE HUMAN BRAIN.

S, Fissure of Sylvius; c, Fissure of Rolando; po, Parieto-occipital fissure. A, Ascending frontal gyrus; B, Ascending parietal gyrus; F₃, Third frontal gyrus; P₂, Gyrus angularis. Circle I, Seat of lesions which (on the left side) cause aphasia. Circle II, Seat of lesions which convulse or paralyze the upper extremity of the opposite side. Dotted Circle III, Seat of lesions which probably convulse or paralyze the face on the opposite side. Dotted Oval IV, Seat of lesions which probably convulse or paralyze the lower extremity of the opposite side. These districts receive their blood supply chiefly from the middle cerebral artery. The remaining letters refer to anatomical points which explain themselves.

by SEGUIN, will be found very useful, as it embodies the results of the recent researches of FRITSCH and HITSIG, FERRIER, DALTON and SEGUIN;* particularly as this subject is now attracting much attention.]

The following is the summary given by SEGUIN (*loc. cit.*):—

PHYSIOLOGICAL.

“ In the first place, it appears almost absolutely certain that in man a lesion involving the posterior part of the third frontal convolution (on the left side usually) causes aphasia; *i.e.* impairment or loss of articulate speech, or even of language in general. It would seem, besides, that (1) lesions of the same part on either side of the brain produce paresis of many muscles concerned in lingual and pharyngeal movements; (2) that lesions of the anterior folds of the island of Reil (convolutions which are continuous with the third frontal), may also produce aphasia; and that (3) loss of speech may result from injury to the white substance lying between the third frontal gyrus and the basis cerebri. I believe in a not too limited localization of the motor functions exerted in language, and would graphically represent this by the circle marked I.

“ In the second place, lesions limited to the inferior portions of the ascending frontal and parietal gyri have produced spasmodic and paralytic phenomena limited to the upper extremity of the opposite side. I am disposed to admit as highly probable that these parts are connected in the healthy living man

PATHOLOGICAL.

“ 1. The symptoms of an *irritative lesion* of these parts consist in convulsions, with or without subsequent transient paralysis; *e. g.*, such a lesion in circle III would give rise to spasmodic movements in the superficial muscles of the face on the opposite side, with slight paralysis. Irritative lesions of the regions inclosed in circles II and IV will cause convulsions limited to, or first appearing in the hand and arm, or foot and leg, of the opposite sides. As regards circle I (Broca's speech centre), we know little of the effects of its pathological irritation. In one case which I have placed on record, a thickening of the meninges involving the third frontal convolution of the left side produced intermittent and incomplete aphasia.

“ It was by the close study of the clinical and pathological aspects of cases of localized epilepsy (fingers and hands), that Dr. J. HUGHLINGS JACKSON was enabled to form his theory of motorial discharges from irritation of the cortex cerebri, and thus pave the way for FERRIER'S admirable researches. Dr. JACKSON must, I think, be considered, after Prof. BROCA, as the founder of our present growing doctrine of cortical localizations.

* See Lectures in *New York Medical Record*, delivered at the College of Physicians and Surgeons, New York, in January, 1878.

PHYSIOLOGICAL.

with the various voluntary movements of the arm and hand. This zone is represented by circle II.

"I am not prepared to go further in admitting pathologically proved cortical centres, but would add that there are some reasons for believing that future autopsies will locate one centre for the external facial muscles just forward of the two centres named above, viz., the region included in the dotted circle III; and another for movements of the legs upon the upper parts of the ascending frontal and parietal, as roughly indicated by dotted oval IV."

PATHOLOGICAL.

"2. *Destructive lesions* of portions of the excitable district produce paralysis in peripheral parts across the median line. The symptoms will, to a certain extent, correspond with the *precise* location of the lesions, very much as in irritative lesions; e.g., embolism of the first branch of the middle cerebral artery on the left side will cause softening of the posterior part of the third frontal gyrus, with the symptom aphasia. A destructive lesion of the principal part of the motor zone on the right side will produce left hemiplegia without aphasia; but if this lesion occupy the left hemisphere, loss of speech will co-exist with the paralysis."

It must be added that secondary descending degeneration ensues after destructive lesions of the motor regions of the cortex, and that we have late contracture or rigidity of the paralyzed limbs as part of the symptom group.

Negative characters of these cortical lesions are preservation of sensibility in the paralyzed parts, and (except with epileptic attacks) preservation of consciousness, and incompleteness of paralysis.

In diffused lesions of the cortex the chief symptoms are delirium, convulsions and pain; evidences of intense irritation. The coma and paralysis which follow may in some degree be caused by impaired nutrition of the cortex, but more probably by circulatory and tension changes in the whole encephalic mass.

As regards sensory cortical centres, Dr. SEGUIN believes that we have as yet no pathological data for their study.

DISEASE OF BRAIN CENTRES OTHER THAN CORTICAL.

The following tabular view of the paralyzes, with the localization of the lesion, is mainly that of Professor DACOSTA.*

* "Medical Diagnosis," 4th Edition, Philadelphia, 1876.

SYMPTOMS.

Hemiplegia, without disturbance of sensation. Incomplete paralysis of face. Electro-muscular contractility normal or increased. Generally accompanied by apoplectic symptoms.

Crossed paralysis (*i.e.* face of right and hemiplegia of left, or *vice versa*). Paralysis of face marked, both of motion and sensation. General symptoms giddiness, nausea.

Same as above, except complete facial paralysis (both sides of face).

Paralysis of arm and leg, slight paralysis of face, dilatation of pupil of opposite side, with external squint (3d nerve paralysis).

General paralysis, more or less complete; sensation diminished upon one side, increased upon the other. Local temperature variations.

SEAT OF LESION.

In corpus striatum, near the internal capsule; on side opposite to hemiplegia.

Pons Varolii upon opposite side to palsy of limbs (below decussation of facial nerve).

Pons at level of decussation of facial nerve.

Crus cerebri on side corresponding with affected eye.

Medulla oblongata on side of increased sensibility and temperature, at the level of decussation of anterior pyramids.

As regards the side of the brain affected, certain peculiarities have been noted by able observers.

LESIONS OF THE RIGHT HEMISPHERE.

Anæsthesia more complete.

Paralysis more complete.

Paralysis of sphincters.

Alterations of nutrition (œdema, eschars, fevers, pulmonary congestion).

Disorders of special senses.

Hysterical symptoms.

LESIONS OF THE LEFT HEMISPHERE.

Loss of speech (aphasia).

Paralysis of muscles of articulation.

Less marked.

Less marked.

Less marked.

Hysteria seldom.

The observations of BROWN SÉQUARD have demonstrated that in exceptional cases the symptoms do not correspond as accurately with the

anatomical position of the lesion as is above indicated. At the present time these cases must be looked upon as really exceptional, and as not affecting the rules which have just been cited. More particularly are these aberrant symptoms likely to appear in tubercular disease of the brain. Indeed, Prof. HENOCI (in *Charitè Annalen*, fourth year), reports nine cases of tuberculosis of the brain that show how risky it is to localize, basing this upon recent physiological investigations. The results are as follows:—

SYMPTOMS.

LESION.

CASE I.—Left hemiplegia.	Multiple tubercles of the cortical layer of both hemispheres, the frontal lobes and tubercle of the left half of the cerebellum.
CASE II.—Tremor and paresis of the right side, finally, contraction of all extremities.	Tubercle of the left frontal lobe, the left corpus striatum, both thalami and right half of the cerebellum.
CASE III.—Hemiplegia and contracture of the left side, as well as of the facial nerve.	Tuberculosis of the right frontal lobe.
CASE IV.—Contracture and involuntary motion on right half of face and body.	Tuberculosis of the left frontal lobe.
CASE V.—Complete absence of symptoms until meningitis set in.	Tuberculosis of the commissure of the cerebellum and of both hemispheres.
CASE VI.—Paralysis of the left abducens, the left iris and right arm.	Tuberculosis of the commissure of the cerebellum.
CASE VII.—Absence until meningitis set in.	Tubercle in the pons.
CASE VIII.—Complete absence.	Tubercle of the left posterior lobe.
CASE IX.—Paralysis of the right abducens.	Tuberculosis of both posterior lobes, the posterior corpora quadrigemina, the pons and left crus cerebelli.

Of all these cases only II and III show the possibility that lesions of the motoric centre of the frontal convolutions produce motoric lesions

of the opposite side. This chance of diagnosis, however, is very limited, as is shown by the other cases where these locations were free from disease, and yet the same symptoms produced with lesions in other parts of the brain, even cerebellum (Case VI). Sometimes the intensity of the symptoms does not seem to correspond with the intensity of the lesion (V and VI). HENOCI believes that a close study of the fibres leading from and to these physiological centres will do much to reconcile the apparent contradictions between pathological and symptomatological differences.*

The subject of insanity will be separately considered at the end of this section.

SPINAL DISEASES.

A leading symptom of many diseases of the spinal cord, whether functional or organic, is *paraplegia*. It is so rarely of cerebral origin that ordinarily the brain may be omitted from the discussion, unless there is the co-existence of distinct evidences of brain disease, as headache, impaired cerebration, and paralysis of parts supplied by nerves arising above the spinal cord.

The following classification of diseases giving rise to paraplegia, with their characters, has been proposed by Prof. H. C. WOOD.†

ORGANIC.	FUNCTIONAL.	HYSTERICAL.
Disease of the cord.	Anæmic. Reflex (from peripheral irritation, renal, preputial, etc.). Dyscrasic (diphtheritic, etc.).	Hysteria.

The last mentioned, hysterical, is also functional, but simulates the organic more closely than does the second group. (For further consideration of Hysterical Paralysis, see Hysteria.) It must be admitted, however, that so-called functional disorder cannot long exist without being followed by change in structure.

The general distinctions between the organic and functional paraplegias may be presented as follows:—

* Cincinnati *Lancet and Clinic*, May 31, 1878.

† "On the Diagnosis of Diseases accompanied by Paraplegia." 1875.

ORGANIC.

FUNCTIONAL.

Onset may be almost instantaneous or very rapid, though sometimes gradual.

Usually at some period spasm or pain in the affected limbs.

Often a sensation of a band or stricture around the waist, *girdle-pain* (pathognomonic).

Anæsthesia frequent and often complete.

Retardation of sensation (a perceptible time elapses between the patient's seeing his feet touched and feeling that they are) (pathognomonic).

Symptoms of paralysis of the bladder.

The onset always more or less gradual, except the hysterical form, where the paralysis is generally abrupt.

Spasms or pain rarely or never present.

Not found.

Anæsthesia absent or but partial.

Sensation, if present at all, is not retarded.

No symptoms whatever of vesical paralysis, except in the hysterical form.

Where the bony canal is involved and caries is present, this condition may generally be discovered by ROSENTHAL'S test. This consists in passing down the back a pair of electrodes attached to a faradaic battery of some power, one pole being placed upon each side of the spine. Under these circumstances if there be any caries or inflammation of the vertebræ, the moment its locality is reached the patient starts or screams, from the burning, sticking pain caused by the passage of the galvanic current through the inflamed tissue. Dr. WOOD states he has not found this test as trustworthy as its originator claimed it to be, and as, apparently, it ought to be. In cases simulating caries, however, the pain is probably not so severe as where the vertebræ are really affected. Moreover, absence of the pain in any case seems to be conclusive evidence of the non-existence of bone disease.

The following study of the principal organic spinal diseases, from the writings of SEGUIN, CHARCOT and other authorities, when taken in conjunction with the tabular view of paralysis, will often enable the diagnostician to determine both the nature and location of a spinal lesion :—

TABULAR VIEW OF SPINAL PARALYSIS.

SYMPTOMS.	SEAT OF LESION.
cles, and consequent interference with inspiration. Paralysis of muscles of upper extremities, except those of the shoulders, which receive their nerves from the higher portions of the cervical region.	In the cord below the middle cervical region.
In addition to the preceding, difficulty of swallowing and vocalization, contraction of pupils, palpitation of heart and priapism.	In the cord, at or above the middle of the cervical region, or the level of the fourth cervical pair of spinal nerves.
In addition to above, paralysis of the phrenic nerve and diaphragm, of the sceleri, intercostals, serrati magni, and many of the accessory respiratory muscles which act upon and from the shoulder. Death resulting at once from suspension of all respiratory movements.	Anterior half of the medulla spinalis or its sheaths.
Paraplegia developing itself symmetrically.	Dorso-lumbar enlargement of cord.
Paraplegia of the legs.	Cervical enlargement of cord.
Paraplegia of the arms.	In both sides of the brain. Exceptions in cases of disease of the medulla oblongata (very rare).
Cerebral paraplegiæ, so-called, are very rare, and are in reality two distinct hemiplegiæ.	Roots of spinal nerves at point of injury, especially posterior roots, which long remain in a state of painful excitation.
Paraplegia from disease of the vertebral column.	Posterior half of med. spinalis.
Characteristic symptoms of tabes dorsalis or locomotor ataxia.	Gray substance of spinal cord in vicinity of the central canal or diffused through anterior roots.
Progressive muscular atrophy.	In one lateral half of spinal cord. The hyperæsthesia of the paralyzed
Hemiplegia with crossed hemi-æsthesia.	

TABULAR VIEW OF SPINAL PARALYSIS.

SYMPTOMS.	SEAT OF LESION.
Bilateral neuralgia of the legs and arms accompanying symptoms of tabes dorsalis.	side is probably due to paralysis of the vaso-motor nerves of that side. In posterior roots of spinal nerves and their prolongation into the gray substance of the cord.
Bilateral contractions affecting the extensor muscles.	In the spinal cord.
Unilateral contractions affecting the flexor muscles.	In the brain.*

The diseases of the spinal marrow are classified by Dr. WOOD according to the *rapidity of their onset*, as follows, the attack being considered rapid when decided paralysis has developed within forty-eight hours:—

RAPID ONSET.	SLOW ONSET.
Congestion. Meningeal apoplexy. Spinal apoplexy. Acute myelitis.	Sexual exhaustion. White softening. Chronic myelitis. Tumors:

In congestion of the cord the diagnosis rests upon: Suddenness of onset; uniform, bilateral loss of voluntary motion, reflex activity and sensation; absence of all symptoms of irritation, such as spasms or violent pains; absence of constitutional disturbance. It must also be remembered that the palsy affects first and most severely the lower limbs, but may rise to the arms, and, finally, to the muscles of respiration, and thus prove fatal; that so far as the paralysis extends, all the muscles are involved; that motion is affected more than sensation; and that very rarely, if ever, does ulceration or other indications of trophic changes occur.

In meningeal apoplexy the symptoms are also due to pressure, but the effused blood not only disturbs the cord by pressing upon it, but also irritates the membranes and the nerve-roots, especially when first thrown out. Consequently, in the first few hours or days of a meningeal hemorrhage, there are violent spasms and pains, due either to an incipient

* From Prof. J. Aitken Meigs' Lectures (not published).

meningitis, or more probably to a direct irritation of the nerve-roots. The extent and amount of the symptoms vary, of course, with the position and amount of the hemorrhage. Later there are symptoms of pressure, varying in intensity with the amount of the effusion ; and absence of febrile symptoms, unless decided meningitis be produced by the clot.

In true *spinal apoplexy* the symptoms come on with absolute abruptness. The cord is so small a body that a clot in its substance interrupts at once its function. The paralyses of motion and sensation are complete, and reflex movements are greatly exaggerated. As there is no correlation of the spinal nerve-roots, the spasms and pains of meningeal hemorrhage are wanting.

Acute primary myelitis is a very rare affection. The diagnosis should present no difficulty. The distinct febrile reaction, which is stated to be always present, separates it at once from all other acute affections of the cord proper, so that it can be confounded only with acute meningitis. Probably, in the majority of cases, it exists coincidentally with this disorder ; but even when it is isolated, the symptoms at first closely simulate those of meningitis.

COMPARISON OF ACUTE SPINAL DISEASES.

MYELITIS.	MENINGITIS.	CONGESTION.
<p>Constant pain in the spine at a point corresponding with the upper limit of inflammation, rendered more acute by pressure on vertebral spine.</p>	<p>Pain usually rheumatic in character, diffused along the spine, not increased by pressure ; but augmented by flexions of trunk.</p>	<p>Formication alternating with numbness in the beginning of the attack, especially in fingers and toes.</p>
<p>The alternate application of ice and hot sponge to spine causes the same burning sensation at seat of lesion, but above the sensation is normal.</p>	<p>Nerves, coming out through the inflamed part of the meninges, the seat of acute pain, much increased by movements of limb.</p>	<p>Only slight pain in spine, scarcely increased by pressure.</p>
<p>Sensation as of a cord or ligature around the body at the limit of paralysis always present when dorsal region is affected ; when higher up spasm of the sphincters and priapism often occur.</p>	<p>Frequent spasms of muscles of the back. Spasm of sphincter vesicæ may occur, followed by retention of urine and paralysis. Convulsive movements of paralyzed parts.</p>	<p>Frequently hyperæsthesia ; sphincters more paralyzed than in other forms of paralysis (BROWN SÉQUARD).</p>

COMPARISON OF ACUTE SPINAL DISEASES.

MYELITIS.	MENINGITIS.	CONGESTION.
Paraplegia complete.	Paraplegia varies in degree, sometimes increasing and subsequently rapidly diminishing.	Paralysis generally not limited to lower limbs, but involves upper extremities and respiratory muscles. In some cases power of moving paralyzed legs is better after resting; ordinarily, however, the paralysis is worse on first rising in the morning.
Anæsthesia or paræsthesia (except when gray matter is not involved, which is rare), muscular sensibility much impaired, early.	Anæsthesia very rare; generally hyperæsthesia.	Frequently morbid increase of sensibility.
When disease is high up in dorsal region energetic reflex movements may be produced.	Increased reflex movements, which cause pain, may be excited.	Slight spasmodic movements sometimes observed in paralyzed parts.
Marked tendency to bed sores; sloughs form early on sacrum and nates.	Less marked in uncomplicated cases of meningitis.	Ulceration occasionally happens.

CHRONIC SPINAL DISORDERS.

In the slow or chronic forms of spinal disease, *spinal tumors* may be considered first. There are three classes of phenomena to be looked for in this disease: local symptoms of diseased structures; atrocious pains at a distance from the seat of the disease, due to the involvement of nerve-roots and nerves, where they pass through the inflamed tissues; and paralytic symptoms, the results of pressure, and to some extent of a local myelitis. In cases of suspected tumors of the spine all these symptoms are to be sought after. In cancer they are often all present, and the distant pains are especially remarkable for their atrocity.

The other chronic spinal diseases may be classified with reference to the characteristic of *tremors* as follows:—

WITHOUT TREMORS.	WITH TREMORS.
Sexual exhaustion.	Paralysis agitans.
White softening.	Multiple sclerosis.
Chronic myelitis. { softening.	
Local myelitis. { sclerotic.	

The difference between *sexual exhaustion* and *myelitis* is probably one of degree only; but the former is curable; the latter is not.

In applying these symptoms in practice, we should, of course, give first attention to those which are most characteristic. Thus, if we observe, in a patient, ataxy with nystagmus, we think at once of multilocular sclerosis and not of locomotor ataxy (tabetic series), because nystagmus is a valuable symptom of multilocular sclerosis. In the same way spasmodic paraplegia (recognized by the continual trembling movements which are produced when a single blow is struck upon the muscle) we find is produced by a localized lesion in the cord, more particularly involving the lateral columns.

In order that these forms shall be better understood, it may not be out of place to review some of the chief points in the clinical history of the chief focal lesions caused by sclerosis of the cord.

In *sclerosis of the antero-lateral white columns* Dr. GOWERS* states that there is loss of voluntary power below the lesion, descending degeneration in the anterior and lateral columns (direct and crossed pyramidal tracts, especially the latter), and over-action of the lower centres. This over-action may be manifested only as excessive knee-reflex† and developed ankle-clonus (tendon-reflex), or it may increase from this to spasm and rigidity—spastic paraplegia. There is no wasting unless the degeneration extends from the lateral columns to the anterior cornua. Then we have a combination of spasm and wasting, in which, if the cornual degeneration proceeds, the spasm and rigidity may lessen as the degeneration advances. In disease limited to the lateral columns (at any rate, when the disease is limited to the pyramidal tracts) there is no loss of sensation or incoördination, and no interference of the nutrition of the skin. These symptoms of “spastic paraplegia” (*lateral sclerosis*) may arise from a primary degeneration in the lateral columns, limited thereto. Such cases are extremely rare, and in the majority the disease is a focal lesion more or less extensive at some level in the dorsal or cervical cord, and the degeneration in the lateral columns is secondary. The evidence of the latter form is afforded by the frequently sudden or rapid onset of the symptoms in the first instance (primary sclerosis being always gradual in onset), and the evidence which may generally be discovered that there has been at some time, or is in some region, damage which extends

* Address delivered before the Medical Society of Wolverhampton, Oct. 7th, 1879.

† See page 102, note on Patellar-Tendon Reflex.

beyond the lateral columns. Descending lateral sclerosis, with secondary spasmodic phenomena in the limbs, may even result from damage to the motor tracts above their decussation—in the medulla, the pons, or the motor parts of the cerebral hemispheres. It occasionally results from bilateral injury to the surface of the brain during difficult birth, but such cases are very rare.

2. In *disease of the posterior columns* there is interference with coördination without loss of power; eccentric pains, impaired sensation and diminution of reflex action, in consequence of the implication of the sensory roots. All these symptoms depend on disease of the root-zone of the posterior column. Disease of the posterior median column gives rise to no known symptoms.

The posterior column may be damaged by any pathological process, and they are frequent seats of primary degeneration. The symptoms of locomotor ataxy usually present the following order: loss of the deep reflexes, pains, incoördination, diminution of sensation; loss of the superficial reflexes, occasionally interference with the nutrition of bones and joints.

There is no loss of motor power or wasting as long as the disease remains limited to the posterior columns. It may, however, extend forward into the anterior cornua, causing muscular atrophy and weakness to be conjoined with the ataxy. Or the lateral columns may be affected at the same time as the posterior; we then have weakness as well as ataxy, but no wasting. The disease of the lateral columns causes increase of the deep reflexes, and this increase may thus coëxist with incoördination, the increased action of the reflex centres being so great that they are not arrested by the damage to the posterior root (which is often, in these cases, slight). Thus we have the anomaly of ataxy with excess of the tendon reflex instead of its loss.

An important fact to remember regarding the posterior columns is their proneness to degenerate; they recover less readily than any other part of the cord. A lesion in one spot may set up a degeneration which ultimately involves them in their whole extent. Damage affecting the whole thickness of the cord may pass away from the rest and persist in the posterior columns, and even spread there. In such a case we have ataxy succeeding loss of power. Strength returns, incoördination remains.

3. The anterior cornua contain the motor nerve-cells, which, (1) influence the nutrition of the motor nerve fibres proceeding from them, and consequently that of the muscles; (2) constitute the terminal link in the path of the voluntary impulse from the brain to the muscles; (3) form part of the reflex loop, probably also of the reflex centre, to which those muscles are connected.

Hence we have as the result of disease of the anterior cornua, (1) degeneration of the motor nerves and wasting of the muscles; (2) loss of voluntary power, *i. e.*, paralysis of those muscles; (3) interference with or arrest of the reflex actions in which these muscles take part.

The extent of these symptoms, whether they are unilateral or bilateral, affect many muscles or few, will depend strictly on the extent of the disease in the spinal cord.

Of the three symptoms the muscular wasting is incomparably the most important. Paralysis may result from disease elsewhere in the motor tract, *i. e.*, disease of the lateral column higher up. Loss of reflex action may depend on disease elsewhere in the reflex loop, *i. e.*, disease of the sensory fibres in or outside the cord. But muscular wasting is due only to a lesion of the motor cells, or to a lesion of the nerves cutting the muscles off from the influence of these cells. In most cases we are able to exclude the latter without difficulty; the state of muscular nutrition comes thus to be of the highest importance as indicative of the state of the anterior cornua of the cord.

Disease of the anterior cornua is often combined with disease of the lateral (pyramidal) columns similar to the descending degeneration. CHARCOT believes that in these cases the degeneration in the lateral column is primary, its symptom, muscular rigidity, preceding the symptom of the cornual disease, muscular wasting, and he terms the affection "lateral amyotrophic sclerosis." I believe, however, that this position will need reconsideration, and that the degeneration in the lateral columns is, sometimes at least, secondary to, or simultaneous with, the disease in the cornua. It often spreads, however, beyond the fibres related to the degenerated cornua, and so may cause weakness and spasm in the limbs below the seat of the muscular atrophy. Thus we have wasting in the arms, and weakness with spasm in the legs, and even, as I have

seen, wasting in the shoulder-muscles, and weakness without wasting in the hands.

Certain lesions may damage the motor tracts slightly and impair conduction in a peculiar way, rendering it apparently unequal in different fibres. As a consequence the muscular action is unequal in different muscles, and instead of a balanced coördinated movement we have an unbalanced jerky movement. This is seen especially when irregular islets of sclerosis affect the cord—disseminated or insular sclerosis—and, according to the researches of CHARCOT, it appears that this irregular conduction is the result of the unequal wasting of the medullary sheaths, the axis-cylinders remaining. A precisely similar symptom may result from pressure on the motor tract—as by a growth. Not rarely this “disseminated” or “insular” sclerosis in one region is combined with a system-degeneration in another. An occasional combination, for instance, is the jerking movement (from cervical insular sclerosis) in the arms, and weakness with spasm (from lumbar lateral sclerosis) in the legs.

4. A total transverse lesion of the cord at any level, however limited in vertical extent, separates all parts below the lesion from the brain, and hence, so far as will and perception are concerned, produces the same effect as if the whole of the cord below the lesion were destroyed. A section across the cord in the middle of the cervical enlargement, for instance, paralyzes all parts below the neck. Hence the extent of the paralysis indicates only the upward extent of the lesion. This is also indicated by the position of the girdle pain, or zone of hyperæsthesia, which is due to the irritation of the sensory roots in the lowest part of the upper segment—an important indication when the lesion is in the dorsal region, where the precise limitation of motor weakness may be recognized with difficulty.

Patellar-Tendon Reflex.—For the *diagnosis* of posterior sclerosis, WESTPHAL has noted the following symptom: “If a healthy man sits with one knee-joint resting upon the other (a very common attitude), and the ligamentum patellæ of the supported leg be smartly struck just below the kneecap with the side of the hand, a sudden contraction takes place in the quadriceps femoris muscle (of which the ligamentum patellæ represents the tendon), and the foot is consequently jerked upward to a degree which

varies in different individuals. Now, in confirmed examples of locomotor ataxia this reaction does not take place. No matter on what part of the ligament below the knee-cap, or with what force the blow is struck, the foot hangs motionless. In order to establish with accuracy the absence of the phenomenon, certain precautions ought to be taken. The leg should be bare; the patient must not offer voluntary resistance to the movement of his leg, and the ligament should be struck with some hard implement which can be swung like a hammer. An ordinary wooden stethoscope answers very well if it is held loosely by the small end, and the blow given with the edge of the ear-piece. But, however administered, several blows should be struck on the ligament, slightly changing the position each time, as there is generally one spot from which the reaction is peculiarly energetic. This is usually a little below but very near to the patella. Ankle-clonus may be similarly developed by tapping the tendo-Achillis.

The following are the conclusions given by ERB* in regard to the interpretation of symptoms:—

In diseases of the spinal cord, paralysis rapidly followed by a marked degree of atrophy and by the reaction characteristic of degeneration, points to disease of the anterior roots (rarely), or of the gray anterior cornua (more frequently). In this case all reflex actions are absent.

Paralysis with tension and contraction of muscles, without atrophy, is very probably due to some affection of the lateral columns.

Paralysis without loss of reflex function and without atrophy, points to an affection of the parts which ascend to the brain, outside of the gray substance, or, at least, outside of the ganglia of the anterior cornua. Such are mostly cases of circumscribed disturbances of conduction, the end of the cord below the lesion remaining intact.

Paralysis, with trophic disturbances, gives room for suspecting an affection of the gray substance, since primary affections of the roots are rare.

Very extensive palsy, with much atrophy, the reaction of degeneration, absence of reflex acts, points to a widely diffused lesion of the anterior gray substance.

Paralysis in the districts supplied by certain pairs of roots (both arms alone, or both crural nerves) points to a strictly localized affection of roots, or

* ERB.—Review in *Journal of Nervous and Mental Diseases*, Chicago, Oct., 1878.

lesion of the gray anterior cornua. The conclusions in regard to the *nature* of the lesion in the cord are far less certain than those relating to its place.

Cases of spinal paralysis, accompanied by atrophy of the muscles, whether in children or adults, acute or chronic, are described under the head of *poliomyelitis anterior, acuta and chronica*.

Destruction of the central trophic apparatus, or its separation from the peripheral parts, produces the symptoms of degenerative atrophy. "Upon the whole, we are justified in assuming a disease of the anterior cornua when the electrical examination shows the existence of the reaction of degeneration, and consequently of degenerative atrophy of nerves and muscles, *provided the disease is clearly of spinal origin*" (ERB).

In infantile palsy (lesion in the anterior cornua), observers are not agreed as to whether the change in the ganglion cell is primary or whether it is the consequence of an interstitial myelitis.

The following table will be found valuable in diagnosing certain chronic disorders (chiefly after Prof. MEIGS).

CEREBRO-SPINAL SCLEROSIS.	PARALYSIS AGITANS.	LOCOMOTOR ATAXIA.
Disease of adult life. Tingling and numbness; diminished muscular power, chiefly in the legs.	In old persons chiefly. Felt mainly in the arms.	In adults. Tingling and numbness of legs without loss of power (want of coördination exists).
Eye symptoms absent in spinal form; when they occur in cerebro-spinal form they are persistent and progressive.	No proper eye symptoms.	Ocular troubles, defective vision and accommodation, strabismus, ptosis or double vision. These symptoms temporary.
Tremor or trembling follows the paralysis.	Precedes paralysis.	Absent.
One or both limbs paretic, ultimately becoming completely powerless.	Muscular weakness in one or both arms, and then extends into lower extremities. Only rarely passing into true paralysis.	No paralysis.
In the paretic stage the gait is distinctive; the foot is swung around, describing an arc of a circle, and brought flatly upon the ground. With this eccentric curvilinear projection of the foot there is an exaggerated alternate semi-rotation of both halves of the pelvis.	In attempting to walk first balances on his feet, and starts with head and trunk bent forward on the toes or fore part of feet, and with short steps goes hopping and trotting along at almost running speed (festination).	A stumbling, staggering gait, without true paralysis.

CEREBRO-SPINAL PARALYSIS AGITANS. LOCOMOTOR ATAXIA.
SCLEROSIS.

No spontaneous tremor ; always caused by motion or excitement.	Trembling early, incessant, even when at rest ; scarcely interrupted by sleep.	No tremor.
Nystagmus, usually binocular.	Never met with.	Not present.
Articulation slow and scanning.	Articulation indistinct ; embarrassed.	Not affected.
Intellect early impaired.	Unaffected until late.	Not marked.
Boring, gnawing and lancinating pains rarely complained of.		Such pains frequently precede the loss of motion.
Early paresis, passing into paralysis, is characteristic.		Paraplegia always a late phenomenon.

(For diagnosis of Locomotor Ataxia from General Paralysis of the Insane, see page 111, under this head.)

Paralysis may also be caused by reflex irritation, and closely simulate organic disease of the cord. BROWN-SÉQUARD* gives the following points of distinction (with unimportant additions) :—

PARAPLEGIA.

FROM REFLEX IRRITATION.

1. Is *preceded* by an affection of uterus, bladder, kidneys, or prostate gland. May be caused by phimosis.

2. Usually lower limbs alone paralyzed.

3. No gradual extension of the paralysis upward.

4. The paralysis is usually incomplete, an extreme debility or weakness of the limbs rather than paralysis.

5. Some muscles more paralyzed than others.

6. Reflex power neither much increased nor completely lost.

FROM MYELITIS.

1. Usually no disease of the genito-urinary organs except as consequent on the paralysis.

2. Usually other parts paralyzed besides the lower limbs.

3. Most frequently a gradual extension of the paralysis upward.

4. Very frequently the paralysis is complete.

5. The degree of paralysis the same in the various muscles of the lower limbs.

6. Reflex power often lost ; or sometimes much increased.

* "Lectures on the Diagnosis and Treatment of Paraplegia," p. 33.

PARAPLEGIA (*Continued*).

FROM REFLEX IRRITATION.

7. Bladder and rectum rarely paralyzed; or at least only slightly so; sphincter ani weak.

8. Spasms in paralyzed muscles extremely rare.

9. Very rarely pains in the spine, either spontaneously or on application of pressure, percussion, or a hot, moist sponge, or ice.

10. No feeling of pain or constriction around the abdomen or chest.

11. No formication, pricking, or disagreeable sensations of cold or heat.

12. Anæsthesia rare, the tactile sensibility being but slightly, if at all, impaired; but the muscular sense is almost lost.

13. Usually obstinate gastric derangement.

14. Variations in the degree of the paralysis corresponding with changes in the primary disease.

15. Usually the urine is acid, unless the urinary organs are diseased.

16. Cure of the paralysis frequently and rapidly obtained, or taking place spontaneously after a notable amelioration or cure of the genito-urinary affection.

17. Usually muscles do not become atrophied, and temperature is little lowered.

18. Therapeutic results good.

FROM MYELITIS.

7. Bladder and rectum usually completely paralyzed, or nearly so.

8. Always spasms, or, at least, twitchings.

9. Always some degree of pain existing spontaneously, or caused by external excitations.

10. Usually a feeling as if a cord were tied tightly around the body at the upper limit of the paralysis.

11. Always formications, or pricking, or both, and very often sensations of pricking or heat or cold.

12. Anæsthesia very frequent and always at least numbness.

13. Gastric digestion good, unless the myelitis has extended high up in cord.

14. Ameliorations very rare, and not following changes in the condition of the urinary organs.

15. Urine almost always alkaline.

16. Frequently a slow and gradual progress towards a fatal issue, and rarely a complete cure.

17. Atrophy of muscles of the paralyzed parts.

18. Treatment of little benefit.

MR. GOWERS divides spinal lesions, according to the time required in their development, into six classes, whose comparative features are shown in the following table:—

Pressure or Growths.	{	Sudden (<i>few minutes</i>).	}	Vascular lesions.
		Acute (<i>few hours or days</i>).		
		Sub-acute (one to four weeks).		} Inflammation (myelitis).
		Sub-chronic (one to two months).		
		Chronic (two to six months).		} Degeneration.
		Very chronic (six months and upward).		

He recommends, in examining a case of disease of the spinal cord, to follow a definite plan. "First endeavor to ascertain the exact seat of the lesion; note how far the several conducting functions of the cord are impaired; and the highest level of their impairment; then ascertain the condition of the central functions, and especially muscular nutrition, and irritability and reflex action (first in the part below the level at which conduction is impaired; and secondly, at the supposed level of the lesion, and in this way you may infer, without much difficulty, what is the extent of the lesion transversely and vertically). In the next place, endeavor to ascertain its nature by considering, first, how the symptoms came on and developed; secondly, which of the lesions having this mode of onset and development are common in the region affected; and thirdly, which of them are produced by the cause or causes to which the disease is apparently due."

Some special forms of paralysis require separate discussion.

PSEUDO-HYPERTROPHIC PARALYSIS.

This is a disease of children, usually attacking them in the second year of life. At that period it is found that when they are placed upon their feet they fall down, or clutch at the nearest object, to support themselves; or in other cases it may be that the child has commenced to walk, when, without pain or fever, or sometimes after convulsions, it is found to be soon fatigued, either by walking or standing, and at length it can no longer walk or hold itself upright; or, again, it may be that the child does not walk until very late, $2\frac{1}{2}$ or 3 years, and then very feebly and imperfectly.

Symptoms.—The principal morbid phenomena are (DUCHENNE)—

1st. In the beginning, feebleness of the lower limbs.

2d. Lateral balancings of the trunk and widening of the legs during walking.

3d. A peculiar curvature of the spine (ensellure), or saddle-back (lordosis), both in walking and standing.

4th. Equinism (talipes equinus), with a peculiar over-extension of the first phalanges of the toes, which DUCHENNE calls "griffe des orteils."

5th. Apparent muscular hypertrophy.

6th. Stationary condition.

7th. Generalization and aggravation of the paralysis.

When the disease has arrived at the stage of apparent hypertrophy, the appearance of the patient is very characteristic, and its true nature would be at once obvious to any one who had any knowledge of its symptoms; but in the earlier stages there is but little to guide us to a diagnosis unless we have some hereditary history. Of the hereditary nature of this affection the published cases give ample proof.

There are apparently two forms of this disease, one of spinal the other of muscular origin.

PARALYSIS, FROM LEAD POISONING AND HYSTERIA.

In this form of paralysis the usual diagnostic symptoms, to wit, a history of exposure to lead, the blue line on the gums, constipation and colic, may all be absent; hence the diagnosis must rest upon the peculiar characters of the palsy—especially the effects of electric currents upon the muscles. These are the only reliable evidences of the nature of the disease. These characteristic reactions, first described by DUCHENNE, are as follows:—

Excitability to Faradism absent or sensibly diminished in all the muscles of the forearm except the supinators longus and brevis. In health the supinator brevis cannot be directly Faradized, on account of its deep position. But in lead palsy it very often happens that the wasting of the extensor communis digitorum has proceeded far enough to uncover the supinator brevis sufficiently to allow a small rheophore to be applied to it in the space of about a square inch at the upper and back part of the forearm. If it be found (both arms being affected) that the common extensor fails to respond to Faradism while the short supinator close by,

on a lower plane, is readily excited by it, the case may be positively set down as one of lead palsy.

Hysterical Paralysis, in spite of its frequent close imitation of the organic forms, is readily diagnosed by attention to the following points:—

1. In hysterical hemiparesis the face is rarely, and the tongue never, affected.

2. In hysterical paraplegia incontinence of urine is never present (HAMILTON).

3. No amount of help can keep the patient from staggering or falling when she attempts to walk (REYNOLDS).

4. The foot in walking is simply dragged along and not swung as in organic hemiplegia (TODD).

5. In all sudden cerebral palsies, the nails of the affected extremities cease to grow. In hysterical palsies, of one limb or both, whether paraplegic or hemiplegic, the rate of nail-growth is unaltered (WEIR MITCHELL).

GENERAL PARALYSIS OF THE INSANE.

This curious disease, long unknown in the United States, has of recent years been frequently observed in the Northern and Eastern States, but so far, rarely or not at all in the South and West. It is a disease of advanced life, whose pathognomonic characteristics are constant troubles of motility, a progressive loss of mental power, and a constant belief on the part of the patient that he is perfectly well, and in the enjoyment of magnificent fortune and gigantic powers (*delires des grandeurs*).

The following are the progressive traits of the disease as generally observed:—

PSYCHICAL SYMPTOMS.—1. General restlessness and unsteadiness of mind, with impairment of attention; alternating with apathy and drowsiness.

2. A change in disposition and temper, and a general loss of self-restraint; at first as regards trivial social observances, and then as regards general conduct.

3. Impairment of the reflective powers, so that there is no logical and systematic development of thought.

4. General exaltation of thought, with a profusion of remembered images and ideas, and numerous extravagant desires.

5. Failure of memory and forgetfulness; at first of words, and then of events.

6. Delirious conceptions, and the transformation of desires into beliefs, these being generally connected with personal greatness and power.

7. Hallucinations of the senses, in which remembered sense impressions are so vivid and intense as to spread to the periphery.

8. Maniacal restlessness and excitement, in which present impulses and feelings instantly pass over into action.

9. Increased mental weakness, with the incoherent and fragmentary repetition of the false ideas previously entertained.

10. Failure of the senses, with more marked impairment of memory.

11. Complete fatuity, passage into coma and death.

MOTOR SYMPTOMS.—1. Persistent contraction of the occipito-frontalis muscle, and some dilatation of pupils, causing the eyes to be widely opened and the forehead wrinkled, and giving an expression of surprised attention to the face.

2. Persistent contraction and frequent tremors of the zygomatic muscles, giving a pleased and benevolent expression of countenance.

3. Slight muscular restlessness and unsteadiness.

4. Impairment of the power of executing fine and detailed movements, so that manipulative skill is lost while movements *en masse* are still well performed.

5. Fibrillar tremors of the tongue, and some loss of control over its movements, so that it is protruded with difficulty; is rolled about when protruded, and is suddenly withdrawn.

6. Twitchings of the nostrils and upper lip, with frequent tremors of the latter.

7. Impairment of articulation, which is thick and wanting in distinctness.

8. An alteration in the voice, as well as thickness and hesitancy in speech.

9. Loss of control over the combined movements of the hand and wrist, so that the handwriting generally deteriorates.

10. Changes in the pupils, which are at first irregularly contracted, and then become irregularly dilated.

11. An alteration in gait, which becomes unsteady; the more complex movements of the thighs, leg, and foot, and the balancing of the pelvis on the hip joints, being performed with difficulty.

12. General muscular agitation and restlessness.

13. Gradual loss of power in the muscles of the face, tongue, neck, and limbs.

14. Spasmodic contraction of the masseter muscles, causing grinding of the teeth.

15. Convulsive seizures—most marked on one side of the body, and followed by transitory hemiplegia.

16. Loss of control over the sphincters.

17. Complete prostration of muscular strength and helplessness, and difficult deglutition.

18. Contractions of the muscles of the limbs, and paralysis of the muscles of respiration.

The main diagnostic difficulty is to distinguish this from some phases of locomotor ataxy. The differences are that in general paralysis the mental symptoms are always present, and always precede the motor phenomena. The first symptoms in general paralysis are chiefly cerebral; viz., mental excitement, great garrulity, noisy hilarity, bragging, early violence of behavior, and very usually some exhibition of libidinous conduct; on the subsidence of excitement, the mind is found to be weak, and the motor phenomena gradually make their appearance.

In ataxia, the commencement is in the spinal functions. There is first an attack of pain of some remote part, occurring most frequently in the lower extremities, and dating several years back, considered at the time perhaps to be rheumatic; this pain is worse toward evening, or when the patient is not mentally occupied; it may improve or disappear for a time and return. Then follows a slight degree of numbness of the part; the patient feels as if he trod on wool; occasionally "pins and needles" attack the part; in fact, those phenomena which we have all experienced after sitting in an awkward position, when one's own leg has "gone to sleep." There is, as most of us know, want of feeling, want of recognition of the member, especially as to its size, and even its ownership, then atrocious pain, and pins and needles. In the disease, on the subsidence of the pain, the patients exhibit some awkwardness in gait; the ataxy or want

of order on the movement is evident. These symptoms may extend over ten or twelve years with very little change, except, perhaps, increasing awkwardness in gait; there is doubtless some numbness of the cutaneous surface in the course of the disease; the phenomena appear to spread upward by involving the functions of the nerves higher up; the erection of the penis, and soon afterward the sexual appetite, are lost, and, as the disease ascends, the expulsive power of the bladder and rectum become impaired. All this occurs while little change takes place in the mental functions; but in other cases the mind appears imbecile, the memory is affected, and there is distinct alteration in behavior and conduct; but there are no lofty ideas, no excessive excitement and garrulity, and in no case paroxysms of violence, or libidinous ideas.

The differences may be better seen in a tabulated form:—

GENERAL PARALYSIS.	LOCOMOTOR ATAXY.
Runs its course in a few years.	Is much slower usually, and may last ten or even twenty years.
Commences with mental symptoms.	Commences with pains in distal nerves.
Is attended with libidinous ideas.	Is attended with absence of sexual feeling.
The motor symptoms are secondary in the order of time.	The motor symptoms are the primary phenomena.
Is only rarely complicated with pelvic difficulties.	Pelvic symptoms are a prominent feature.
There often is great violence.	The mental phenomena are imbecility and impaired memory.

There is also a form of general paralysis due to syphilis. The differential features of this variety have been clearly defined by Dr. E. C. SEGUIN.* We do not obtain the regular gradations and stages of the true disease. The moral perversion which is peculiar to general paralysis is absent, neither do we see the pure, exalted notions. The fibrillary tremors that are so well marked in general paralysis are not present here. The articulation is more mumbling in character. We, likewise, are apt to have a great deal of actual paralysis of cranial nerves or body in these

* *Hospital Gazette*, September, 1878.

cases. In true general paralysis, after attacks of hemiplegia, the patient regains his full strength, whereas this is not apt to occur in the syphilitic variety. The following table will perhaps show clearly the main differences:—

TRUE GENERAL PARALYSIS.	SYPHILITIC GENERAL PARALYSIS.
Prodromic stage.	Absent.
Exalted notions, numerous and varied, and relatively exalted according to the position in life.	Rare or absent.
Speech is tremulous and jerky.	Speech is thick.
Tremor of hands and lips.	Absent as a rule.
Preservation of strength.	Paresis or actual paralysis.
Pupils are apt to be contracted.	Apt to be open or wide.
None.	Palsy of third or of other cranial nerves.
None.	Headache nocturnal.
Transient aphasic attacks.	More serious aphasic attacks.
Spontaneous remissions.	Progressive except under treatment.

Some other differences between the two conditions are as follows: The patient with syphilis has none of the cravings or abnormal appetites of the other; the latter feels an impulse to get drunk or to have an excess of coition. The tendency to excessive coition is absent in syphilitic paralysis, and, indeed, there is a marked loss of the virile power. The temperature changes are also absent in syphilis of the brain. The rise in temperature in general paralysis of the insane is very great, often reaching 103° in exacerbations. There is no rise of temperature in syphilis of the brain, except, perhaps, when the patient has hemiplegia from a large lesion.

The most important point is, that in syphilis there is a paralysis; in general paralysis there is irritation and incoördination without true paralysis.

SPINAL IRRITATION (SO-CALLED) AND SPINAL WEAKNESS.

This affection has been described by some writers as spinal hyperæmia, by others as spinal anæmia; again as spasms of the spinal muscles, and lastly as abnormality of the spinal cells. Some have denied its existence altogether; but in fact it is a distinctly defined and not unusual disorder. About five-sixths of the cases are females, and it is often associated with uterine or ovarian disease; and as often has some antecedent history of a blow upon or other slight injury to the spine.

Its symptoms are of the most varied kind, so much so that it may simulate almost every known ailment; but a *careful examination of the spine* will reveal its true character. The diagnostic rules laid down by Drs. WILLIAM and DAVID GRIFFIN, in 1834, who first described the disease, have never been improved upon. They are as follows:—

1. The pain or disorder of any particular organ complained of is altogether out of proportion to the constitutional disturbance.

2. The complaints, whatever they may be, are usually relieved by the recumbent position, are always increased by lifting weights, bending, stooping, or twisting the spine; and, among the poorer classes are often consequent to the labor of carrying heavy loads, drawing water, etc.

3. The existence of tenderness at that point of the spine which corresponds to the disordered organ, and the increase of pain in that organ by pressure on the corresponding region of the spine.

4. The disposition to a sudden transference of the diseased action from one organ or part to another, or the occurrence of hysterical symptoms in affections apparently acute.

5. The occurrence of continued fits of yawning or sneezing. These are not very common in the disease; but when they do occur, they may generally be considered as characteristic of nervous irritation.

To this it may be added that the tenderness may extend along the spine generally; but is always greater in one or two spots. Gastric symptoms, headache and languor are usually well marked in spinal irritation; but there is neither atrophy, paralysis (except hysterical) nor waist constriction, which serve to distinguish it from a large class of spinal diseases.

In regard to the nature of *spinal irritation*, we know nothing definite at present (ERB).

Neurasthenia spinalis, spinal nervous weakness, is defined as a diseased condition in which marked and unquestionable disturbances of the functions of the cord exist, for which no considerable anatomical basis can be found or assumed; a disease, therefore, which must at present be classed among the functional disorders. It is most common among men, is caused by successive mental efforts, sexual excesses, bodily over-exertion. Weakness and inability from prolonged over-exertion, dysæsthesia, paræsthesia, cold hands and feet, sleeplessness and general malaise are the prominent symptoms.

“We do not at all know what may be the nature of the processes of nutrition which cause these symptoms. We, however, believe, that we are certainly entitled to locate them in the cord, especially in the lower portion, the lumbar region. The most obvious view is that which supposes that the physiological fatigue of the nervous elements, which always occurs after severe and protracted irritation, becomes exaggerated and assumes a fixed form; in such a case we may suppose that the fatigue of the nervous elements does not become repaired in the prompt manner which is usual under physiological conditions.”

In making a diagnosis, these characters will be distinguished from those of disease of the vertebræ by careful physical examination, and the age, sex and history of the case.

· HYSTERIA.

Few diseases present at times greater difficulties to diagnosis than this protean complaint. Its counterfeits of various maladies will be considered elsewhere (see the Index); at present we shall seek for a pathognomonic symptom of the general condition.

One is offered by Dr. THOMAS BARLOW.* Rejecting as unsatisfactory all statements depending upon the patient's veracity, he finds a diagnostic test in the presence of *analgesia*. If, while the patient's attention is directed to something else, a needle be introduced into the forearm and no wincing occurs, there is the strongest presumption that we have to do with a case of hysteria. Again, it has been long known that hysterical patients are

* *Medical Times and Gazette*, February 1878.

often extremely tolerant of laryngoscopic examination. Great advantage will be found in examining a presumed hysterical patient's larynx, and thus fixing her attention while somebody at the same time inserts a needle into her forearm. Absolute tolerance of these two simple methods of examination is quite decisive.

Another characteristic relates to the pain so frequently complained of. While it is stated to be exceedingly acute, and the part tender to the slightest pressure, if the attention of the patient is engaged, very firm pressure may be made without the patient wincing. Moreover, there is noted very often a co-existence of severe pain in the epigastrium, the left side and spinal column—the *trepied hystérique*, or hysterical tripod of French authors.

The *globus hystericus*, a sensation of a foreign body in the throat caused by spasmodic contraction of its muscles, is a common symptom. The urine may be suppressed, or may be limpid and watery, and of unusual quantity.

If with these traits are united youth and female sex; ovarian or uterine disturbance; the general symptoms harmonious and exaggerated; the mind clear; and the disappearance of contractions, etc., under anæsthesia; the diagnosis is complete.

The most serious mistake would be the confounding of a hysterical paroxysm with an epileptic fit. The following table of distinctions between the two is given after CHARCOT and DA COSTA:—

EPILEPSY.

Sudden and complete loss of consciousness.

Livid face; escape of frothy saliva from the mouth; eyelids half open; eyeballs rolling; grinding of the teeth; biting of the tongue; more or less insensibility of the pupils to light.

Distortion of countenance.

Patient evinces no feeling.

Aura epileptica of short duration.

HYSTERIA OR HYSTERO-EPILEPSY.

Gradual or only partial or apparent unconsciousness.

Face flushed or complexion unaltered; no froth on lips; eyelids closed; eyeballs fixed; neither grinding of the teeth nor biting of the tongue; pupils react readily.

No distortion of countenance.

Patient sighs, or laughs, or sobs.

Aura often prolonged one or two days. Globus hystericus.

EPILEPSY.

Convulsions often more marked on one side than on the other; and more tonic than clonic. Agitation maniacal and disorderly.

Paroxysms generally of short duration.

Paroxysm followed by a heavy, half comatose sleep, by headache, and dullness of intellect. Stertor. No hallucinations.

Frequently occurs at night.

No particular connection with uterine disturbance; although a paroxysm often takes place at the menstrual period.

HYSTERIA OR HYSTERO-EPILEPSY.

No such differences; convulsions clonic. Agitation emotional, often *en pose*.

Paroxysms generally of longer duration.

Paroxysm not followed specially by sleep; patient often, after attack, wakeful and depressed in spirits. Little or no stertor. Hallucinations.

Rarely occurs at night.

Often connected with disorders of the uterus, or of menstruation.

NEURALGIA.

The positive signs which distinguish a case of pain to belong to the neuralgiæ are succinctly set forth by Dr. FRANCIS E. ANSTIE,* as follows:—

(1) The first and most essential characteristic of a true neuralgia is that the pain is invariably either frankly intermittent, or at least fluctuates greatly in severity, without any sufficient and recognizable cause for these changes.

(2) The severity of this pain is altogether out of proportion to the general constitutional disturbance.

(3) True neuralgic pain is limited with more or less distinctness to a branch or branches of particular nerves; in the immense majority of cases it is unilateral, but when bilateral it is nearly always symmetrical as to the main nerve affected, though a larger number of peripheral branches may be more painful on one side than on the other.

(4) The pains are invariably aggravated by fatigue or other depressing physical or psychological agencies.

These four cardinal points of the diagnosis may be further supported

* "Neuralgia and its Counterfeits," p. 565.

by the history of the patient. Either (1) he has previously been neuralgic, or liable to other neuroses, or comes of a neurotic family; or (2) there has been malarial poisoning of the blood; or (3) there has been some long continued peripheral or central irritation; or (4) finally, there has been constitutional syphilis.

The pains with which neuralgia is most likely to be confounded are those arising from myalgia, spinal irritation, locomotor ataxia, cerebral abscess, alcoholism, syphilis, chronic rheumatism, and latent gout.

In comparing the pains of myalgia and neuralgia the following are the more important points:—

NEURALGIA.

Follows the distribution of a recognizable nerve or nerves.

Accompanies an inherited or acquired nervous temperament which is obvious.

Is usually not much or at all aggravated by movement.

Is at first not accompanied by local tenderness.

Painful points, when established in a later stage, correspond to the emergence of nerves.

Pain not materially relieved by any change of posture.

MYALGIA.

Attacks a limited patch or patches that can be identified with the tendon or aponeurosis of a muscle which, on inquiry, will be found to have been hard worked.

Often occurs in persons with no special neurotic tendency.

Is inevitably and very severely aggravated by every movement of the part.

Distinguished from the first by localized tenderness on pressure as well as on movement.

Tender points correspond to tendinous insertions and origins of muscles.

Pain usually completely and always considerably relieved by full extension of the painful muscle or muscles.

Treatment also offers a diagnostic means. The pains of myalgia will ordinarily disappear at once by retaining the affected muscle at full extension, surrounding it with moist warmth, and giving 20 or 30 grains of muriate of ammonia internally.

Spinal irritation (spinal congestion or spinal anemia) is almost exclu-

sively confined to women. There are nearly always hysterical symptoms, marked superficial tenderness over large portions of the surface, often merely cutaneous and becoming less acute with firm pressure. There are nearly always tender spots along the spine, and also over the epigastrium and the left hypochondrium.

Locomotor ataxia is mentioned elsewhere, and its symptoms described in sufficient detail. (See page 100 *et seq.*)

Cerebral abscess, though rare, may give rise to a regrettable mistake, especially in its early stages, where severe pain in the head is almost the only conspicuous symptom. At this period the diagnosis from neuralgia must rest on the following points of contrast:—

CEREBRAL ABSCESS.

Often occurs secondarily to caries of internal ear, and purulent discharges, the result of scarlet fever, measles, etc., in childhood.

Frequently follows a blow or injury.

No true "points douloureux."

Usually the pain does not completely intermit.

Pain often excruciating from a very early period.

Pain often limited in situation, seems deep-seated, though as often as not it has no relation to the site of the abscess.

No well localized vaso-motor or secretory complications.

Very rare in old age; then usually traumatic.

Relief from stimulant narcotics very transitory.

NEURALGIA OF THE HEAD.

Rarely appears before puberty. No local assignable cause.

Comparatively seldom caused by a blow or other external injury, or caries of the bone.

If severe, soon presents, in most cases, the "points douloureux."

Intermissions of pain complete and of considerable length.

Pain usually not very violent at first.

Pain superficial; follows distribution of recognizable nerve branches belonging to the trigeminus or great occipital.

Usually lachrymation, or congestion of the conjunctiva, etc.

Severe neuralgia is commonest in advanced life.

Relief from opium, etc., is much more considerable and permanent.

The pains of *chronic alcoholism* often closely simulate those of true neuralgia. The habits and history of the patient, when known, point to the true origin of the suffering; also the insomnia, loss of appetite, foul breath, furred tongue and haggard countenance of the drunkard; and especially that the pains complained of *encircle the limbs near the joints*, rather than run longitudinally the course of the nerves in the limb, are all significant.

The osteocopic pains of *sypphilis* are usually symmetrical; are aggravated by the warmth of the bed; are generally referred to the superficial bones, and do not exist long without some other and decisive symptoms of the poison manifesting themselves.

Chronic rheumatism and gout are each attended with such marked collateral disturbances that the suspicion of their presence can readily be set at rest or sustained.

INSANITY.

The principal forms of insanity are commonly considered under the head of Mania, Monomania, Melancholia, Dementia, Idiocy and Imbecility; to which may be added emotional and impulsive insanity, which is now usually admitted. There is no ground for recognizing as a distinct variety, moral insanity (FLINT).

The pathological anatomy of insanity is thus stated by SEGUIN:—

- | | | | | |
|---|---|--|---|---|
| I. Acute Recent Cases. | { | Vascular changes..... | { | Anæmia.
Hyperæmia.
Serous effusion. |
| | { | Old congestion.
Hemorrhages into perivascular sheaths.
Changes in gray matter (not demonstrable under the microscope). | | |
| II. Chronic Insanity and True Dementia..... | { | Congestion or anæmia.
Atheroma of vessels.
Membranes changed, diseased and thickened.
Nerve elements degenerated and atrophied.
General atrophy of convolutions; most marked in anterior convolutions. | | |
| III. General Paralysis.. | { | Similar to preceding, but differs in distribution of lesions; particularly in neuralgia; also granular ventricular changes. Often lesions in spinal cord co-exist. Sub-arachnoid hemorrhage not a frequent change. | | |
| IV. Syphilitic Insanity. | | | | |

In mania, chronic alcoholism and general paralysis, *pachy-meningitis hemorrhagica* is often found. Section through the thickened dura reveals alternate layers of tissue and coagulated blood.

Patients under twenty-five years of age seldom have chronic insanity; when they do the vascular changes are less marked; in older patients the vessels become fatty and atheromatous. The capillaries show fatty change, their nuclei being first affected; in the arteries the muscular coat becomes fatty. Through large tracts of brain granular and amyloid bodies are found. Old inflammatory changes in the meninges are quite common. The arachnoid is opaque in spots.

The causes of insanity are very complex; the PHYSICAL CAUSES are thus given by the same author:—

Injuries to head.....	{	Concussion. Hemorrhages. Meningitis. Depressed fracture. Abscess. Tumor.
Syphilis.....	{	Gummy formations. Arteritis. Meningitis, etc.
Dyscrasiæ	{	Malaria. Narcotic poisoning, etc.
Alcohol	{	Delirium ebriosum. Delirium tremens.
Sexual excess.....	{	Individual venery. Masturbation.
Peripheral irritation.....	{	Uterine difficulty. Vaginismus. Masturbation.

The MORAL CAUSES are misery, depression, emotions, excitement, remorse, fear, grief, religious fervor, excess of joy, the spirit of speculation, etc.

The two forms, *mania* and *melancholia*, have their general distinctions, as follows:—

MANIA.

Ego elated and over active.

Joy and excitement generally prevail, sometimes comic emotions characterize attacks.

Over-ideation and over-action. Resulting therefrom incoherence and delirium and violent acts; general restlessness.

Insomnia.

MELANCHOLIA.

Ego is depressed and does not react normally on external world.

Sadness and fear; religious feelings strongly developed.

Reduced ideation. { few motions
and even absolute
silence.

Reduced action. { Immobility
relative or total,
and even cataleptoid state.

Insomnia (less marked).

PHYSICAL SYMPTOMS.

Increased circulation.

Increased calorification.

Increased (?) assimilation.

Increased voracity.

Lessened circulation.

Lessened calorification.

Lessened assimilation.

The earliest symptoms of insanity are a marked change in the habits; proneness to excitement and loss of control; an alteration in the emotions; failure of memory; untidiness of dress; insomnia and disturbing dreams; unusual loquacity or taciturnity; defective reasoning; accepting as real various fancies and illusions; a furtive, watchful air; groundless suspicions of those around. In combination with these mental symptoms, the pupils are often dilated, frequently irregular, and sluggish in obeying the stimulus of light; and a pulse hard, rapid and variable, 100 or over, a pulse which is not equal in both wrists (HENRY HOWARD). The tongue is pasty, the breath foul, and the bowels constipated. The digestion is impaired, and the appetite irregular and capricious. There is encroachment of the senses upon each other; the sense of sight, for instance, is substituted by audition, and the patient will describe scenes with the greatest minuteness of detail as occurring in the neighborhood, even to the color of clothing, and peculiarities of appearance which could

only be learned by inspection, but which he knows, because he "heard the dogs barking," or the noise of some fancied tumult. The hearing of inaudible voices calling opprobrious names is even popularly recognized as indicating insanity. Where no organic changes can subsequently be detected in the brain, we are forced to the conclusion that there is either some non-recognizable defect, or that there must be functional disorder of the brain, as of other organs in the economy.

CHAPTER II.

DISEASES OF THE RESPIRATORY APPARATUS.

DISEASES OF THE LARYNX.—*Symptoms of Laryngeal Diseases—Diagnostic table of Acute Laryngitis, Chronic Laryngitis, Syphilitic Laryngitis, Tubercular Laryngitis, Perichondritis, Benign Growths, Malignant Growths, and Neuroses of the Larynx—Croup and Diphtheria; Spasmodic Croup, Inflammatory Croup, Membranous Croup, and Diphtheria—Tonsillitis, Catarrhal and Parenchymatous.*

DISEASES OF THE LUNGS.—*The Regions of the Chest—Normal Differences between the two sides of the Chest—Methods of Physical Examination—Normal Respiratory Sounds—Normal Voice Sounds—Abnormal Percussion Sounds—Abnormal Respiratory Sounds—Abnormal Voice Sounds—General Rules for Diagnosis—The Forms of Phthisis (Catarrhal, Fibroid, Tubercular)—The Diagnosis of Incipient Phthisis—Diagnosis between Incipient Phthisis and Bronchitis—Clinical History of Phthisis—Acute Phthisis—Syphilitic Phthisis—Bronchitis, Acute and Chronic—Capillary Bronchitis compared with Pneumonia—Pneumonia and Pleurisy—Pleurisy with Effusion and Pneumonia with Consolidation compared—Diagnosis between Pneumonia and Pulmonary Apoplexy—Pulmonary Embolism—Asthma—Pneumothorax and Pneumo-hydrothorax—Emphysema, Vesicular and Interlobular—Cancer of the Lung.*

In studying diseases of the respiratory apparatus, we find in addition to the disorders of the lung proper and its serous investment, the pleura, that there are associated organs which likewise may be the seat of disease; these are the bronchi and trachea, the larynx, pharynx and upper air passages. We commence with:—

DISEASES OF THE LARYNX.

The general symptoms of laryngeal diseases, together with their causes and examples, may be arranged in the following tabular form:—

SYMPTOMS OF LARYNGEAL DISEASES.

SYMPTOMS.	CAUSES.	EXAMPLES OF DISEASE.
Dysphonia.	Alteration in the vocal cords from thickening, ulceration, diminished tension, morbid growths, etc.	Acute and chronic laryngitis. Laryngeal phthisis. Papillomata, etc.
Aphonia.	Non-approximation of the vocal cords, either mechanical or due to paralysis of some of the muscles attached to them.	Cicatrization. Swelling of arytenoid cartilages. Tumors. Hysteria. Pressure on recurrent laryngeal nerves, etc.
Dyspnœa.	Narrowing of the orifice of the glottis.	Paralysis of muscles opening glottis. Laryngismus stridulus. Œdema, growths and cicatrices contracting rima glottidis, and pressure external to larynx.
Stridor.	Always accompanied by dyspnœa, and produced by the same causes.	As in dyspnœa.
Cough.	Irritation of the laryngeal mucous membrane, or the nerves of the larynx.	In most laryngeal diseases it is of a peculiar shrill, brazen character.

Laryngitis has been divided by some writers into the following forms:—

Œdematous laryngitis.	Diphtheritic laryngitis.
Catarrhal laryngitis.	Syphilitic laryngitis.
Erysipelatous laryngitis.	Tubercular laryngitis.
Croupous laryngitis.	Exanthematous laryngitis.
	Traumatic laryngitis.

Among authors who have paid especial attention to this subject, there are few that stand higher than Mr. LENNOX BROWNE, of London, who in his recent work on the *Diseases of the Larynx* gives the diagnostic table presented in the following pages.

SYMPTOMS.	ACUTE LARYNGITIS.	CHRONIC LARYNGITIS.
A.—Functional, or Subjective.		
VOICE.	Hoarse, becoming aphonic.	Hoarse, uncertain, easily fatigued.
RESPIRATION.	Not embarrassed prior to œdema; then stridor, dyspnoea, and even apnoea.	Seldom embarrassed.
COUGH.	Dry, hard, shrill, metallic; aphonic; on exudation, moist.	Irritative, with slight expectoration of glutinous pellets.
DEGLUTITION.	Painful when œdema has taken place, or from associated pharyngeal inflammation.	Rarely affected.
PAIN AND ALTERED SENSATION.	Sensation of tightness and constriction; tender to external pressure.	Painless; sense of fatigue after vocal exercise.
B.—Physical, or Objective.		
COLOR.	Intense, uniformly increasing superficial hyperæmia; translucent on event of œdema.	Partial and modified submucous hyperæmia.
FORM AND TEXTURE.	Thickening and stenosis from œdema, loss of tissue rare, except in phlegmonous form.	Occasionally slight erosion, never ulceration, thickening or narrowing.
POSITION.	Unaltered.	Unaltered.
C.—Miscellaneous.		
EXTERNAL.	Pharynx usually synchronously implicated.	Pharynx usually synchronously implicated.

SYPHILITIC LARYNGITIS.	TUBERCULAR LARYNGITIS.
<i>Secondary.</i> Hoarse.	Sometimes aphonic in earlier stages ;
<i>Tertiary.</i> Characteristically raucoux ; seldom aphonic.	completely lost in advanced disease.
<i>Secondary.</i> Unchanged.	Early hurried ; greatly embarrassed
<i>Tertiary.</i> Increasing embarrassment according to amount of stenosis.	with advance of disease.
<i>Secondary.</i> Slight hacking.	Greatly influenced by amount of
<i>Tertiary.</i> Infrequent, with but slight expectoration, unless perichondritis supervene.	lung disease ; painful. Expectoration variable ; generally frothy.
<i>Secondary.</i> Normal, unless deposit on epiglottis or arytenoids.	Extremely difficult and painful, from early period to termination.
<i>Tertiary.</i> Often difficult ; very rarely painful.	
Characteristic absence of pain except when cartilages are attacked.	Pain only experienced in functional acts.
<i>Secondary.</i> Mottled, more or less symmetrical hyperæmia.	Anæmia followed by opaque grayish color ; margins of ulcers hyperæmic.
<i>Tertiary.</i> Hyperæmia of portion attacked prior to ulceration ; permanent infiltrated appearance.	
<i>Secondary.</i> Occasional superficial ulceration at vocal process ; slight general submucous infiltration.	Solid submucous thickening of epiglottis and aryepiglottic folds, elevation and ulceration of racemose glands giving worm-eaten ulcers, which commingle and attack deeper tissues.
<i>Tertiary.</i> Deep, circumscribed destructive ulcers, of yellowish color, followed by cicatricial narrowing, occasionally paralysis and quasi-new formations.	
<i>Secondary.</i> Unaltered.	No displacement ; tendency for
<i>Tertiary.</i> Deformity from intrinsic cicatrices and pharyngeal outgrowths.	thickened parts to transgress boundaries of pharynx.
<i>Secondary.</i> Pharynx and skin generally recently implicated.	Lungs either primarily, synchronously, or subsequently involved.
<i>Tertiary.</i> Seldom synchronous implication, but usually scars of previous similar pharyngeal ulceration, and possible adhesion.	Generally anæmia, rarely ulceration of pharynx. General emaciation.

SYMPTOMS.	PERICHONDRITIS.	BENIGN GROWTHS.
A.—Functional, or Subjective.		
VOICE.	Painful, easily fatigued, but not necessarily impaired.	Very variable, from slight hoarseness to complete aphonia, even in the same case.
RESPIRATION.	Variable, according to cartilage attacked.	Seriously embarrassed in one-sixth of cases; depends on situation.
COUGH.	Generally early spasmodic; with caries characteristic. Purulent expectoration, unless abscess is encysted.	Generally limited to effort to dislodge foreign body; may be expectoration of atoms of growth.
DEGLUTITION.	Varying from dysphagia to aphagia, according to pressure on gullet.	Only impaired in rare cases, in which epiglottis or aryepiglottic fold is involved.
PAIN AND ALTERED SENSATION.	Pain variable with cause; most severe in gouty form, but not then constant.	Characteristically absent.
B.—Physical, or Objective.		
COLOR.	Hyperæmia generally limited to portion attacked, sometimes extending to contiguous vocal cord.	Variable with nature of neoplasm; slightly increased vascularity of mucosa generally.
FORM AND TEXTURE.	Ulceration often absent, substituted by encysted abscess, causing narrowing, compression and paralysis.	Varies with situation, size, and nature of growth, never ulceration. May cause narrowing and paralysis.
POSITION.	May be considerable alteration of supra and infraglottic space.	Position of normal parts seldom changed.
C.—Miscellaneous.		
EXTERNAL.	Occasional constitutional manifestations.	Nil.

MALIGNANT GROWTHS.	NEUROSES.
Impaired by mechanical causes when invaded from pharynx; may be early lost in primary disease.	Lost in bilateral paralysis of adductors; impaired in other paralyses; not necessarily in spasm.
Early quickened on exertion; later paroxysmal dyspnoea from stenosis or compression.	Only embarrassed in paralyses of adductors and in spasmodic affections.
Not necessarily present; expectoration scanty; occasionally blood and portions of neoplasm.	Paroxysmal, when recurrent is implicated and in spasmodic affections.
Always difficult and painful; often the earliest symptom.	But slightly impaired or unaffected.
Ever present and severe, extending upward to the ears, and to sympathetic glandular enlargements.	Only experienced when sensory system affected. Diminished sensation in motor paralyses and in anæsthesia.
Increasing localized vascularity tending to lividity in any part except vocal cords or ventricles, when neoplasm is whitish-gray or pale rose.	In paralysis of abductors, occasional vascularity of affected vocal cords.
May cause compression, narrowing and paralysis before ulceration, which is always accompanied by thickening. Extensive indolent, gray, greenish, or almost black ulcers.	Form of glottis varying with nature of paralysis, without extrinsic thickening.
Early displacement, especially when invading from pharynx, and when neighboring glands enlarged.	Paralyzed cord not displaced, but often fixed in one position.
Glandular infiltration, but complete immunity of other organs of body from similar disease both prior and subsequent to appearance in laryngo-pharynx. General emaciation.	Sympathetic functional disturbances in other organs, or organic disease of cardiac or lymphatic system, or associated cerebral disease or chronic toxæmia.

The chronic laryngitis of syphilis cannot with certainty be distinguished from the other forms of chronic laryngitis without inquiry into the history of the case; although a probable diagnosis may be made where treatment by anti-syphilitic remedies is successful.

In tertiary syphilis there is deep and extensive ulceration, not necessarily preceded by thickening; the epiglottis is attacked early, the ulceration is often followed by cicatrization and contraction, causing stenosis of the larynx.

In the study of laryngeal diseases the use of the laryngoscope is indispensable to correctness of diagnosis. We take it for granted that the practitioner is conversant with this instrument, and the proper methods of employing it. It reveals the physical or objective local symptoms, which are of much more value than the subjective ones derived from the patient's statements.

CROUP AND DIPHTHERIA.

The general sign common to this class of diseases is a *laryngeal stridor*; they are divided into those where there is a formation of false membrane and where there is not.

Without false membrane.

Spasmodic croup or laryngismus stridulus.

Inflammatory croup, simple catarrhal laryngitis.

With false membrane.

True croup or membranous croup.

Diphtheria.

The diagnosis between spasmodic and inflammatory croup is as follows:—

SPASMODIC CROUP.

Onset sudden, usually at night, with few or no prodromal symptoms.

Difficulty of swallowing absent or temporary.

Febrile symptoms absent, or much less marked.

Larynx little affected.

Intermission complete, or nearly so, between the croupous attacks.

Very rarely fatal.

INFLAMMATORY CROUP.

Onset gradual, with sore throat, tickling, tenderness of larynx and catarrh.

Increasing difficulty in swallowing.

Flushed face, hot, dry skin, high temperature (105°), frequent pulse.

Mucous membrane of larynx red and swollen, sometimes œdematous.

Remission but slight; local symptoms and pyrexia continue.

In early life a dangerous disease.

Very considerable differences of opinion are entertained as to the formidable and frequent disease *diphtheria*. Some maintain its identity with membranous croup, others with scarlatina, while others believe it to be a malady distinct in origin, course, result and treatment from them both. The last mentioned opinion appears to have the most adherents, and the most facts on its side. The differences between the diseases are fully set forth in the table subjoined:—

MEMBRANOUS CROUP.

It is a local complaint. Rarely or never occurs after puberty.

It is not contagious. Type sthenic.

Commences with a cough, catarrh and hoarseness; little or no sore throat and difficulty of swallowing. Cough shrill, metallic; breathing stridulous from the outset.

The membranous affection begins in the larynx and extends to the throat.

Fauces injected but rarely swollen, and generally without exudation.

Exudation never cutaneous.

No swelling of the submaxillary glands.

Epistaxis and albuminuria absent.

Little and often no prostration of the general strength.

Improves under emetics, local counter-irritants, expectorants and depressants.

Is never followed by paralysis.

Rarely fatal. Death from apnoea. Blood not changed. Spleen not affected.

DIPHTHERIA.

Is a general disease, common to all ages.

It is decidedly contagious. Type asthenic.

Commences with a chill, sore throat, difficulty of swallowing; but neither hoarseness nor cough at the outset. Stridulous breathing a late symptom.

The membranous affection begins in the throat and thence extends to the larynx (DA COSTA).

Fauces injected, swollen and presenting exudation.

Exudation often cutaneous.

Submaxillary glands swollen.

Epistaxis and albuminuria frequent.

Considerable, often extreme prostration.

Demands a stimulating and sustaining treatment.

Subsequent paralysis not infrequent.

Frequently fatal. Death usually by asthenia. Blood after death usually fluid and dirty brown. Spleen enlarged and softened (J. W. HOWARD).

TONSILLITIS.

Inflammation of the tonsils assumes two forms, in one of which, the *catarrhal* form, the inflammation extends to the secreting tissues and lining membrane of the crypts, and in the other to the parenchymatous structure of the gland. These two forms differ widely in cause, in symptoms, in treatment and result. Their diagnostic symptoms, as tabulated by Mr. ARTHUR TREHERNE NORTON,* are as follows:—

CATARRHAL TONSILLITIS.

Is a mucous inflammation of three or four days' duration.

Is caused by exposure to draft, damp, cold, etc.

Prostration and often profuse perspiration. Pulse small and quick. Never runs on to abscess.

Both tonsils affected.

Lacunæ filled with masses of morbid secretion resembling ulcers.

No œdema around.

Treatment.—Tonics, stimulants and astringent gargles.

PARENCHYMATOUS TONSILLITIS.

Is a fibrous inflammation of from two to four weeks' duration.

Often caused by neighboring inflammation, cutting wisdom teeth.

High fever, with hot, dry skin. Pulse strong and hard. Commonly forms an abscess.

Rarely both affected.

Often covered with lymph, but no collection of secretion in lacunæ.

Extensive œdema.

Treatment.—Antiphlogistics and depressants, but never gargles, except in the form of warm water.

DISEASES OF THE LUNGS.

In passing from the consideration of the disorders of the upper air-passages to the diseases of the lungs, it is thought advisable to discuss somewhat in detail the several methods of examination of the patient, and to consider systematically the various objective phenomena presented by them as introductory to the study of their alterations, which are characteristic of certain diseases. Palpation, mensuration, auscultation and percussion, therefore, furnish evidence of great clinical importance, which may be considered collectively under the head of physical diagnosis.

The study of Physical Diagnosis necessarily commences with a correct appreciation of the location of organs, their functions and physical charac-

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ters in health; to which must follow a clear understanding of the specific and peculiar alterations which each of these elements undergoes when it becomes a factor in disease. To acquire this, we give on the following pages tabular arrangements of the following subjects:—

I. The Regions of the Chest, their Contents and Normal Signs. II. The Normal Differences between the two Sides of the Chest. III. Methods of Physical Examination. IV. Normal Respiratory Sounds. V. Normal Voice Sounds. VI. Abnormal Resonance on Percussion, and its Causes. VII. Abnormal Intensity, Rhythm and Quality of Respiratory Sounds. VIII. Abnormal (dry) Respiratory Sounds. IX. Abnormal (moist) Respiratory Sounds. X. Abnormal (amphoric) Respiratory Sounds. XI. Abnormal Voice Sounds.

I. THE REGIONS OF THE CHEST.

REGION.	CONTENTS.	RESONANCE ON PERCUSSION IN HEALTH.	AUSCULTATION IN HEALTH.
1. CERVICAL.	Larynx and trachea.		Tracheal breathing and voice.
2. SUPRA-CLAVICULAR.	Apex of lung.	Clear.	Very pure vesicular murmur (scarcely audible): voice scarcely audible.
3. CLAVICULAR.	Clavicles and vesicular structure of lung.	Clear.	Pure vesicular murmur and scarcely audible voice, except at the sternal end, where there are bronchial breathing and bronchophony.
4. SUBCLAVIAN.	Vesicular structure of lung.	Clear.	Pure vesicular murmur and scarcely audible voice. Heart sounds on left side below.
5. MAMMARY.	Vesicular structure of lung. Heart on left side.	Clear on right side. Dull on left in greater part of region.	Pure vesicular murmur above. Heart sounds below on left side, and feeble vesicular murmur on right. Voice scarcely audible.

I. THE REGIONS OF THE CHEST—(Continued).

REGION.	CONTENTS.	RESONANCE ON PERCUSSION IN HEALTH.	AUSCULTATION IN HEALTH.
6. INFRA-MAMMARY.	Anterior portion of base of lung. Stomach below, on left side, liver on right.	Generally tympanitic on left side; dull on right.	Distinct vesicular murmur. Voice scarcely audible.
7. SUPERIOR STERNAL.	Division of trachea, aorta, and great vessels.	Clear.	Bronchial breathing and bronchopony.
8. INFERIOR STERNAL.	Anterior mediastinum above. Stomach below	Clear above; tympanitic below.	Pure vesicular murmur above, becoming feeble below. Voice scarcely audible.
9. AXILLARY.	Vesicular structure of lung.	Clear.	Pure vesicular murmur. Voice scarcely audible.
10. LATERAL.	Vesicular structure of lung.	Clear above; dull below on right side.	Pure vesicular murmur. Voice scarcely audible.
11. SUPRA-SCAPULAR.	Apex of lung.	Clear.	Pure vesicular murmur. Voice scarcely audible.
12. SCAPULAR.	Vesicular structure of lung.	Rather less clear.	Pure vesicular murmur. Voice scarcely audible.
13. INTER-SCAPULAR.	Roots of lung and large bronchi.	Clear.	Bronchial breathing and bronchophony.
14. INFRA-SCAPULAR.	Base of lung.	Clear.	Very pure vesicular murmur. Voice scarcely audible.

II. NORMAL DIFFERENCES BETWEEN THE TWO SIDES OF THE CHEST. (A. H. SMITH.)

	RIGHT SIDE.	LEFT SIDE.
<i>Percussion Resonance.</i>		A little more intense than on the right side.
<i>Vocal Resonance.</i>	Decidedly greater on the right side.	
<i>Bronchial Whisper.</i>	More intense than on the left, and a little lower in pitch.	
<i>Inspiratory Sound.</i>		A little lower on this side, more vesicular in quality, and lower in pitch.
<i>Expiration.</i>	Frequently prolonged in healthy individuals on this side.	

III. METHODS OF PHYSICAL EXAMINATION.

METHODS OF EXAMINATION.	SHOWS.	INSTRUMENTS USED.
1. INSPECTION.	Form, symmetry and capacity of the chest. Local bulging, depression or retraction. Condition of intercostal spaces. Character and frequency of respiratory movements. Comparative size and degree of movement of the two sides. Position and extent of impulse of heart.	
2. PALPATION. (Application of the Hand.)	Comparative movement of the two sides. Vibration communicated to the chest wall by the voice (vocal vibration or vocal fremitus). Force of the heart's impulse. Occasionally certain morbid phenomena, as pleural and pericardial friction, valvular thrill.	
3. MENSURATION. (a) Of Size. (b) Of Movement.	Comparative size of the two sides of the chest. Actual and comparative movement of the chest in respiration.	Graduated tape. Cyrrometer. Dr. Sibson's stethometer. Dr. Quain's " Dr. Edward's chest calipers. Dr. Hutchinson's spirometer.
4. PERCUSSION.	Degree of resonance in various parts of the chest. Extent of cardiac dullness.	Plessor—A hammer tipped with india rubber. The first and second fingers of the right hand will be found to be the best plessor. Pleximeter—A thin plate of ivory or bone. The forefinger of the left hand will be found to be the best pleximeter.
5. AUSCULTATION.	Character of respiratory murmur. Abnormal respiratory sounds. Heart sounds. Abnormal cardiac sounds.	Stethoscope—Made of wood, metal, or vulcanite. Dr. Scott Alison's bin-aural stethoscope.
6. SUCCUSSION.	Presence of air and fluid in pleural cavity.	

PERCUSSION may be—*Immediate*.—Where the chest is struck *directly*, without the interposition of any pleximeter.

(2) *Mediate*.—Where an instrument termed a pleximeter is interposed between the chest and the substance with which the stroke is made. This may be either a thin plate of ivory or bone, or, still better, the first and second fingers of the left hand.

AUSCULTATION may be—*Immediate*.—Where the ear is applied *directly* to the walls of the chest.

(2) *Mediate*.—Where the stethoscope is interposed between the ear and the walls of the chest.

IV. NORMAL RESPIRATORY SOUNDS.

SOUND.	SITUATION WHERE HEARD.
VESICULAR BREATHING.	All over the chest except the upper part of the sternum and the space between the scapulæ, the inspiratory sound being louder, and three or four times longer than the expiratory.
PUERILE BREATHING.	Is the loud vesicular breathing of children, audible over the same parts of the chest as the ordinary vesicular breathing.
BRONCHIAL BREATHING.	Upper part of the sternum and the space between the scapulæ in many healthy persons.
TRACHEAL OR LARYNGEAL } BREATHING.	Over the trachea and larynx.

V. NORMAL VOICE SOUNDS.

SOUND.	SITUATION AND CHARACTER.
ORDINARY VOCAL RESONANCE.	Is the voice-sound heard over the pulmonary regions where vesicular murmur is audible. A muffled, diffused sound; the articulation of the voice is not appreciable.
NATURAL BRONCHIOPHONY.	Heard over the upper part of the sternum, and between the scapulæ in a certain number of healthy persons. A more distinct and concentrated sound than the last variety.

V. NORMAL VOICE SOUNDS—(Continued).

SOUND.	SITUATION AND CHARACTER.
LARYNGOPHONY AND TRACHOPHONY.	Voice-sounds heard over the larynx and trachea. Voice transmitted imperfectly articulated to the ear of the observer, with so much loudness and concentration as even to be painful.

VI. ABNORMAL RESONANCE ON PERCUSSION.

RESONANCE.	CAUSE.	EXAMPLES OF DISEASE.
DIMINISHED in various degrees, or altogether ABSENT.	Deficiency of air, or abnormal deposit, in the lung beneath the part percussed, or solid or liquid matter between the walls of the chest and the lung containing air; or extreme distention of the chest with air.	Pneumonia, first stage. Phthisis; contracted lung, with thickened pleura. Œdema and congestion of lung. Tumors. Collapse of lung. Pneumonia, second and third stages. Intra-thoracic tumors and aneurisms. Effusions into pleural cavity, or its extreme distention by air.
INCREASED.	Air increased in quantity, or air in pleural cavity.	Emphysema. Tubercular cavity, having thin walls, and situated near the surface.
TYMPANIC. AMPHORIC.	A large cavity (or conditions resembling it) with very tense walls, containing air.	Pneumothorax. Extreme emphysema. Upper part of lung compressed by fluid below. Pneumothorax. Cavities.
BOX-LIKE. CRACKED-POT SOUND.	Air expelled from cavity by sudden pressure.	Cavity of considerable size, with large bronchus opening into it, mouth of patient being open during percussion

VII. ABNORMAL INTENSITY, RHYTHM, AND QUALITY OF
RESPIRATORY SOUNDS.

	SOUNDS.	CHIEF CAUSES.	CONDITION OF ORGANS.	EXAMPLES OF DISEASE.
I. Changes in Intensity.	Feeble Breathing.	Air entering the air-cells in diminished quantity and force.	Lung partially solidified either by increase of solid or fluid within it, or by pressure from without; dilatation of the air-vesicles; in some cases lungs not affected.	Incipient phthisis. Bronchitis. Pneumonia, first stage. Tumors. Pleurisy. Emphysema. Pleurodynia.
	Extinct Breathing.	The presence of a non-conducting medium between the lung and the chest-wall, or some impediment to the entrance of air into the bronchi.	Lung solidified by pressure upon its surface; plug of mucus, fibrinous exudation or foreign body in the bronchi, or tumor compressing the bronchi.	Pleuritic effusion. Pneumothorax. Plastic bronchitis. Tumors. Foreign body in bronchus.
	Puerile, or Supplementary Breathing.	Air entering the air-cells with increased rapidity and force.	Healthy, but exaggerated in function.	Disease of opposite lung or of other parts of the same lung. Met with as a normal condition in childhood.
II. Changes in Rhythm.	Interrupted, Jerking, Cogged-wheel Breathing.	Respiratory movements restrained by pain, or mental emotion, or some temporary local obstruction of the air-tubes.	Varies with the disease causing it.	Pleurodynia. Pleurisy. Debility, with palpitation. Hysteria. Incipient phthisis. Spasmodic asthma.
	Prolonged Expiration.	Loss of elasticity in the lung tissue.	Thinning of the walls of the air vesicles, with dilatation and destruction of the alveolar septa.	Emphysema.

ABNORMAL INTENSITY, RHYTHM AND QUALITY OF RESPIRATORY SOUNDS—(Continued).

SOUNDS.	CHIEF CAUSES.	CONDITION OF ORGANS.	EXAMPLES OF DISEASE.
III. Changes in Quality. { EXAGGERATED, COARSE } BREATHING. { BLOWING, TUBULAR, OR BRONCHIAL CAVERNOUS } BREATHING. { AMPHORIC } BREATHING.	Increased friction in the air-cells and smaller bronchial tubes.	{ Lung not solidified (soft sound). Lung solidified or bronchial tubes obstructed (harsh sound).	Generally consistent with health and supplementary. Heard in cases of uremia and other blood poisoned diseases, and in hysteria and nervous diseases. Incipient phthisis.
	Friction of air in the bronchial tubes, or in cavities of the lung.	Condensation of the lung between the chest wall and the larger bronchi or cavities.	Phthisis. Pneumonia. Tumors. Tubercular and other cavities. Pneumothorax.
	Air passing into a large cavity with dense walls.	Cavities with dense walls.	Dilated bronchi. Large cavities.

VIII. ABNORMAL DRY RESPIRATORY SOUNDS.

SOUND.	SITUATION.	CAUSE.	EXAMPLES OF DISEASE.
SIBILUS.	Lesser bronchial tubes.	Vibration of thick mucus attached to the wall of the tube, or contraction of the tube, due either to swelling or spasm; not easily removed by cough.	Bronchitis. Emphysema. Asthma.
RHONCHUS.	Larger bronchial tubes.	Vibration of thick mucus in tubes; generally easily removed by cough.	Bronchitis.

CLICKING OR CRACKLING.

DRY CRACKLING.	Smaller Bronchi.	{ Separation of the adherent walls of the bronchi—the dry tending to pass into the moist variety. Movement of opposed surfaces of pleura roughened by the deposit of lymph or tubercle.	Incipient phthisis.
HUMID CRACKLING. PLEURAL FRICTION SOUND.	Smaller Bronchi. { Layers of pleura.		Phthisis, first stage. Pleurisy before effusion has commenced, or after absorption of the fluid.
CREAKING SOUND.			

IX. ABNORMAL MOIST RESPIRATORY SOUNDS.

SOUND.	SITUATION.	CAUSE.	EXAMPLES OF DISEASE.
CREPITANT RALE. (<i>Fine or pneumonic crepitation</i>).	Air-vesicles.	Opening up of collapsed air-cells, or separation of their adherent walls	Pneumonia in first stage. Edema of lungs. Collapse.
SUBCREPITANT RALE (<i>Medium crepitation</i>).	Smaller bronchial tubes.	Bursting of air bubbles in fluid.	Capillary bronchitis. Phthisical bronchitis. Resolution of pneumonia. Edema of lung. Pulmonary apoplexy.
MUCOUS RALE (<i>Large Crepitation</i> .)	Larger tubes and small or moderate-sized cavities.	Bursting of air-bubbles in fluid.	Phthisis. Bronchitis.
GURGLING OR CAVERNOUS RALE.	Large cavities (or number of small cavities).	Bursting of air-bubbles in fluid.	Hæmoptysis. Phthisis (3d stage). Bronchiectasis.
CHURNING SOUND.	Lung in a state of disorganization.		Abscess of lungs. Gangrene of lung.

X. ABNORMAL AMPHORIC SOUND.

SOUND.	SITUATION.	CAUSE.	EXAMPLES OF DISEASE.
SPLASH OR SUCCUSSION.	Cavity of pleura or large cavity.	Sudden disturbance of air and fluid existing together.	Pneumothorax with effusion. Very large cavity.
BELL SOUND.	Cavity of pleura.	Auscultation of an air-containing cavity while an assistant uses two coins, one as a hammer, the other as a pleximeter.	Pneumothorax.
AMPHORIC ECHO AND METALLIC TINKLING.	Cavities.	Vibration of air in large cavities with tense walls. The former may be produced by râles and rhonchi in the chest, by the voice, and by the act of coughing; the latter requires, in addition, a little fluid at the bottom of the cavity, set in vibration by a momentary impulse, such as the fall of a drop of fluid, and is essentially the echo of a bubble.	Phthisis with very large cavities. Pneumothorax with effusion.

XI. ABNORMAL VOICE SOUNDS.

SOUND OF VOICE.	CHARACTER OF SOUND.	CAUSE.	EXAMPLES OF DISEASE.
FEEBLE OR ABSENT VOCAL RESONANCE.	The obscure humming or buzzing noise heard over the normal chest either very feeble or altogether absent.	Primary bronchus obstructed; non-conducting medium in pleura or rarefied condition of lung.	Tumors compressing or foreign body in bronchus. Pneumothorax. Pleuritic effusion. Emphysema.
EXAGGERATED VOCAL RESONANCE.	Voice sounds unaltered in quality or distribution, but louder and of greater intensity than natural.	Increased resounding or conducting power due to consolidation of the lung, or to the formation of abnormal spaces.	Incipient phthisis, Dilatation of bronchi.
BRONCHOPHONY.	Voice-sounds heard louder, clearer, and more vibratory than natural, but unattended with articulation or tactile sensation to the ear.	Much increased resounding or conducting power.	Cavities due to phthisis or dilatation of the bronchi. Consolidation of the lung resulting from collapse, hæmorrhagic infarctions, pneumonia, phthisis, cancer, etc.
PECTORILOQUY.	Voice-sounds distinctly articulated and concentrated and as if spoken into the end of the stethoscope.	Large abnormal cavity with dense walls.	Phthisis, dilated bronchi, etc.
AMPHORIC RESONANCE OR ECHO.	A ringing metallic sound resembling that produced by speaking into an empty jar.	The voice reverberating in a large cavity with a small aperture.	Phthisis. Pneumothorax.
EGOPHONY.	A tremulous vibratory sound resembling the bleating of a goat, or the nasal Punchinello voice.	A thin layer of fluid in the pleural cavity, with condensed lung behind.	Pleurisy with effusion.

The quality and pitch of the vocal resonance varies greatly in different individuals, and as a diagnostic aid is almost useless in women. Whispering, sometimes, will give more satisfactory results than the loud "one, two, three," that is so constantly heard in our clinical amphitheatres. The use of a small reed whistle, such as is found in childrens' rubber toys, will often give more uniform effects for comparison than the voice.

GENERAL RULES FOR THE DIAGNOSIS OF DISEASES OF THE RESPIRATORY SYSTEM.

The late Dr. JOHN HUGHES BENNETT laid down the following practical rules:—

1. A friction murmur heard over the pulmonary organs indicates a pleuritic exudation.
2. Moist or dry râles, without dullness on percussion, or increased vocal resonance, indicate bronchitis.
3. Dry râles accompanying prolonged expiration, with unusual resonance on percussion, indicate emphysema.
4. A moist râle at the base of the lung, with dullness on percussion, and increased vocal resonance, indicates pneumonia.
5. Harshness of the respiratory murmur, prolonged expiration and increased vocal resonance confined to the apex of the lung, indicates incipient phthisis.
6. Moist râles with dullness on percussion, and increased vocal resonance at the apex of the lung, indicate either advanced phthisis or pneumonia, generally phthisis.
7. Circumscribed bronchophony or pectoriloquy, with cavernous dry or moist râles, indicates a cavity. This may be dependent on tubercular ulceration, a gangrenous abscess, or a bronchial dilatation. The first is generally at the apex, and the last two about the centre of the lung.
8. Total absence of respiration indicates a collection of fluid or of air in the pleural cavity. In the former case there is diffused dullness, and in the latter diffused resonance on percussion.
9. Marked permanent dullness, with increased vocal resonance and diminution or absence of respiration, may depend on a chronic plastic pleurisy, a thoracic aneurism, or a cancerous tumor of the lung.

THE FORMS OF PHTHISIS.

The most recent writers, both in the United States and Europe, are agreed in recognizing three principal varieties of phthisis.* It is of import, both to the prognosis and therapeutics of the case, to distinguish these aspects of the disease; and although in many cases the type is by no means prominently defined, in the majority there is no great difficulty in assigning them to one or another class. The three forms are:—

1. Catarrhal or inflammatory phthisis: “Desquamative pneumonic phthisis.” (BUHL.) Chronic broncho-pneumonia.

2. Fibroid phthisis. Cirrhosis of the lung. Chronic pneumonic phthisis. Bronchial phthisis. Chronic interstitial pneumonia.

3. True primary tuberculosis. Tubercular phthisis. Tubercular pneumonia (DA COSTA).

On the clinical recognition of these three varieties, Dr. ALFRED L. LOOMIS says:—

If a case of phthisis present himself for examination, and it is found that the disease began with the ordinary symptoms of a cold, and that this cold periodically improved and relapsed, but that the cough never left him, but became more pronounced and deepened into what we usually find in advanced phthisis, accompanied with emaciation and occasional hæmoptysis, we are in a position to say that the patient presents the usual characteristics of *catarrhal* phthisis.

If, however, he gives a history of persistent cough for many years, as is found in chronic bronchitis, and eventually furnishes the rational history of advanced phthisis, with the presence of cavities in the lung, we may consider him as having the disease of the *fibrous* form, in which cavities are the result of dilated bronchi.

Finally, if the patient says that an early symptom was emaciation, with impaired digestion, accompanied by a dry, hacking cough, and if, moreover, there was a steady rise in the temperature, we are justified in suspecting the presence of *tubercular* phthisis.

* Dr. A. B. SHEPHERD, *Medical Press and Circular*, July, 1876; A. L. LOOMIS, *New York Medical Journal*, February, 1877; Roswell Park, *Chicago Medical Journal*, September, 1878; Prof. BARTHOLOW, *Medical News and Abstract*, etc.

THE FORMS OF PHTHISIS—CHRONIC CATARRHAL PNEUMONIA.

PERIOD OF INVASION...	<p>Precursory catarrh, sometimes pneumonia, croup, measles, or other inflammatory disease; cough "deepens," proceeding from the trachea to the alveoli and bronchioles, indicated by dark yellow streaks in the sputum. Fever and wasting not marked at outset. Hæmoptysis not common at this period.</p>
TEMPERATURE.....	<p>The hectic is more of a <i>remittent</i> or <i>intermittent</i> than of a continued type; with a range of, say, 1.1° C. between evening and morning temperature; the evening elevation being a constant feature.</p> <p>The fever may present all possible variations in the same individual. A sudden accession may be regarded as an indication of some fresh inflammatory process; <i>e. g.</i>, pleuritis, pneumonia.</p> <p>With marked evening rise of temperature, the rate of respiration does not correspondingly accelerate; hardly ever more than six or eight breaths per minute.</p>
PHYSICAL SIGNS.....	<p>In the first stage, feeble, harsh or puerile respiratory sounds are heard, with all the signs of catarrh at apices and elsewhere.</p> <p>Dullness usually marked; when its area accords with the other signs it is a comparatively favorable feature.</p> <p>The presence of lobular infiltration may, in some cases, cause a hollow or tympanitic note. "Cracked-pot" resonance over a cavity with thin walls.</p> <p>Fremitus is intensified over cavities connecting with bronchi and containing air.</p> <p>Bronchial respiration, bronchophony, and sonorous râles are heard after extensive induration.</p>
GENERAL NUTRITION....	<p>Not impaired in the early stage, but when cavities form, hectic and emaciation set in and we have "pneumonic phthisis."</p> <p>May continue for years, until pneumonic phthisis is developed, when it lasts only a few months.</p>

COMPARISON OF THE FORMS OF PHTHISIS.

FIBROID.

More or less dyspnœa, gradually increasing. Cough, worse in winter, sometimes absent in summer. Hæmoptysis frequent. Pulse slightly rapid, perhaps irregular. Expectoration often profuse, mucous or muco-purulent.

Elevation of temperature and other febrile symptoms very variable, sometimes wholly absent (BRISTOWE). No special type.

Notable dullness on percussion, resonance sometimes tympanitic. Respiration bronchial, or bronchovesicular. Bronchophony and increased vocal resonance. The affected side becomes contracted either entirely or in part.

Bronchial dilatation (fusiform) gives the physical sign of a cavity.

Not incompatible with apparent good health.

Duration indefinite.

TUBERCULAR.

Commences in the alveoli, bronchioles, or connective tissue. Pallor, fever, emaciation and night-sweats early. Cough hoarse and hard, voice hoarse or inaudible, distressing laryngitis. The sputa retain the crude character of the mucous sputa of acute bronchitis. Spleen somewhat enlarged.

The hectic is of a *continued* type; temperature always above normal, but not much higher in the evening than in the morning; *i. e.*, the remissions *not* well marked; moreover it resists treatment.

Signs not well marked, not sufficiently so to account for the symptoms. Solidification not extensive. Expansion unequal.

Cavities form after softening, with destruction of lung tissue.

Health obviously impaired.

Lasts about a year.

THE DIAGNOSIS OF INCIPIENT PHTHISIS.

There is no absolutely sure symptom of phthisis previous to percussion dullness, but a very strong presumption of its approach can be drawn from the presence of the following changes:—

1. *Emaciation.* Where there is progressive emaciation without assignable cause, and especially if the appetite continue good, phthisis should always be suspected. The loss of flesh first shows itself in a retraction of the skin over the cheeks, a thinning of the lips and ears, and a

pinched appearance of the nose. The nostril on the affected side is usually slightly more dilated than the other.

2. *Anæmia*, seen in the bluish hue of the sclerotic, and in the pallor of the cheeks.

3. *Sore throat* and hoarseness. A very early symptom. On examination the pillars of the fauces are found hyperæmic, the throat congested and the bronchial glands enlarged.

4. *Swelling of mucous membrane of larynx*, especially forming a turban-shaped epiglottis, which at the same time assumes a horse-shoe bend; and pyriform enlargement over the arytenoid cartilages (SEILER).*

5. *Depression of the acromial end of the clavicle*, on the affected side. In health the acromial end is slightly higher than the sternal end.

6. *Rheumatoid pains* in the arms coming suddenly at night or in the early morning, not increased on moving the arms.

7. *Pityriasis versicolor*, in the form of pale yellow or reddish spots appearing on the skin of the chest, neck and arms. This is considered by AUFRECHT a very characteristic symptom.

8. In regard to the *breathing*, what is considered as suspicious are weak, jerking, "cogged wheel" or sonorous sounds, rough breathing, a lengthened strong expiration after soft inspiration, especially when in circumscribed regions these sounds differ from those on the other side of the chest. The most appropriate spot to note the duration of expiration is over the larynx or trachea. In proportion as the tubercular deposit is more extended, the expiratory murmur becomes more tubercular in quality and higher in pitch (ARMOR). In normal cases the respiratory sound becomes weaker in the supra-spinous region outward from the vertical column. Dr. HEITLER considers it, therefore, strong evidence of incipient pulmonary phthisis if the respiratory sounds during expiration are more sonorous over these regions than nearer to the vertebral column.†

9. *Unequal expansion of chest* is an early sign of commencing disease of the apex. The expansion is less on the diseased side.

10. *Alterations in temperature curve* frequently take place early. The temperature may be low, but its characteristic range will be: (1) a

* Proceedings Philadelphia Co. Medical Society, vol. ii, p. 101, Philada., 1880.

† DOBELL'S Reports on Diseases of the Chest, 1877.

marked rise after 2 P.M.; (2) a rapid fall after 10 P.M.; (3) minimum about 7 A.M.; (4) recovery to normal about 10 A.M. (C. T. WILLIAMS). Such a curve must always excite grave suspicions.

11. *Rapidity of pulse.* A persistent and sustained increase in the pulse rate, without cardiac disease, is a valuable rational sign, present very early in most cases.

12. The *cough* of incipient phthisis is usually short, hacking, and dry, or with a slight, glairy, mucous expectoration only. From the presence of fragments of the pulmonary fibrous tissue in the sputum, "we are sometimes enabled to suspect the existence of consumption before the physical signs of even its early stages are well defined." (DA COSTA.)*

13. *Hæmoptysis.* The appearance of hæmoptysis is always a serious element of diagnosis. Light, frothy, red blood, rising without apparent exertion, is an indication which, in America at least, has proved of graver meaning the more it has been investigated.† On the other hand, cases will be met with sometimes, in whom there may be considerable hæmoptysis, with marked dullness at the apex, without the significance of tubercle.‡

14. *Clubbing of the finger ends*, when associated with incurvation of the sides and tips of the nails, means obstruction of the subclavian veins, which is one of the earliest effects of tuberculosis; but clubbing without this incurvation is rather against the probability of tubercle (DOBELL).

15. *Amennorrhœa* is, in young females, often one of the earliest signs of phthisis.

16. A *red line* is occasionally noticed on the gums at the base of the teeth.

17. *Arthritis.* M. LAVERAN§ has drawn attention to the occasional occurrence of arthritis as the first symptom of a general tuberculosis.

* To examine sputa for elastic fibres, mix it with a soda solution:—

R. Liquor sodæ,	1 part	
Aquæ destill.,	2 parts.	M.

And boil for four or five minutes. Then dilute with an equal quantity of distilled water, and pour into a flat porcelain vessel. The particles suspended in the water may then be taken out and examined under the microscope. The fibres in this process are brown, slightly reticulated, and a fraction of a millimetre in length (SOKOLOWSKI).

† See second Report of the New York Mutual Life Insurance Company, 1877.

‡ See Prof. DA COSTA, in *Medical and Surgical Reporter*, July 13, 1878.

§ *Le Progres Medical*, October 25, 1876. Quoted by Dr. M. ANDERSON.

DIAGNOSIS BETWEEN INCIPIENT PHTHISIS AND BRONCHITIS.

INCIPIENT PHTHISIS.

1. The cough commences gradually, without marked disturbance or coryza, often preceded by slight loss of flesh and strength.

2. The cough is generally dry and hacking at commencement, followed by the expectoration of a thin mucous fluid, which soon becomes thick and opaque, or is slightly streaked with blood.

3. Examination by the microscope shows portions of lung tissue (yellow elastic fibres) in the sputa.

4. Pain of a wandering character about the chest, especially under the clavicles or between the shoulders.

5. Evening rise of temperature.

6. The morbid physical signs usually confined to upper lobe of one side; are very persistent, and if on both sides at first, apt to subside on one and increase on the other.

7. Family history and general appearance indicate tuberculous cachexia. Most frequent in youth.

8. Essentially chronic.

BRONCHITIS.

1. The cough commences suddenly, and is usually ushered in by feverishness and coryza.

2. The cough is accompanied with expectoration almost from the first; generally abundant; frothy or muco-purulent; not often blood-stained.

3. No evidence of destruction of lung tissue.

4. A feeling of tightness and rawness behind the sternum, aggravated by coughing.

5. Elevation of temperature not particularly marked toward evening.

6. Morbid signs usually predominate in the lower lobes; are on both sides; are of temporary duration, and subside gradually and equally on both sides.

7. No marked hereditary tendency; common at all ages.

8. Has an acute beginning.

While these points of difference between tubercular disease and catarrhal inflammation of the mucous membrane lining the bronchial tubes are in the main reliable, yet it must not be forgotten that chronic bronchitis is often attended by structural changes in the lung, leading in one set of cases to increase of connective tissue, with dilated bronchiæ-fibroid degeneration, chronic broncho-pneumonia—and in another to deposits, chiefly epithelial, in the air cells, producing spots of consolidation.

The general clinical history of phthisis may be summed up in the following brief table:—

PULMONARY PHTHISIS. (CHRONIC TUBERCULAR PNEUMONIA.)

STAGE OF DISEASE.	SYMPTOMS.	PHYSICAL SIGNS.
1st stage (incipient). Stage of invasion.	Cough at first dry, then with expectoration of mucus, frequently streaked or dotted with blood, or with copious hæmoptysis. Dyspnoea. Pains in the various parts of the chest, especially on the affected side. Dislike to fatty articles, and other dyspeptic symptoms; tendency to vomiting after paroxysms of coughing. Night-sweats. Emaciation. In females, disturbance of the catamenial functions. Occasionally hectic.	Diminished movements. Increased vocal fremitus. Loss of percussion resonance, rise in pitch, or a boxy, wooden note beneath the clavicle or in the interscapular region. Feeble, coarse, or interrupted vesicular murmur, with prolonged expiration. Increased vocal resonance. Occasional sibilus or creaking friction sound. Heart sounds abnormally loud over affected side. Subclavian murmur. Puerile (exaggerated) respiration on sound side.
2d stage (confirmed). Stage of deposit.	Cough more severe, with puriform expectoration, of a yellow or greenish hue, and often bloody. Profuse night-sweats and rapidly progressive emaciation. Pinched and anxious expression. Loss of appetite. Thirst. Diarrhœa. Sometimes hectic.	Greater diminution of movement of the affected side, and some amount of flattening. Increased vocal fremitus. Increased dullness, extending downward. Bronchial breathing, mixed with mucous râles or with click at the end of each inspiration. Bronchophony.
3d stage (advanced). Stage of softening and formation of cavities.	Cough rather looser, still with puriform (nummular) expectoration, or attacks of copious hæmoptysis. Extreme emaciation and debility, with or without night-sweats. Voice husky and hollow. Aphthæ on mouth and fauces. Hectic. Clubbed fingers and talon-like nails.	Scarcely any movement of the affected side. Marked flattening. Increased vocal fremitus. Dullness less marked. Box-like resonance or cracked-pot sound. Cavernous breathing, with gurgling and splash on cough. Occasionally metallic sounds. Pectoriloquy.

PHTHISIS—(*Continued*).COMPLICATIONS NOT RESTRICTED TO ANY PARTICULAR STAGE
OF PHTHISIS.

The chief of these are: Affections of the larynx and trachea, especially ulceration; bronchitis, intercurrent pneumonia, or pleurisy; perforation of the pleura, with pneumo-hydrothorax or empyema; enlargement of the external absorbent glands, or of those in the chest and abdomen; tubercular peritonitis; ulceration of the intestines, especially the ileum; fatty or amyloid liver; fistula in ano; various forms of Bright's disease; diabetes; pyelitis; tubercular meningitis, or tubercle in the brain, and thrombosis of the veins of the legs.

POST-MORTEM APPEARANCES.

First stage. Usually most marked at, or even confined to, one apex, where are to be seen gray, semi-transparent nodules, varying in size from a small pin's head to a hemp-seed; the lung-tissue around these nodules may be healthy, but is generally hyperæmic and congested, slightly increased in density. In more advanced cases, in addition to the miliary nodules, there may be small yellow masses, less defined, but larger than the gray variety. Both kinds may either be scattered or several in one group, forming a considerable mass.

Second stage. Commencement of caseation and softening in the centre of the consolidated portions, inflammation of the surrounding parenchyma, together with obliteration of the blood-vessels and formation of cicatricial tissue.

Third stage. Cavities of various sizes and forms, and either single or numerous, generally containing puriform fluid. Ulceration and dilation of the bronchial tubes. Lung indurated and puckered in proportion to chronicity of disease.

ACUTE PHTHISIS, ACUTE MILIARY TUBERCULOSIS,
GALLOPING CONSUMPTION.

The formidable disease known under these names is probably, as M. BOUCHUT remarks, more common than is generally supposed, as it is generally mistaken either for capillary bronchitis or typhoid fever, especially the latter. Its duration is brief, sometimes less than a fortnight (DA COSTA), and its termination almost invariably fatal. Its features

are thus so entirely distinct from the chronic form, from the clinical point of view, as to really constitute it a separate disease.

Its onset is marked by chills and feverishness, nausea, vomiting and diarrhœa. There is a rapid pulse; dyspnœa; slight pain in the chest; cough, usually with profuse expectoration. Great exhaustion, sweats, rapid emaciation, and delirium, soon follow. One or both lungs exhibit unusual dullness, while the auscultatory sounds differ greatly in different cases.

The following are the marked diagnostic features of the disease:—

1. *Facial expression.* The countenance is livid, indicating plainly an impediment to the passage of blood through the lungs. In severe typhoid fever the cheeks are slightly flushed, the facial muscles tremulous, the eyes dull, and the mouth partly opened, presenting an appearance characteristic of the disease.*

2. *The delirium* of acute phthisis is restless and often violent, but the rambling and wild talk is connected usually with things present or near. In typhoid fever the delirium is generally muttering and low; the mind deals with things absent, and the patient "is like a man talking in his dreams" (WATSON).

3. *The tongue* in acute phthisis, at first covered with a white fur, soon becomes red, glassy and dry. In typhoid it usually changes to a brownish hue.

4. *The ophthalmoscope* is a most positive aid to the diagnosis, according to M. BOUCHUT. In all cases of acute, general, miliary tuberculosis, an ophthalmoscopic examination will reveal the presence of tubercular granulations in the choroid,† thus placing the nature of the disease beyond doubt.

5. *Abdominal symptoms.* Diarrhœa and gastric and abdominal pains are often present in acute phthisis; but the red spots of typhoid are not seen.

6. *Chest symptoms.* Dyspnœa is present always, but the orthopnœa of capillary bronchitis is rare (SHAW). The respiration is greatly quickened, and the proportion to the pulse averages 1:3 (WALSHE). The presence of percussion dullness, a sinking in at the upper part of the chest, and the occurrence of hemorrhage, are conclusive evidence of tubercle (DA COSTA).

* L. J. WOOLLEN, *American Practitioner*, July, 1871.

† *Medical Times and Gazette*. January, 1875.

7. The sputum shows the characteristic serous and muco-purulent character, and may contain the elastic fibre of lung tissue.

DIAGNOSIS OF SYPHILITIC PHTHISIS.

The distinctive traits of this form of consumption have lately been separately studied by Dr. MACSWINNEY, of Dublin, and Dr. PENTMALLI, of Naples. Their results are combined in the following scheme:—

1. Absence of hereditary tendency, of a phthisical habitus, and of preceding pulmonary affections.

2. History of syphilitic disease in other organs, and presence of the syphilitic cachexia in its tertiary stage.

3. The disease never begins in the apex, and is limited in its seat, being unilateral and generally posterior (PENTMALLI).

4. Hæmoptysis rare, febrile symptoms absent or slight.

5. Slowness in development, the acuter phthisical symptoms not manifest.

6. Exacerbation of pain during the night.

7. A peculiarly fetid breath.

8. Reference of the feeling of oppression to the larynx rather than to the chest.

9. Failure of ordinary measures, and improvement under specific medication.

BRONCHITIS, ACUTE AND CHRONIC.

In most cases of bronchitis the inflammation is seated in the larger bronchial tubes. There is more or less swelling of their lining mucous membrane, not generally sufficient to prevent a free passage to the breathing air. The characters of the acute and chronic form are set forth in the tables, in the following pages.

There is a variety of chronic bronchitis, in which the material exuded on the surface of the air passages contains a large proportion of a fibrinous constituent which makes it tough and consistent, so that when expelled the substance appears as a perfect cast of the bronchial tube in which it was formed. This is called fibrinous bronchitis, and does not differ in pathology from the ordinary chronic variety, but is less susceptible to treatment. Its diagnosis is made from the appearance of the casts, and needs no further mention here.

ACUTE BRONCHITIS.

	SYMPTOMS.	PHYSICAL SIGNS.
1st or Dry Stage.	Chilliness, followed by frequent pulse and febrile symptoms; pains in limbs. Substernal pain. Hoarse dry cough. Feeling of oppression and tightness about the chest.	Breathing hurried. Rhonchal fremitus may be felt. Resonance on percussion unimpaired. Feeble vesicular murmur, mixed with rhonchus and sibilus. Puerile breathing in unobstructed parts of lung. Vocal resonance not materially altered.
2d or Moist Stage.	Cough, with expectoration of frothy, transparent mucus, mixed with air-bubbles of various sizes, and occasionally tinged or streaked with blood. Urgent dyspnoea, often amounting to orthopnoea. Lividity and febrile symptoms increased. Restlessness at night.	Breathing hurried. Rhonchal fremitus may be felt. Resonance on percussion clear or only very slightly impaired. Feeble vesicular murmur mixed with rhonchus, sibilus and mucous râles. Vocal resonance unaltered.
3d Stage. (Termination favorable.)	Gradual remission of the symptoms. Expectoration becomes thick, greenish, and opaque, and sometimes nummulated.	Less amount of sonoro-sibilant and mucous râles, with return of normal vesicular breathing.
Unfavorable.	Dyspnoea very urgent, signs of impending suffocation. Profuse cold sweats. Sinking, drowsiness and delirium. Less cough, absence of expectoration.	In addition to the signs of the second stage, tracheal râles may be heard.

The *post-mortem* appearances are: Congestion of mucous membrane of bronchial tubes, with some degree of swelling and dryness of surface.

Lungs do not collapse when the chest is opened; nor do sections sink in water. The mucous membrane of the bronchi is red and swollen, and the tubes filled with frothy, adhesive mucus.

CHRONIC BRONCHITIS.

SYMPTOMS.

Two chief forms: the one characterized by the sputa being expectorated with great difficulty, consisting of small, gray, semi-transparent pellets, and tending toward emphysema; in the other the sputa are abundant, muco-purulent, and brought up with ease; dilatation of the bronchi frequently associated with this form. The cough generally comes on at the approach of winter; with the history of former attacks. Dyspnoea; lividity of surface; and in some cases the symptoms resemble those of chronic phthisis, as wasting, with night sweats and hectic.

PHYSICAL SIGNS.

Respiration labored and abdominal. Vocal fremitus not materially altered; rhonchal fremitus can generally be felt. Impairment of resonance or a hyper-resonant note, according as collapse of lung and consolidation, or emphysema predominate, the former most marked at the bases, the latter at the anterior part. Feeble vesicular murmur. Rhonchus, sibilus, and mucous râles. Vocal resonance varies.

The *post-mortem* appearances are:—

Lungs generally much congested, presenting a dark livid hue, with portions collapsed, and others emphysematous. Bronchial tubes frequently dilated. Mucous membrane thickened, uneven, sometimes ulcerated, covered by a thick, puriform secretion, or sparingly coated by a tenacious, glairy, semi-transparent substance.

The principal diseases with which bronchitis may be confounded are pneumonia, pleurisy and phthisis. But each of these is characterized by the presence of definite physical signs, which are not to be found in ordinary bronchitis. For instance, in this disease there is no disparity between the two sides of the chest in the resonance obtained by percussion, nor in vocal resonance, the bronchial whisper and fremitus. The swelling of the bronchial mucous membrane may cause some diminution of the intensity of the vesicular murmur; but as the affection is bilateral and the bronchial tubes on both sides are affected equally, both in degree and extent, there is no appreciable disparity between the two sides. Sometimes temporary weakening or suppression of the murmur may be caused by a plug of mucus, which will be detected on a second examination (FLINT), or by instructing the patient to cough, so as to dislodge it.

CAPILLARY BRONCHITIS.

Acute capillary bronchitis may, however, be taken for some of the forms of pneumonia, and in fact the descriptions of some writers would lead to the belief that they have committed this error. The following distinctions will make the diagnosis easy in most cases:—

CAPILLARY BRONCHITIS.

Commences in the external air passages as a common cold and extends downward.

Always bilateral.

Normal or exaggerated resonance on percussion unless collapse has commenced.

Sub-crepitant râles on both sides of the chest.

Respiration not bronchial, 50 or more; pulse 150 or more.

Muco-purulent expectoration; no plastic lymph.

Dyspnœa intense; cyanosis early. No pain or but little.

Death from asphyxia; mortality more than half.

A disease of children.

PNEUMONIA.

Commences suddenly with a chill, and attacks the lungs directly.

Generally unilateral.

Dullness on percussion more or less extensive at the outset.

Crepitant râle.

Respiration bronchial, 25 to 40 per minute. Pulse 100 to 130.

Rust-colored expectoration; plastic lymph in pulmonary air cells.

Dyspnœa less; cyanosis late if at all. Pain in the side.

Death from asthenia; mortality ten per cent.

A disease of adult life.

PNEUMONIA AND PLEURISY.

Ordinary acute inflammation of the lungs in its early or first stage is well marked by the presence of a moderate or slight dullness on percussion over the affected lobe, and the detection on auscultation of the *crepitant râle*. The latter is indeed not invariably present, but when it is, taken in connection with the symptoms, it is pathognomonic.

Later in the disease the rust-colored expectoration of pneumonia on the one hand, and the physical signs of effused liquid into the pleural cavity in pleurisy on the other hand, offer distinctive features.

The general clinical histories of the diseases are given in the following tables:—

PNEUMONIA.

	SYMPTOMS.	PHYSICAL SIGNS.
1st Stage. (Engorgement.)	Single, severe rigor (or convulsions in children), followed by heat of skin. Increased frequency of pulse. Respiration greatly accelerated, with consequent disturbance of the pulse-respiration ratio. Dyspnoea. Pain in the side, increased by cough or deep inspiration. Cough, at first dry, with rusty sputa about the second or third day. Inability to lie on affected side. Dilated <i>alae nasi</i> . Herpes about lips.	Diminished movement on the affected side. Respiration abdominal. Vocal fremitus normal. Percussion note not materially affected. Feeble vesicular breathing. Fine crepitant r�le, most frequently heard at base of lung and at the end of inspiration.
2d Stage. (Red hepatization.)	Increased distress and dyspnoea. Respiration and speech panting. Cough more urgent, and sputa still rust-colored, extremely viscid, and tenacious. Absence or deficiency of chlorides in the urine.	Very slight movement. Vocal vibrations well marked. Dullness on percussion. Tubular breathing and bronchophony, generally accompanied by some r�les, if at the commencement of the 2d stage of a crepitant character, and afterward of a mucous nature.
3d Stage. <i>a</i> (Gray hepatization.)	Aspect much distressed. Face pale and livid. Great failure of vital powers. Hectic and delirium. Cough continues, and the sputa are either absent, or sometimes they remain rust-colored; at others become like prune-juice, even fetid.	Absolute dullness on percussion. Tubular breathing and bronchophony, frequently with gurgling r�les where the lung is disorganized.
or		
<i>b</i> (Resolution.)	Symptoms yielding about 7th day of disease. Cough less troublesome, expectoration easier. Patient evidently convalescing.	Dullness diminishing by absorption. Bronchovesicular breathing with <i>crepitant redux</i> r�les yielding to normal vesicular murmur, and percussion-note.

POST-MORTEM APPEARANCES.—*Lungs*: 1st stage. Engorged with frothy and bloody serum. Dark-red color externally, and on section. Crepitating less, and heavier, than sound lung, but still floating in water. Pulmonary tissue slightly softened.

2d. Red externally, red or mottled and granular on cut surface, and of liver-like solidity. Easily torn, and with fluid exuding on pressure less abundant than in first stage, but thicker, and towards the end of this stage becoming purulent. Not crepitating, and sinking in water.

3d. Reddish-yellow or gray. More rotten and friable. Purulent fluid exudes from the cut surface; and, on pressure, the whole lung may be reduced to a pulp-like mass.

PLEURISY.

The symptoms of pleurisy are attributed to inflammation of the serous covering of the lungs, and is to be distinguished from passive effusion into the pleural sac, which is known as *hydrothorax*, which is readily recognized by the following points of difference:—

PLEURISY.

Due to inflammation (active).

Has an acute beginning, accompanied by stitch in the side, cough, constitutional disturbance, pyrexia, etc.

May be traced to traumatic causes, or to exposure to wet and cold; or may complicate zymotic diseases.

One side only affected, as a rule.

Runs its course in a few days, terminating in chronic pleuritic effusion, or in absorption of fluid.

No complications generally.

HYDROTHORAX.

Due to transudation (passive).

Effusion takes place insidiously, without local or general symptoms, beyond those caused mechanically by pressure on the thoracic viscera.

Due to blood disorder, accompanying renal disease, or more rarely to obstruction to circulation by morbid growths, or valvular disease of heart.

May be bilateral.

Remains stationary for months, or may slowly increase.

Accompanied by albuminuria, heart disease, and dropsy elsewhere in the body.

PLEURISY.

	SYMPTOMS.	PHYSICAL SIGNS.
PLEURISY : 1st Stage, or Stage of Hy- peræmia.	Rigors, or more fre- quently mere chilliness. Sharp, stabbing pain in the side, increased by deep inspiration or cough, accompanied by more or less ten- derness on pressure. Breathing short and hurried. Respiration chiefly abdominal, with inability to lie on the affected side. Short, dry cough, or none at all. Pulse full and bounding. Febrile symptoms.	Diminished movement on the af- fected side. Friction fremitus may sometimes be felt. Percus- sion sound not materially alter- ed. Vesicular murmur feeble and jerking in rhythm. To- and-fro-friction sound.
2d Stage, or Stage of Ef- fusion.	Cough, dyspnoea, sense of weight and fullness of the affected side. Fe- brile symptoms less marked. Patient lies toward, not on, the af- fected side. Complex- ion inclined to be dusky.	Almost total absence of move- ment of the affected side, which is unduly prominent, the inter- costal spaces being obliterated or even bulging. Integuments occasionally œdematous. Vocal vibrations absent. Complete dullness on percussion, most marked in the dependent por- tions of the chest, and some- times altered by change of pos- ture. Heart pushed over to sound side, and diaphragm pushed down, so that the liver and stomach descend lower into the abdomen than in health. Vesicular murmur almost, or quite, absent. Frequently bron- chial breathing near the spine. Voice sounds absent or feeble, except when the layer of fluid is thin, and then there may be ægophony. No friction sound. Puerile breathing in sound lung.

PLEURISY—(*Continued*).

	SYMPTOMS.	PHYSICAL SIGNS.
3d Stage (Resolution after Effusion).	Gradual diminution of the cough, dyspnœa, and other symptoms. Returning ability of the patient to lie on the sound side. Gradual return of displaced organs to their normal position.	The movement of the chest gradually increases. Return of vocal vibration and friction fremitus. The dullness on percussion diminishes from above downward, but the resonance generally remains box-like for a considerable period. Gradual restoration of the vesicular murmur, at first weak and distant, then somewhat harsh, and subsequently of a normal character. Reappearance of the friction sound for a time. Pseudo râles occasionally to be heard. Ægophony sometimes to be heard, more often bronchophony, and ultimately normal vocal resonance.

POST-MORTEM APPEARANCES.—1st stage. Pleura opaque and drier than natural, roughened and highly vascular, and presenting a close network of blood-vessels with ecchymoses.

2d. Fluid, either serous or purulent, mixed with shreds of creamy lymph, in the cavity of the pleura. Lung pushed upward and backward towards the spine, its surface coated with a layer of lymph of the same kind as that mixed with the fluid. The lung collapsed and carnified.

3d. If the effusion has been of long duration the lung remains carnified and bound down by adhesions, and the chest-wall undergoes retraction or depression, the ribs overlap, and there is more or less lateral curvature of the dorsal spine toward the diseased, and of the lumbar toward the healthy, side.

The diagnosis can be made by drawing off part of the fluid from the chest by means of a hypodermic syringe. If purulent, it should be evacuated, or it may lead to amyloid change in the liver and kidneys.

The effusion may become purulent at first; there are no reliable means of recognizing the exact time when this occurs, but later it assumes all the characteristics of empyema, as follows:—

	SYMPTOMS.	PHYSICAL SIGNS.
Empyema.	More decided febrile disturbance of a hectic type, night sweats. Morning remission and evening exacerbations. Face puffy and semi-transparent. Clubbing of the finger ends. If pointing inwardly, abundant purulent sputa.	The physical signs are those of the stage of effusion. The diagnosis is often to be determined only with the aid of the aspirator, or the hypodermic needle.

DIAGNOSIS BETWEEN PLEURISY WITH EFFUSION AND PNEUMONIC CONSOLIDATION.

PLEURISY.

1. Begins with chilliness or several slight rigors.
2. Sharp, catching, stitch-like pain in the side.
3. Cough, dry or with little mucous expectoration, very painful, and repressed by the patient.
4. Pyrexia is not great and the skin may be moist.
5. Excretion of chlorides not affected.
6. Pulse-respiration ratio not affected, except in excessive effusion.
7. Affected side rounded; displacement of heart.
8. Feeble or absent vocal fremitus.
9. Absolute dullness on percussion, transgressing the median line in front.
10. Feeble or absent vesicular breathing; bronchial breathing at the root of the lung.
11. Vocal resonance absent, sometimes egophonic.

PNEUMONIA.

1. Begins with a severe and protracted rigor.
 1. Pain does not catch the breath; is more of a dull character.
 3. Cough frequent and severe, with rusty, viscid expectoration.
 4. Great febrile disturbance, skin hot and pungent.
 5. Diminution or absence of chlorides in the urine.
 6. Pulse-respiration ratio may fall to 2:1.
 7. No alteration in shape of the chest or of the intercostal spaces; heart not displaced.
 8. Vocal fremitus usually much intensified.
 9. Less intense dullness, not transgressing the median line.
 10. Marked tubular breathing, often of a metallic character.
 11. Loud bronchophony.

DIAGNOSIS BETWEEN PNEUMONIA AND PULMONARY APOPLEXY. (PULMONARY EMBOLISM.)

PULMONARY APOPLEXY.

Nearly always associated with heart disease or pyæmia.

Onset sudden. Fever absent except in pyæmia. Pulse irregular and intermittent.

Expectoration blackish, with small dark clots.

Dyspnœa severe at first, afterwards diminishing.

Dullness distinctly circumscribed; respiration bronchial, with moist râle.

A peculiar acid and alliaceous odor to the breath "like the smell of tincture of horse radish" (GUE-NEAU DE MUSSY).

PNEUMONIA.

Generally an independent disease in robust individuals.

Onset with malaise and chill. Fever. Pulse rapid.

Expectoration rust-colored; no clots.

Dyspnœa gradually grows in intensity.

Dullness larger and extending. Crepitant râle. Tubular breathing, bronchophony.

Not present.

THROMBOSIS OF PULMONARY ARTERY.

The symptoms of an immediately fatal attack are: Sudden extreme dyspnœa with open tubes, cough and thoracic pain, lividity or pallor, rapidly failing pulse, cold sweats, intense anxiety, and attacks of fainting or unconsciousness, with or without spasms.

In the diagnosis, the suddenness of the conditions being of the chief interest, all those forms of suffocation requiring time for their production may be disregarded, and there remain:—

1. Closure of the greater air passages or of a large number of small ones, from without or from within.
2. Nervous lesions, particularly intra-cranial, affecting respiration and circulation.
3. Obstruction to the pulmonary circulation from emboli, of blood and air particularly, fat being more gradual in its effects.

Physical and rational evidence of open air passages eliminate the first series. In intra-cranial origins of suffocation the predominant early

symptoms are those of cerebral anæmia, namely, pallor, relaxed muscles, disturbed hearing and vision, contracted pupils, fainting and convulsions. Dyspnœa may sometimes precede these symptoms, but it is not of so severe a character as in the other series.

In favor of the third is the history of an antecedent thrombus, or of a disease of the heart likely to be associated with thrombosis or of septicæmia.

ASTHMA.

SYMPTOMS.

There may be premonitory symptoms, such as gradually increasing dyspnœa or the passing of a large quantity of limpid urine; but the attacks usually come on suddenly at an early hour in the morning; the patient awakes in a start, with a sensation of suffocation and oppressiveness of the chest; he either sits upright in bed, or sometimes stands holding on to a piece of furniture, so as to bring into play the accessory muscles of respiration. Countenance pale and anxious; in bad cases cyanotic. Skin covered with sweat; extremities cold. Pulse frequently feeble. The attacks generally terminate with the expulsion of tough, ashy gray pellets of mucus.

PHYSICAL SIGNS.

Chest generally distended, though there is scarcely any expansive movement. Recession of the intercostal spaces, supra-sternal and supra-clavicular fosse and epigastrium during inspiration, which is short and jerky, while expiration is prolonged and wheezing. Vocal vibration not markedly affected. Rhonchal fremitus may be felt. Resonance on percussion increased all over the chest. Almost complete absence of vesicular murmur. Every variety and kind of sibilus and rhonchus, whistling, squeaking, cooing, snoring sounds, and occasionally mucous râles towards the termination.

POST-MORTEM APPEARANCES.—The appearances found after death are principally the result of chronic bronchitis and emphysema, with dilatation of the right side of the heart.

PNEUMOTHORAX.

This condition is generally found with serous effusions—pneumo-hydrothorax; but occasionally presents itself as an independent affection. The characteristics of the two forms are as follows:—

	PNEUMOTHORAX.	PNEUMO-HYDROTHORAX.
Symptoms.	Generally sharp, stabbing pain, with the sensation of something having given way. Urgent dyspnoea and evidences of shock. More or less cyanosis. Posture assumed by patient varies. Pulse frequent, weak and small. Respiration may be 40 to 60 in the minute. Troublesome cough without expectoration. In some cases of phthisis, or where there are extensive pleural adhesions, pneumothorax may come on quite imperceptibly.	Symptoms the same, except that the cough is usually attended by fetid, puriform expectoration. The patient lies on or toward the affected side.
Physical Signs.	Dilatation of the affected side, with obliteration or bulging of the intercostal spaces. Movements of respiration diminished or absent. Increased elasticity of the walls of the chest. Feeble or absent vocal fremitus. Clear tympanitic resonance on percussion. If the amount of air is extreme there may be high-pitched dullness. No true vesicular murmur; bronchial breathing may be heard along the spine. Amphoric sounds, with inspiration, voice, and cough, also a metallic echo; the bell sound may be elicited. The neighboring viscera are displaced to a variable degree.	Same as in true pneumothorax, except that percussion is dull in the lower part of the chest, and tympanitic above the level of the fluid. Metallic tinkling and splashing sounds on succussion are also frequently heard.

PNEUMOTHORAX—(*Continued*).

	PNEUMOTHORAX.	PNEUMO-HYDROTHORAX.
Post-mortem appearance.	Lung collapsed, lying near vertebral column, unless bound down by old adhesions to some other part of the chest wall. The gas is composed chiefly of carbonic acid and nitrogen, containing but little oxygen, and occasionally some sulphureted hydrogen.	Lung collapsed. Air, mixed with fluid, in pleural cavity. Mostly arises as a termination to phthisis, a superficial cavity becoming ruptured. May occur in pneumonia, emphysema, or gangrene of the lung, and more rarely in other diseases.

EMPHYSEMA.

This affection presents itself in two forms, the vesicular and the interlobular, which are distinguished as follows:—

	VESICULAR EMPHYSEMA.	INTERLOBULAR EMPHYSEMA.
Symptoms.	Habitual shortness of breath, with occasional paroxysms of urgent dyspnoea, most frequently supervening on catarrh. Cough, with or without expectoration of thin, transparent, frothy mucus. In the last stage of the disease there are symptoms due to interference with the circulation, as palpitation, cyanosis, general dropsy, and congestion of the abdominal viscera. The disorder is essentially chronic in its course, and may progress so slowly as not to materially shorten life. It generally occurs in persons who are otherwise vigorous, and is hence supposed to grant immunity from consumption.	Urgent dyspnoea and oppression, generally occurring suddenly after some violent effort, the subcutaneous areolar tissue frequently becoming oedematous.

EMPHYSEMA—(Continued).

	VESICULAR EMPHYSEMA.	INTERLOBULAR EMPHYSEMA.
Physical Signs.	Chest "barrel shaped" and almost circular. Sternum projecting forward. Scapulae and clavicles raised and ill-defined. Ribs more horizontal and intercostal spaces widened. Respiration abdominal. Movement of chest much diminished. Heart beating in the epigastric region. Resonance on percussion greatly increased or tympanitic. Feeble inspiration, prolonged expiration, the former wheezing, the latter generally with rhonchus or sibilus. Vocal fremitus and resonance usually deficient.	Percussion tympanitic over the affected part.
Post-mortem appearance.	Lung does not collapse as usual when the chest is opened, but, on the contrary, may rise up and bulge out of its cavity. It is pale and anæmic, and does not crepitate when pressed, but feels soft and downy, and is drier than ordinary. The air cells are dilated, or several have become one cavity from the rupture of the septa between them. Cells vary from the size of a millet-seed to that of a swan-shot, or larger.	Bead-like bubbles of air seen through the pleura, or partitions between the lobules much widened. Sometimes air is found beneath the areolar tissue of the neck.

CANCER OF THE LUNG.

The principle obstacle in recognizing this disease is the liability to confound it when primary and unilateral (as it usually is when primary)

with phthisis. Similar cough, emaciation, hæmoptysis, night sweats, etc., occur in both. The points of difference are:—

PULMONARY CANCER.

PHTHISIS.

Sides of chest more markedly asymmetrical; the tumor may bulge through the intercostal spaces.

Percussion dullness very great; may extend beyond median line.

Frequent changes in the signs of auscultation, râles, bruits, etc.

Hæmoptoic sputa, "resembling currant jelly."

Pain constant, severe, lancinating.

Cancerous cachexia, tinge of skin, etc.

One side may be sunken; never bulging.

Percussion dullness moderate; never extends beyond median line.

Changes much more gradual.

Sputa never present this appearance.

Pain variable, intermittent.

Absent.

Pulmonary cancer is sometimes so masked that its diagnosis requires the closest attention. It may be present without the characteristic sputa, without cachexia, and even without pain at cancerous spot.* Such instances are, of course, very rare.

It is liable to be mistaken for *chronic pleurisy*, or *vice versa*. The distinguishing features are, that in cancer there is an absence of the complete consolidation of chronic pleurisy; the consolidation of the latter is at the lower portion of the lung; the expectoration of cancer is quite different from that of pleurisy and bronchitis; and the previous history, both of the individual and his family, in cancer, points to this disease, while chronic pleurisy has as an antecedent an acute attack.

The deposits of gummatous nodules in the lungs consequent on *secondary syphilis*, together with the cachexia attendant on that disease, may simulate a cancerous deposit. The history of the case, the presence of syphilitic signs in other organs and tissues, and the fact that cancers tend to spread and infiltrate the surrounding tissue, while the syphilitic nodule remains isolated and circumscribed, are the distinctive points.

* See case recorded in the *Boston Medical and Surgical Journal*, January, 1876.

CHAPTER III.

DISEASES OF THE CIRCULATORY APPARATUS.

CONTENTS.—*The Præcordial Region—Normal Sounds and Impulse of the Heart—Endocardial Murmurs—General Rules for the Diagnosis of Heart Diseases—Constitutional Symptoms of Heart Disease—Clubbing of the Fingers—Differential Signs Between Anæmic and Organic Blood Murmurs—Pain at and near the Heart—Aphorisms Regarding Angina Pectoris—Differential Signs of Aortic Obstruction and Aortic Incompetency; of Mitral Obstruction and Mitral Incompetency; of Pulmonary Obstruction and Tricuspid Regurgitation—Pericarditis—Diagnosis Between Acute Endocardial and Exocardial Sounds; Between Cardiac Dilatation and Pericarditis with Effusion; Between Simple Hypertrophy, Hypertrophy with Dilatation, and Simple Dilatation—Fatty Degeneration of the Heart.*

The anatomical positions of the several parts of the heart are as follows:—

RELATIONS OF THE HEART TO THE PRÆCORDIAL REGION.

REGION.	SITUATION.
APEX OF HEART.....	Between fifth and sixth ribs on left side, about two inches below the nipple and one inch on its sternal side.
BASE OF HEART.....	On a level with the third costal cartilages.
TRICUSPID ORIFICE.....	Extends from the junction of the fourth left costal cartilage with the sternum, behind that bone to the articulation of it with the sixth right cartilage.
MITRAL ORIFICE.....	To the left of the tricuspid valves, immediately behind the fourth costal cartilage; but less superficially placed than the tricuspid.

THE PRÆCORDIAL REGION—(*Continued*).

REGION.	SITUATION.
PULMONARY ORIFICE.....	Immediately behind the left border of the sternum at the junction of the third costal cartilage with that bone.
AORTIC ORIFICE.....	About half an inch lower than and to the right of the pulmonary orifice, behind the sternum, on a level with the third interspace.

Let it be remembered that the tricuspid orifice is the most superficial, then the pulmonary, next the aortic, and deepest of all is the mitral orifice. Ranged from above downward, the pulmonary orifice comes first, then the aortic, then the mitral, and lastly the tricuspid.

PHYSICAL EXAMINATION OF PRÆCORDIAL REGION.

EXAMINATION BY	SHOWS.
INSPECTION	Form of chest. Point at which the apex of the heart strikes the wall of the chest. Regularity of impulse, and extent over which it is perceptible.
PALPATION.....	Force and regularity of impulse. Presence or absence of purring tremor or of friction fremitus.
PERCUSSION.....	Extent and intensity of præcordial dullness.
AUSCULTATION.....	Character of rhythm. Character of sounds, normal or abnormal,

THE AREA OF SUPERFICIAL CARDIAC DULLNESS

Is roughly triangular in shape, the right side of the triangle being the mid-sternal line from the level of the fourth chondro-sternal articulation downward; the hypotenuse being a line drawn from the same articulation to a point immediately above the apex-beat; the base being a line drawn from immediately below the apex-beat to the point of meeting between the upper limit of liver dullness and the mid-sternal line (Dr. GEE).

NORMAL SOUNDS AND IMPULSE OF HEART.

SOUND.	CHARACTER.	POINT OF GREATER INTENSITY.	CAUSE.	TIME.	CONDITION OF CIRCULATION.
FIRST SOUND. (Systolic).	Dull and prolonged.	Fourth and fifth intercostal spaces just within left nippleline.	Closure of auriculo-ventricular valves, and, perhaps, muscular contraction of the ventricles themselves; also impact of apex against the chest-wall, and vibration of papillary muscles and chorda tendineæ.	$\frac{1}{10}$	Contraction of ventricles, following that of auricles. Closure of auriculo-ventricular valves, open aortic and pulmonary valves; propulsion of blood into the arteries. Impulse of the heart immediately followed by pulse at the wrist.
FIRST PAUSE.	$\frac{1}{10}$	Auricles dilating.
SECOND SOUND (Diastolic).	Short and clear.	Base of heart, opposite the third right costal cartilage.	Sudden closure of the aortic and pulmonary valves.	$\frac{2}{10}$	Filling of both auricles and ventricles. Closure of arterial valves, opening of auriculo-ventricular valves.
SECOND PAUSE	$\frac{3}{10}$	Complete distention of auricles, followed by their contraction, and distention of ventricles. Auriculo-ventricular valves open, arterial valves closed.
IMPULSE.	..	Between fifth and sixth ribs on left side, about one and a half or two inches below the nipple, and one inch to its inner side.	In part due to the tilting upward of the apex, but chiefly to the recoil of heart and change in shape, for during the systole it becomes harder and more globular.		

ENDOCARDIAL MURMURS.

TIME.		SITUATION.	ORIFICE.	NATURE.
SYSTOLIC	1 .	Basic (right).	Aortic.	Obstructive.
	2 .	“ (left).	Pulmonary.	“
	3 .	Apical.	Mitral.	Regurgitant.
	4 .	“	Tricuspid.	“
DIASTOLIC	1 .	Basic.	Aortic.	“
PRESYSTOLIC	1 .	Apical.	Mitral.	Obstructive.

Pulmonary regurgitant murmur (diastolic) and tricuspid obstructive murmur (presystolic) are very rarely met with clinically, and for all practical purposes they may be disregarded.

The most frequent combinations of these murmurs are:—

1. Combined aortic obstruction with regurgitation.
2. Mitral obstruction and regurgitation.
3. Various combinations of the two preceding forms, the aortic and mitral valves being both diseased.
4. Mitral obstruction with dilated right ventricle, and consequently tricuspid regurgitation (Dr. AITKEN).

Order of frequency of endocardial murmurs, commencing with the most common:—

- | | | |
|-------------------------|---|----------------------------|
| 1. Mitral regurgitant. | } | 5. Tricuspid regurgitant. |
| 2. Aortic constrictive. | | 6. Pulmonary constrictive. |
| 3. Aortic regurgitant. | | 7. Pulmonary regurgitant. |
| 4. Mitral constrictive. | | 8. Tricuspid constrictive. |

Order of relative gravity as “estimated not only by their ultimate lethal tendency, but by the amount of complicated miseries they inflict.”—
Dr. WALSHE.

- | | | |
|--------------------------------|---|----------------------------|
| 1. Tricuspid regurgitation. | } | 3. Aortic regurgitation. |
| 2. Mitral constriction and re- | | 4. Pulmonary constriction. |
| gurgitation. | | 5. Aortic constriction. |

GENERAL RULES FOR THE DIAGNOSIS OF HEART DISEASE.

Dr. JOHN HUGHES BENNETT* gives the following rules:—

1. In health the cardiac dullness, on percussion, measures, immediately

* “Lectures on the Principles and Practice of Medicine.”

below the nipple, two inches across, and the extent of dullness beyond this measurement commonly indicates either the increased size of the organ or undue distention of the pericardium.

2. In health the apex of the heart may be felt and seen to strike the chest between the fifth and sixth ribs, a little below and a little to the inside of the left nipple. Any variations that may exist in the position of the apex are indications of disease either of the heart itself or of the parts around it.

3. A friction murmur synchronous with the heart's movements indicates pericardial or ex-pericardial exudation.

4. A bellows murmur with the first sound heard loudest over the apex indicates mitral insufficiency.

5. A bellows murmur with the second sound heard loudest at the base indicates aortic insufficiency.

6. A bellows murmur with the second sound heard at the apex is rare. It indicates—1st, aortic disease, the murmur being propagated downward to the apex; or 2d, roughened auricular surface of the mitral valves; or 3d, mitral obstruction.

7. A murmur with the first sound loudest at the base, and propagated in the direction of the large arteries, is more common. It indicates—1st, an altered condition of the blood, as in anæmia; or 2d, dilatation or disease of the aorta itself; or 3d, stricture of the aortic orifice, or disease of the aortic valve.

8. Hypertrophy of the heart may exist independent of any valvular lesion, but this is rare.

9. The pulse as a general rule is soft and irregular in mitral disease, but hard, jerking, or regular in aortic disease.

10. Cerebral symptoms are more marked in aortic disease; pulmonary symptoms in mitral disease.

Various constitutional symptoms should, in default of other obvious causation, lead to the suspicion of disease of the heart. These are mainly:

1. *Symptoms referred to the circulation.* Violent, continued *pulsation* may arise from cardiac hypertrophy, and especially aortic regurgitation. *Cyanosis*, blueness of the lips, coldness of the finger tips, etc., are common in many cardiac cases. *Dropsy* is a late and dangerous symptom.

2. *Symptoms referred to the lungs.* These are frequent cardiac complications, especially dyspnoea, orthopnoea and cough.

3. *Symptoms referred to the brain.* Vertigo, languor, chorea, epilepsy, apoplexy and paralysis may all be brought about by heart disease. In sudden cerebral attacks in patients suffering with valvular disease, *embolism* is often at work.

4. *Stomach symptoms.* Dyspepsia and hemorrhoids may find their origin in cardiac lesions.

5. *Throat symptoms.* Pain in the throat is complained of in angina; hoarseness and aphonia sometimes signify pericarditis.

6. *Renal symptoms* may follow heart disease. In all cases of cardiac disease the urine should be tested for albumen, as this condition may excite cardiac symptoms.*

CLUBBING OF THE FINGER ENDS IN CHRONIC HEART DISEASE AND PHTHISIS.

The following aphorisms on this point are laid down by Dr. HORACE DOBELL:†—

Aphorism I. Clubbing of the finger ends on one or both sides of the body, with or without incurvations of the nails, may occur whenever the return of blood by one or both subclavian veins is seriously obstructed for a considerable length of time.

II. Symmetrical clubbing of the finger ends of both hands without incurvation of the sides and tips of the nails, is presumptive evidence of the existence of heart disease.

III. Clubbing of the finger ends without incurvature of the sides and tips of the nails is presumptive evidence *against* the existence of phthisis.

IV. Symmetrical clubbing of the finger-ends conjoined with incurvation of the sides and tips of the nails, is a sign that obstruction of the return blood by the subclavian veins and wasting of adipose tissue have co-existed.

* See also paper by Prof. DA COSTA and Dr. LONGSTRETH in *Am. Journal for Med. Sciences*, for July, 1880, for pathological relationship of heart disease and chronic kidney disorder.

† "Affections of the Heart." London, 1876.

DIFFERENTIAL SIGNS BETWEEN ANÆMIC AND ORGANIC CARDIAC SOUNDS.

ANÆMIC SOUNDS.

First sound heard over the right ventricle is distinct, second ringing; a *soft* murmur is heard over the left ventricle.

Sounds vary in character.

Sounds increase in intensity in following the aorta.

Pressure with the stethoscope increases or develops the sound.

Bruit du diable, a continuous musical hum, can be heard in the hollow above the right clavicle.

Co-existence of pallor or anæmia; amenorrhœa; leucorrhœa; nervous exhaustion; chorea; renal disease; phthisis.

ORGANIC SOUNDS.

Murmur generally *harsh* and blowing, and takes the place of one or both sounds of the heart. It may be distinctly located at apex or base.

Sound the same after several examinations.

Sounds diminish in intensity in receding from the heart.

Not affected by pressure.

Not present.

Co-existence of alteration in size of the heart; other organic signs; history of rheumatism.

PAIN AT THE HEART.

Pain is by no means a common symptom of heart disease. Not more than one in a dozen cases of chronic organic cardiac disease complain of pain at all.* In acute cardiac affections it is more frequent. In most cases of alleged pain at the heart, it is found on examination to proceed from dyspepsia, muscular rheumatism, intercostal neuralgia, enlarged spleen, or the like.

APHORISMS OF DR. HORACE DOBELL.†

I. Pain in the region of the heart and down the left arm does not necessarily indicate heart disease.

II. The conjunction of pain in the region of the heart and pain in the left arm may be a most important symptom of heart disease, and is never to be disregarded.

* SANSOM, "Diagnosis of Diseases of the Heart," p. 3.

† "On Affections of the Heart." London, 1876.

III. If pain is excited by exercise taken when the stomach is not distended with food or gas, and especially if it comes on quickly and increases steadily in severity with the continuance of exercise, it is almost certain there is some serious disease of the circulatory organs.

IV. When it is found that flatulence or a full meal embarrasses the heart painfully, a careful investigation should be made into the condition both of the organ itself, and of the blood.

V. Important heart disease may exist, and yet pain at the heart and in its neighborhood be absent.

VI. The appalling import of *pain in the throat* in heart disease increases in proportion as the period of its onset deviates from the following order of severity:—

1. Pain under the left breast.
2. Pain extending from under the left breast to mid-sternum.
3. Pain extending from mid-sternum toward the left shoulder.
4. Pain extending from the left shoulder down the left arm.
5. Pain extending from mid-sternum toward the right shoulder.
6. Pain extending from the left shoulder down the right arm.
7. Pain extending up the sternum toward the region of the throat.
8. Pain in the thyroid cartilage.

When this order of advance is maintained as the exciting cause is continued, pain in the throat expresses the degree of dangerous persistence in the exciting cause of heart distress, rather than the degree of danger in the disease itself.

VII. In proportion as the *right* side of the chest and *right* arm take precedence in the order of extension of pain at the heart and its neighborhood, the probability increases that the aorta is more diseased than the heart.

VIII. The volume of blood and other conditions being normal, the facility with which the pulse at the wrist is stopped by inspiration measures the *loss of heart power*.

ANGINA PECTORIS.

This disease is usually quoted as one typically connected with pain at the heart. This is by no means the case, as in many instances there is merely a sense of præcordial distress, but no actual pain (SANSOM). The diagnostic characters are:—

1. The attacks are paroxysmal, coming on at varying intervals and duration (from a minute to an hour), without assignable cause.

2. There is always a *sensation of coldness* experienced, and often a cold sweat.

3. The heart's action is not increased, and may be diminished.

4. The chest is fixed and the breathing slow.

5. The pain, when present, may be of great intensity, of a cold, sickening character, directly referred to the heart, with an accompanying sense of impending dissolution.

Though at first a neurosis, probably of the sympathetic (cardiac ganglia) angina pectoris, is generally associated with some progressive degeneration of the muscular texture of the heart.

DIFFERENTIAL SIGNS OF AORTIC OBSTRUCTION AND AORTIC INCOMPETENCY.

AORTIC OBSTRUCTION.		AORTIC INCOMPETENCY.
Hypertrophy of left ventricle.	Effect on Heart.	Hypertrophy and dilatation of left ventricle.
To left.	Apex Displaced.	Downward and to left.
To left greatly.	Cardiac Dullness Increased.	Downward and to left, more increased than in obstruction.
Forceful.	Character of Impulse.	More forcible than in obstruction, and over wider area.
To left of sternum.	Impulse Felt.	To left of sternum.
Onward, ventriculo-aortic.	Murmur, its Direction.	Backward; aortic-ventricular.
Systolic; loudest at beginning of systole.	Time of Murmur.	Diastolic; post-systolic; loudest at beginning of diastole.
Right border of sternum, in second intercostal space.	Point of Greatest Intensity.	Right border of sternum opposite third intercostal space.
Upward to right sternoclavicular articulation.	Direction in which Propagated.	Downward along sternum and toward apex.

DIFFERENTIAL SIGNS OF AORTIC OBSTRUCTION AND AORTIC INCOMPETENCY—(Continued).

AORTIC OBSTRUCTION.		AORTIC INCOMPETENCY.
Loud, harsh, or blowing.	Character of Sound (very uncertain and of little value for diagnosis).	Of higher pitch than in obstruction, and loudness decreases rapidly from commencement.
Replaces first at base.	Relation to Normal Heart Sounds.	Replaces second at base, and occupies more or less of the pause.
Depends on condition of valves, but aortic second sound generally feeble.	Effect on Second Sound.	Apparent intensification of pulmonary second.
Systolic; in second right intercostal space.	Thrill.	Down sternum; diastolic.
	Effects on Pulse.	Visible pulsation in arteries (locomotive pulse).
Normal, or perhaps decreased.	Frequency.	Normal, or perhaps decreased.
Diminished.	Volume.	Increased.
Diminished.	Power.	Increased.
Regular.	Rhythm.	Regular.
Slow.	Duration.	Quick.
Arterial anæmia; angina pectoris often present.	General Tendency.	As in obstruction, but sudden death more common than in any other form of valvular disease.

DIFFERENTIAL SIGNS BETWEEN MITRAL OBSTRUCTION AND MITRAL INCOMPETENCY.

MITRAL OBSTRUCTION.		MITRAL INCOMPETENCY.
Hypertrophy and dilatation of left auricle and right chambers.	Effect on Heart.	Hypertrophy and dilatation of all four chambers.
To left and slightly downward.	Apex Displaced.	To left and downward.

DIFFERENTIAL SIGNS BETWEEN MITRAL OBSTRUCTION AND
MITRAL INCOMPETENCY—(Continued).

MITRAL OBSTRUCTION.		MITRAL INCOMPETENCY.
To right of sternum, also to left at base, greatly.	Cardiac Dullness Increased.	To right of sternum, and also to left and downward.
Feeble, undulating, and diffused.	Character of Impulse.	Even more deficient in force.
To right of sternum and in epigastrium.	Impulse, where?	Generally increased all over cardiac region.
Onward; auriculo-ventricular.	Murmur, its Direction.	Backward; ventriculo-auricular.
Diastolic, presystolic, loudest at termination of diastole.	Murmur, Time.	Systolic, loudest at beginning of systole.
A little within and upward from apex beat.	Point of Greatest Intensity.	A little outward and upward from apex beat.
Upward and inward toward right base.	Direction in which Propagated.	Upward toward left base, and backward into axilla, and behind.
Generally rough and harsh.	Character of Sound (very uncertain and of little value for diagnosis).	Blowing, bellows murmur.
Immediately precedes the first at apex, which is often very loud.	Relation to Normal Heart Sounds.	Replaces first at apex.
Intensification of pulmonary second.	Effect on Second Sound.	Intensification of pulmonary second.
Presystolic; upward and inward from apex.	Thrill.	At apex and toward axilla.
Increased. Diminished. Diminished greatly. Very irregular. Quick.	Effect on Pulse. Frequency. Volume. Power. Rhythm. Duration.	Increased. Somewhat diminished. Diminished a little. Somewhat irregular. Nearly normal.
Pulmonary and venous congestion and slow death by asphyxia.	General Tendency to.	As in obstruction, but there is more tendency to dropsy.

DIFFERENTIAL SIGNS BETWEEN PULMONARY OBSTRUCTION
AND TRICUSPID REGURGITATION.

PULMONARY OBSTRUCTION.		TRICUSPID REGURGITATION.
Systolic, onward, ventriculo-pulmonary.	Murmur.	Systolic, backward, ventriculo-auricular.
Left border of sternum, in second interspace.	Point of greatest intensity.	Base of ensiform cartilage.
Generally anæmia. Sometimes pressure of solidified lung (phthisical or pneumonic) upon the artery. Rarely organic, and then usually congenital.	Cause.	Generally secondary to disease of the lung or of left side of the heart.
Frequently <i>Bruit de diable</i> in the jugular veins.	Associated Signs.	Systolic pulsation of the distended jugular veins.

Endocardial murmurs can be distinguished from pericardial by attention to the following physical signs:—

PERICARDITIS.

STAGE.	SYMPTOMS.	PHYSICAL SIGNS.
1st Stage. Inflammation without effusion).	If occurring during the course of acute rheumatism the disease may come on insidiously. Pain and tenderness in the cardiac region. Palpitation. Increased frequency of the pulse. Shortness of breath. Anxiety. Pyrexia.	Greater extent of visible impulse than natural, and on palpation the impulse is found to be more forcible, but unequal. Friction fremitus rare. Area of dullness not altered. Single or double friction sound, often preceded by a cantering action of the heart.* Heart sounds may be unchanged or even louder than in health, or they may be masked by the friction sounds.

* Cantering action of the heart, beside being met with in commencing pericarditis, is also caused by reduplication of the first or second sound of the heart against the thoracic wall at the moment of diastole, generally due to pericardial adhesions.

PERICARDITIS—(*Continued*).

STAGE.	SYMPTOMS.	PHYSICAL SIGNS.
2d Stage. (With effusion).	Less pain. Pulse small, frequent, and sometimes irregular. Dyspnoea and often orthopnoea. Irritable cough. Loss of voice. Dysphagia. Fullness of veins in the neck. Duskiness of complexion. Great anxiety. Sleeplessness. Delirium.	Bulging of the præcordial region. Impulse displaced upward and outward; undulatory. On palpation, feeble and sometimes not perceptible; irregular. Area of cardiac dullness increased, first noticed at the base of the heart, and afterward extending to left of apex beat, increased by the recumbent posture. Heart sounds feeble, distant and muffled at apex, louder and more superficial at base. Friction may or may not be heard.
3d Stage. (Resolution).	A gradual subsidence of the symptoms of the second stage.	Diminution of the dullness from above and laterally. Heart sounds become clearer. Friction sounds may be heard with increased intensity.

POST-MORTEM APPEARANCES.—1st. Pericardium is dry, inflamed and has lost its polish. Exudation of lymph on both surfaces, but more on the visceral. The membrane may have a shaggy appearance.

2d. Fluid in variable quantity in the sac of the pericardium. Usually sero-fibrinous, containing flocculi of lymph. It may be purulent or bloody.

3d. Organized lymph on the pericardium, with or without adhesions between the two surfaces, adherent or united by mesh-like adhesions.

The Pain of Pericarditis.—Rheumatic pericarditis is more or less painful; but secondary pericarditis developing in the acute stage of infectious or the chronic period of cachectic diseases, is invariably painless.

Peripheric pain nearly equal on both sides of the chest; or remaining localized at the præcordial region, at the epigastrium, or at the left side of the xyphoid cartilage, does not increase the danger of the pericarditis. But if *central*, giving rise to disturbance of circulation and respiration, and simulating that of angina pectoris, it means acute inflammation of the cardiac nerves, and marks an exceptionally bad case of pericarditis. (Dr. WERTHEIMER, "Thèse de Paris," 1876; Dobell's Reports.)

DIAGNOSIS BETWEEN ACUTE ENDOCARDIAL AND EXOCARDIAL
(PERICARDIAL) SOUNDS.

The sounds respectively perceptible in *endocarditis* and *pericarditis* and allied disorders, may be discriminated by the following table:—

ENDOCARDIAL.	EXOCARDIAL.
1. A blowing sound, soft and bellows-like; not affected by pressure.	1. A creaking, rubbing, rough, to-and-fro sound, intensified by pressure of the stethoscope and by the patient bending forward.
2. A thrill may be felt on palpation.	2. On palpation friction fremitus may be felt.
3. The sound appears distant.	3. The sound appears near.
4. May exist only with the systole or the diastole.	4. Exists with diastole as well as systole.
5. Accompanies the heart sounds.	5. Does not correspond with the rhythm of the heart.
6. Heard along the course of the great vessels, or conducted round to the back	6. Confined to the region of the heart and limited to site of production.
7. Persistent character.	7. Rapid and frequent change in character; here to-day and gone to-morrow.
8. Area of cardiac dullness not altered.	8. Increased area of dullness, if fluid be also present.

DIFFERENTIAL SIGNS OF CARDIAC DILATATION AND PERICARDITIS
WITH EFFUSION.

CARDIAC DILATATION.	PERICARDITIS WITH EFFUSION.
Dullness increased in the horizontal axis, of a square outline.	Præcordial dullness extends upward and is of a rounded pyramidal outline, with apex above.
Heart sounds feeble but clear.	Heart sounds feeble, and distant sounding.
Transition from dullness to lung resonance more gradual	Transition from dullness to lung resonance abrupt.
No friction sound.	Occasionally friction sound.
Limits of dullness persistent.	Limits of dullness often vary from day to day or week to week.
Apex beat felt at lower limits of cardiac dullness.	Apex beat some distance above lower limit of cardiac dullness. (SANSOM).

There is no doubt but that the general rules laid down for diagnosing pericardial effusion have been too vague. Dr. T. M. ROTCH, of Boston, has lately re-examined the subject, and succeeded in fixing a more perfect diagnostic sign than any hitherto mentioned. He shows that *an area of flatness at from two to three centimeters from the right edge of the sternum in the fifth intercostal space* is almost absolutely sufficient to mark the presence of an effusion, and differentiate it from enlarged heart.*

DIFFERENTIAL SIGNS OF HYPERTROPHY AND DILATATION.

	SIMPLE HYPERTROPHY.	HYPERTROPHY WITH DILATATION.	SIMPLE DILATATION.
Palpation.	Cardiac area extended. Impulse strong, lifting, or forcing.	Extent of visible impulse greatly increased. Action regular, strong.	Extent of impulse greatly increased, but feeble, without lifting or forcing character.
Percussion.	Dullness increased laterally and downward.	Dullness lateral and downward.	Dullness increased in the horizontal axis of the heart.
Auscultation.	First sound dull, prolonged, intensified; second sound intensified. No respiratory murmur over præcordium.	Both sounds prolonged.	Both sounds short, abrupt and feeble. Feeble respiratory murmur.
Pulse.	Strong, full, incompressible.	Less strong, variable.	Weak, compressible, irregular.
General symptoms.	Fullness in the head, epigastric weight, short breath, rarely debility; Bright's disease.		Dyspnœa, cough, palpitation, portal congestion, debility, ascites.

FATTY DEGENERATION OF THE HEART.

This condition of the heart is frequently associated with dilatation. Generally the area of præcordial dullness is normal or slightly increased; the impulse weak; the apex beat indistinct; the action irregular; the first

* "Medical Communications of the Massachusetts Medical Society." 1878.

sound short and feeble; the second prolonged and intensified; pulse is irregular.

These physical signs obviously offer very little ground for a diagnosis. Of rational signs the following have been mentioned:—

1. Attacks of faintness attended with sensations of great coldness, recurring without obvious cause. (DA COSTA).

2. *Arcus senilis*. For this to be significant of cardiac degeneration, the ring must be ill-defined, rather yellowish than white, and the rest of the cornea be slightly cloudy or opaque, not clear and translucent, a tinge of jaundice being present. When this is the case, “the chances of cardiac degeneration are formidable” (SANSOM).

3. Paroxysms of severe pain across the upper part of the sternum, and in the region of the heart.

4. Stomach derangements, accompanied sometimes by constipation, but more generally by diarrhoea and frequent vomiting. This Dr. L. H. J. HAYNE thinks “almost pathognomonic of this disease.” (*Lancet*, January, 1875.)

5. The “Cheyne-Stokes” Respiration of ascending and descending rhythm is present in about one-third of the cases, and is probably dependent on atheroma of the aorta (HAYDEN). This symptom was first described in a case by Dr. CHEYNE, in 1818, as follows:—

“For several days his breathing was irregular; it would entirely cease for a quarter of a minute; then it would become perceptible, though very slow; then, by degrees, it became heaving and quick; and then it would gradually cease again. This revolution in the state of breathing occupied about a minute, during which there were about thirty acts of respiration.” In this case fatty disease of the heart was very marked, while the valves were healthy, and the aorta was “studded with steatomatous and earthy concretions.”

No general attention, however, was directed to the peculiarity and striking character of this symptom, until, in 1846, Dr. STOKES urged its significance as a sign of fatty degeneration of the heart, believing that its presence was pathognomonic of this affection, and that it always betokened a fatal and not far distant termination. That it did not necessarily depend on fatty degeneration of the heart itself, was soon shown by Dr. SEATON REID, who described a case in which the muscular structure was

healthy, while the mitral and aortic valves were both incompetent, the left ventricle was hypertrophied, and the aorta dilated and atheromatous. It remains an important and significant, if not pathognomonic sign.

Dr. HAYDEN is of opinion that the absence of the impulse, or its extremely feeble character; the brief duration of the first sound, whether marked or sharp, in primary cases, and its almost complete or absolute extinction in those preceded by hypertrophy; the restriction of the sounds within a very limited area; and the occasional irregularity of the heart's action, will suffice, in the majority of cases, to establish the diagnosis of fatty heart from the physical signs alone. He adds that the incipency of primary fatty degeneration may be suspected, if the pulse, previously regular, becomes weak and irregular; if the surface be pale, the patient subject to dizziness or syncope, and the cardiac impulse feeble; although the sounds of the heart may not appreciably differ from their normal character.

A *slow pulse* sometimes is associated with fatty heart; but it also occurs as a result of disorders of the vagus nerve, following diphtheria; or after an attack of malarial fever; or after the administration of certain drugs, such as digitalis or aconite. In all cases it is necessary to exclude a slow pulse which is natural and peculiar to the patient. Irregular or slow pulse due to adherent pericardium may be distinguished by the history and physical signs.

CHAPTER IV.

DISEASES OF THE DIGESTIVE SYSTEM.

THE STOMACH AND BOWELS.—*Principal Symptoms—The Tongue—The Appetite—Acidity* (1) *from Fermentation*, (2) *from Hypersecretion—Pain—Flatulence—Vertigo*, (1) *Stomachal*, (2) *Cerebral—Vomiting*, (1) *Stomachal*, (2) *Cerebral—Comparison of Atonic Dyspepsia, Chronic Gastritis, Gastric Ulcer and Gastric Cancer—Indigestion or Dyspepsia—Abdominal Phthisis—Obstruction of the Bowels, Enteritis and Colitis.*

THE LIVER.—*Method of Examination—Significance of Pain in the Liver—Significance of Jaundice—Jaundice with Obstruction—Jaundice without Obstruction—Diseases Characterized by Enlargement with Smooth Surface; Enlargement with Uneven Surface; with Diminution of the Organ—Hepatic Abscess.*

INTERNAL PARASITES.—*Tape-worm—Hydatids—Round Worms—Thread Worms—Trichinosis.*

The principal symptoms to which the attention is directed in the diagnosis of diseases of the digestive organs are those connected with the tongue, the appetite, pyrosis, vomiting, flatulence, vertigo and pain.

THE TONGUE.

Late writers have shown considerable skepticism on the accuracy of the appearance of the tongue as indicative of the condition of the lining membrane of the stomach. It is true that a white and furred or a red and cracked tongue is occasionally seen in healthy subjects; but the standard of comparison should not be an ideally clean tongue, but the condition of the organ in the patient under inspection when in health. Local causes, such as carious teeth and irritating agents (tobacco, tea, mercury, etc.), must be allowed for in the examination. When these and similar considerations are weighed together with the repeated instances of simultaneous affections of the stomach and tongue revealed by *post-mortems*, no question remains that the appearance and state of the latter organ often is of high diagnostic worth.

Dr. ROBERT FARQUHARSON states, in a recent lecture on the diagnosis of dyspepsia,* that in his experience the class of tongue which coincides most commonly with digestive disturbance is that in which the tongue seems to be covered with a thin, white fur, which on minute inspection is seen to be composed of a series of minute raised dots, and this usually coincides with pain immediately following meals.

If the tongue is raw and quite stripped of epithelium, with enlarged and prominent papillæ, as we often see in phthisis, pain immediately after food and vomiting are usual symptoms, or large, red papillæ may stand in bold relief through a pale coating, or the tongue may be simply large and pale and flabby, as though too big for the mouth.

Dr. WILSON FOX specifies the following conditions of the tongue as valuable aids to diagnosis in this class of diseases:—

Dyspepsia with distinct atony of the stomach. The tongue broad, pale, and flabby, the papillæ generally enlarged, more especially on the tip and edges.

Dyspepsia from irritative causes. The tongue is redder than usual, often of a bright florid color, or even raw looking. It is often pointed at the tip, which, together with the sides, presents an extreme degree of injection, the papillæ standing out as vivid red points. This form is often associated with aphthæ, and is most common in scrofulous children and phthisical adults.

Dyspepsia from excessive or hurried eating is apt to present a tongue uniformly covered throughout the greater part of its surface with a thick fur, whitish or brownish, with some degree of enlargement and redness of the papillæ at the tip and edges.

Neuroses of the stomach display a tongue which, as a rule, is clean, though often pale, broad and flabby.

THE APPETITE.

Anorexia, or loss of appetite, is observed in cancer, in most inflammatory states of the stomach, in obstinate constipation, as well as in the pyrexial state.

Boulimia, or excessive appetite, is found associated with enlargement of the stomach, induration of its coats, also in diabetes and various forms of mental alienation.

* *Medical Press and Circular*, July, 1877.

Capricious and depraved appetite is met with in sufferers from intestinal worms, in some cases of chronic inflammation of the stomach, as well as in chlorosis, pregnancy and hysteria.

ACIDITY OF THE STOMACH, (1) FROM FERMENTATION, (2) FROM HYPER-SECRETION.

Acidity of the stomach, pyrosis, heartburn, and water-brash, are disturbances of the digestion frequently included in one category. In all, an excessive amount of acid is formed in the stomach; but in some cases the origin of the acid is to be sought in fermentative action, and in others in hypersecretion from the coats of the stomach, thus calling for different lines of treatment.

The following differential table, based on one given by Dr. WILSON FOX, exhibits in a concise form the distinction between the two forms of acidity:—

ACIDITY FROM FERMENTATION.	ACIDITY FROM HYPER-SECRETION.
Occurs in connection with causes which impede digestion.	Is most common as a reflex symptom, or in connection with other nervous disturbance, or with ulcer and cancer of the stomach.
Usually attains its height some hours after food, and is more marked in proportion to the size of the meal, and inversely to the digestive powers.	Occurs in the empty stomach, or rapidly after food, and is often of great intensity after a small meal.
Flatulence is common.	Flatulence is rare.
Pain not severe, and but slightly or not at all relieved by eating.	Pain more severe, most felt when the stomach is empty, and is relieved by food.
Vomiting is rare.	Vomiting is common.
Vomited matters may contain organic acids, torulæ and sarcinae.	Vomited matters apt to show hydrochloric acid in excess.
Urine frequently shows an alkaline reaction.	Urine rarely alkaline.

In both forms the process of digestion is impaired, but to a more marked degree in the fermentative variety, in which also, as a natural consequence, the impairment of nutrition of the patient is more obvious. As the fermentative action interferes with the functions of the liver, the stools are apt to be pale, and the patient suffer from constipation. The frequency with which attacks of gout and rheumatism are preceded by this form of acidity points to a diathetic process involving the general constitution.

PAIN.

Pain in the stomach is indicative of one of the following conditions:—

1. The presence of irritating foreign bodies, as mechanical substances, corrosive poisons, blood or bile in large quantities, inflation from air or gases, etc.

2. Organic diseases altering the anatomical structure of the coats, especially gastritis, chronic ulcer, cancer and thickening of the pylorus.

3. Perverted secretions, as in acidity.

4. Perverted innervation, which may be a local neurosis, as in forms of dyspepsia where pain is the prominent symptom, or as in cramp of the muscles of the stomach; or it may be from general disorders, as in patients of a rheumatic or gouty diathesis; or it may be referable to the general nervous system, as in pure neuralgia of the stomach and hysteria.

Pain in the stomach must be distinguished from rheumatic and other pains in the abdominal muscles immediately over the stomach. In the latter the superficial tenderness is much greater; it is usually more marked in the left recti and obliqui abdominis muscles, and especially near their attachment to the ribs, where moderate pressure cannot affect the stomach, and by its independence of the digestive acts (BRICQUET).

Pain in the stomach is also liable to be simulated by pain in the course of the transverse colon, especially when the colon is distended with gas. The diagnosis may usually be made by gentle percussion, the note arising from tapping a distended colon being less prolonged and of a higher pitch than that elicited from the stomach. The pain from the colon is also less felt at the ensiform cartilage than in the hypochondriac regions, and often extends toward the sigmoid flexure, and is associated with other signs of intestinal flatulence.

Pain in the stomach depending on diseases of the spinal cord is dis-

tinguished by its superficial tenderness, by the presence of other painful points in the affected nerve, and by the co-existence of other nervous, and the absence of digestive symptoms.

FLATULENCE AND ERUCTATION.

Dyspeptics generally suffer from gases in the stomach, producing eructations. These gases are either generated from imperfectly digested food or are secreted from the capillaries.

Eructations having the taste or odor of spoiled eggs, and occurring during the process of digestion, indicate the presence of sulphuretted hydrogen, from the decomposition of food.

When the eructations are odorless, and occur chiefly in an empty state of the stomach, they indicate a gaseous secretion of carbonic acid, hydrogen or nitrogen from the coats of the capillaries.

In the former case the indications are to use anti-ferments; while in the latter relief is often attained by simply regulating the hours of meals, so as to avoid long intervals between the times of taking food.

VERTIGO. (VERTIGO E STOMACHO LCESO.)

Stomachal vertigo is not unfrequently difficult to distinguish as such, because in all severe vertigoes the stomach is disturbed. In undoubted examples the vertigo always bears some distinct relation to the condition of the stomach, coming on only when that organ is full, or only when it is empty, or only after certain articles of food, as shell-fish, strawberries, coffee, fresh bread, etc. There are also generally some dyspeptic symptoms other than vertigo complained of. Some other points are mentioned in the following table:—

STOMACHAL VERTIGO.

Usually appears in definite relation to taking food; either after a meal, after particular ingesta, or on an empty stomach.

Generally occurs in middle life.

The apparent motion is felt to be subjective, not real (GOWERS).

Special senses not involved beyond perverted vision. Consciousness never lost.

CEREBRAL VERTIGO.

Occurs without relation to the taking.

Occurs in advanced life.

A sense of movement or actual turning of objects.

Deafness and tinnitus aurium often present. Sometimes loss of consciousness.

VOMITING, (1) FROM DISEASE OF THE STOMACH, (2) FROM DISEASES OF THE BRAIN.

Persistent vomiting is a frequent symptom of obstinate gastric disturbance; and it has also been frequently noted as a symptom associated with organic diseases of the brain and cord, not unfrequently masking them and diverting the attention of the practitioner from the real seat of lesion. Thus in suddenly induced cerebral anæmia, in the commencement of the paralysis which follows diphtheria, in tubercular meningitis, in concussion of the brain, in poisoning affecting the brain and cord, and in fact in almost any disease of the cerebral centres, but especially the meninges, it is possible that one of the earlier and prominent symptoms will be obstinate vomiting.

A comparison of the leading clinical features of these two forms shows that they may be readily distinguished.

In a general way it may be stated that vomiting arising from the stomach is attended with more or less pain, with a furred tongue, with constipation or diarrhœa, sense of weight at the epigastrium, and preceded for a considerable period by a sense of nausea.

Vomiting from cerebral causes, on the other hand, is usually characterized by an absence of these symptoms, by a clean tongue and a history of freedom from digestive disturbance.

Dr. ROMBERG has given the following criteria for its discrimination when the vomiting is of cerebral origin :—

1. The influence of the position of the head; the vomiting is arrested in the horizontal, and recurs and is frequently repeated in the erect position.

2. The prevailing absence of premonitory nausea.

3. The peculiar character of the act of vomiting; the contents of the stomach are ejected without fatigue or retching, as the milk is rejected by babies at the breast.

4. The complication with other phenomena, the more frequent of which are pains in the head, and irregularity of the cardiac and radial pulse, increased during and subsequent to the act of vomiting.

The following differential table further exhibits the points of contrast (from Dr. W. FOX):—

GASTRIC VOMITING.

Epigastric pain and tenderness are common, and in some cases very marked.

Nausea is constant.

Oppression and weight at the epigastrium are constant.

Bowels are variable.

The tongue is loaded, except in certain cases of cancer or ulcer.

Headache is absent, or not intense, chiefly frontal, of gradual invasion, and relieved by vomiting.

Vertigo is rare and relieved by vomiting.

Other nervous phenomena are rarely present, and then only in slighter forms, and relieved by vomiting.

CEREBRAL VOMITING.

Epigastric pain and tenderness are rare.

Nausea is frequently absent.

These are rare.

Bowels are constipated.

The tongue is usually clean.

Headache often violent, the invasion sudden, and not relieved by vomiting.

Vertigo is very frequent and not relieved by vomiting.

Indistinctness of vision and diplopia. Confusion of ideas. Loss of memory. Not relieved by vomiting. Anæsthesia or paræsthesia, paralysis or cramp, convulsion or coma, are common or soon supervene.

The indications derived from the nature of the matters thrown up in vomiting are as follows:—

Ingesta. The food is returned unaltered, or but slightly changed, in nervous vomiting; in a half digested state and strongly acid in chronic inflammation and cancer of the stomach; mixed with the microscopic forms known as *sarcinæ* and *torulæ* in chronic gastritis, gastric ulcer and cancer.

Mucus is vomited in a catarrhal or sub-inflammatory condition of the stomach.

Bile appears whenever the retching is long and violent, and does not indicate any special disease.

Pus is not formed in the stomach, and when present in the vomit indicates disease in the œsophagus.

Fæces also indicates a disease elsewhere than the stomach, usually an obstruction of the intestinal canal.

Blood is thrown up in gastric cancer and ulcer, in severe gastritis, in ex-

ternal injuries, vicariously (of the uterus), and frequently from disease of the heart or liver, producing distention of the capillaries. The presence of blood directly proceeding from the stomach, says Dr. FOX, if accompanied by severe pain, is almost pathognomonic of either gastric ulcer or cancer.

ATONIC DYSPEPSIA, INFLAMMATORY DYSPEPSIA, GASTRIC ULCER, GASTRIC CANCER.

The chief points in the diagnosis of diseases of the stomach are those connected with the differentiation of simple dyspepsia (atony of the stomach), inflammatory dyspepsia (gastritis, gastric catarrh, catarrhal inflammation of the stomach), gastric ulcer and gastric cancer.

From this group the nervous disturbances of the stomach are broadly marked off by the superficial character of the pain in these latter, its independence of the acts of digestion and the nature of the food, the co-existence of other neuralgia, the frequent absence of emaciation and other disturbances of nutrition, and the sex and age of the patients.

In reference to the value of *percussion* in diagnosing gastric cancer, Professor PETER, of Paris, has directed attention to the fact that when superficial percussion, *percussion en dèdolant*, is made over the stomachal region somewhat distended by gas, there is found at certain points, especially in the region of the greater curvature, a certain obscurity of the note alternating with the zones of sonority. But this sign is absolutely wanting on deep percussion such as is ordinarily employed. Prof. PETER, by this means, diagnosed a cancer of the stomach situated at the posterior surface of the greater curvature, with some cancerous nodules probably disseminated through the epiploon below the splenic region and also in the hypogastric region. At this last point also superficial percussion gave the same results.

An early sign of gastric cancer is the presence of enlarged glands in the *skin of the navel* (MAUNDER). To ascertain the mobility and outline of the stomach, the patient may be desired to drink one or two tumblers of soda water. This distends the stomach and makes the tumor prominent.

The following comparative table, drawn from the works of Drs. W. FOX, WILLIAM BRINTON and DA COSTA, illustrates the main points of difference in the four diseases named:—

ATONIC DYSPEPSIA.

No pain or soreness at the epigastrium. Sensation of weight or load, rather than tenderness.

Symptoms of indigestion. Appetite impaired. Thirst generally absent. Tongue pale and flabby.

Vomiting rare.

No hemorrhage.

Bowels may be regular.

No febrile symptoms.

Nutrition not materially interfered with.

Not confined to any age.

Course of disease uniform; may be cured.

No tumor. Percussion resonant.

CHRONIC GASTRITIS.

Pain at the epigastrium somewhat augmented by food; also soreness. Both constant, though not severe.

Indigestion present. Appetite capricious. Thirst increased. Tongue furred and red at edges.

Sometimes vomiting.

Hemorrhage absent, or rare and trifling.

Bowels constipated.

Occasional slight pyrexia (Fox).

Slight emaciation; slightly earthy tint to skin (Fox).

More common in middle or advanced life.

Disease with marked exacerbations and remissions. May be relieved or cured.

No tumor. Percussion resonant.

GASTRIC ULCER.

Pain at the epigastrium much augmented by food; is intermittent; subsides after digestion; pain in paroxysms, but not lancinating; epigastric soreness strictly localized. Sometimes a painful spot over lower dorsal vertebra.

Symptoms of indigestion slight.

Vomiting may be present or not. Usually relieves the pain.

Abundant hæmatemesis.

Bowels slightly or not constipated.

No fever present.

Frequently extreme pallor and debility.

May occur in middle-aged persons, but is frequently seen in young adults, especially females.

Duration uncertain; may get well; may run on rapidly to perforation; or may last for years.

No tumor. Percussion resonant.

GASTRIC CANCER.

Pain paroxysmal, radiating, often severe and lancinating; rarely remitting; never intermitting; little or not at all affected by food; not always accompanied by soreness.

Symptoms of indigestion more marked. Extreme acidity of the stomach.

Vomiting a very frequent symptom. Does not relieve the pain.

Hæmatemesis not very abundant; but occasioning frequently vomiting of a substance resembling coffee grounds.

Constipation obstinate.

Fever not uncommon.

Gradual and progressive loss of flesh and debility.

Most common in elderly people; rarely occurs in persons under forty years of age.

Duration about one year; very rarely reaches two. Termination fatal.

Generally a tumor. Percussion variable.

INDIGESTION AND DYSPEPSIA.

Although the distinction is not generally drawn in ordinary language between dyspepsia and indigestion, it should not be forgotten that they are not synonymous. Dyspepsia has reference to an altered condition of the digestive fluid, its deficiency or excess, or to an organic affection of the muscular walls of the stomach, which has for its result imperfect or difficult chymification of the food; indigestion merely expresses a disturbance of function, and refers to the result rather than the cause. In dyspepsia the peptic glands or muscular apparatus of the stomach are defective, in indigestion they may be normal, but have their functions interfered with by improper and unaccustomed articles of food, or by reflex influence from other organs.

The symptoms of INDIGESTION are tabulated by Dr. MURCHISON as follows* :—

1. A feeling of weight and fullness at the epigastrium and in the region of the liver.

2. Flatulent distention of the stomach and bowels.

3. Heartburn and acid eructations.

4. A feeling of oppression, and often of weariness and aching pains in the limbs, or of insurmountable sleepiness after meals.

5. A furred tongue, which is often indented at the edges, and a clammy, bitter, metallic taste in the mouth, especially in the morning.

6. Appetite often good; at other times anorexia and nausea.

7. An excessive secretion of viscid mucus in the fauces, and at the back of the nose.

8. Constipation, the motions being scybalous, sometimes too dark, at others too light, or even clay colored. Occasionally attacks of diarrhoea, alternating with constipation, especially if the patient be intemperate in the use of alcohol.

9. In some patients attacks of palpitation of the heart, or irregularity or intermission of the pulse.

10. In many patients occasional attacks of frontal headache.

11. In many, restlessness at night and bad dreams.

12. In some, attacks of vertigo and dimness of sight, often induced by particular articles of diet.

* "Functional Derangements of the Liver." London, 1874.

DYSPEPSIA may be due to impaired motion as well as to deficient secretion. The following table will give the distinctive points for diagnosis:*

	1. DYSPEPSIA FROM IMPAIRED MOTION.	2. DYSPEPSIA FROM DEFECTIVE SECRETION.
Uneasiness after meals.	Constant symptom, generally soon replaced by sense of tension accompanying flatulency.	Not infrequent, but commonly soon merged into acute pain.
Flatulence	Characteristic symptom.	Comparatively infrequent; some of the worst cases, in which pain after food and other symptoms are particularly severe, are entirely free from flatus. The tendency is to lactic, butyric, and perhaps other forms of fermentation, in which gases are not evolved.
Gastric pain.....	Infrequent, but occurs occasionally, as a result of flatulence, and is peculiar in kind.	Variouly described as sharp, shooting, dull, or dragging, is the most characteristic symptom of defective secretion of gastric juice.
Constipation	Almost always a marked symptom.	Not generally present, and the bowels are in many cases relaxed.
Treatment.....	Strychnia, carbolie acid, thymol, charcoal.	Diet, tonics, pepsin acids, hygienic treatment.

Hyperperistalsis, dyspepsia of fluids, flaccid stomach, and other states remain for discussion.

ABDOMINAL PHTHISIS.

Abdominal phthisis (tubercular peritonitis) in its acute forms, closely simulates typhoid fever. There are febrile symptoms attended with remissions, heat and dryness of the surface, pains in the limbs, drowsiness and disordered secretions, and diarrhœa. It differs from typhoid in these particulars:—

1. The pain is diffused over the abdomen, not limited to the cœcal region.
2. There are no red spots (with rare exceptions).
3. There is generally tubercular disease in other organs.
4. The temperature has not the morning remissions of typhoid.

* ARTHUR LEARED. "Dyspepsia." *British Medical Journal*, May, 1879. p. 660.

OBSTRUCTION OF THE BOWELS.

The causes of a mechanical stoppage of the bowels are principally the following: intussusception; impaction of feces; strictures; twisting of the bowel (volvulus); herniæ; pressure of tumors.

The symptom first noticed is constipation with colicky pains, which do not yield to ordinary remedies; slight distention of the abdomen and some soreness on pressure. Vomiting follows, very severe, even becoming fecal. It is liable to be confounded with peritonitis and strangulated hernia. The following rules for diagnosis have been laid down by the eminent surgeon, Mr. JONATHAN HUTCHINSON, of London:—

1. When a *child* becomes suddenly the subject of symptoms of bowel obstruction, it is probably either intussusception or peritonitis.

2. When an *elderly person* is the patient, the diagnosis will generally rest between impaction of intestinal contents and malignant disease (stricture or tumor).

3. In *middle age* the causes of obstruction may be various; but intussusception and malignant disease, both of them common at the extremes, are now very unusual.

4. Intussusception cases may be known by the frequent straining, the passage of blood and mucus, the incompleteness of the constipation, and the discovery of a sausage-like tumor, either by examination *per anum* or through the abdominal walls.

5. In intussusception, the parietes usually remain lax, and, there being but little tympanites, it is almost always possible, without much difficulty, to discover the lump (or sausage-like tumor) by manipulation under ether.

6. Malignant stricture may be suspected when, in an old person, continued abdominal uneasiness and repeated attacks of temporary constipation have preceded the illness. It is to be noted also that the constipation is often not complete.

7. If a tumor be present and pressing on the bowel, it ought to be discoverable by palpation, under ether, through the abdominal walls, or by examination by the anus or vagina, great care being taken not to be misled by scybalous masses.

8. If repeated attacks of dangerous obstruction have occurred with long intervals of perfect health, it may be suspected that the patient is the sub-

ject of a congenital diverticulum, or has bands of adhesion, or that some part of the intestine is pouched and liable to twist.

9. If, in the early part of a case, the abdomen become distended and hard, it is almost certain that there is peritonitis.

10. If the intestines continue to roll about visibly, it is almost certain that there is no peritonitis. This symptom occurs chiefly in emaciated subjects, with obstruction in the colon of long duration.

11. The tendency to vomit will usually be relative with three conditions and proportionate to them. These are (1) the nearness of the impediment to the stomach, (2) the tightness of the constriction, and (3) the persistence or otherwise with which food and medicine have been given by the mouth.

12. In cases of obstruction in the colon or rectum, sickness is often wholly absent.

13. Violent retching and bile vomiting are often more troublesome in cases of gall-stones or renal calculus simulating obstruction than in true conditions of the latter.

14. Fecal vomiting can occur only when the obstruction is moderately low down. If it happen early in the case, it is a most serious symptom, as implying tightness of constriction.

15. The introduction of the hand into the rectum as recommended by Prof. SIMON, of Kiel, may often furnish useful information.

INFLAMMATORY DIARRHŒA (ENTERITIS) AND DYSENTERY (COLITIS).

These diseases, both alike in being inflammations of the mucous membrane of the intestinal tract, are frequently associated. But for therapeutic as well as prognostic purposes, it is desirable to recognize the distinctions which they present in well marked types. They are—

ENTERITIS.

Seat of inflammation is in the small intestine.

Usually begins with colic, nausea and vomiting, constipation (rarely diarrhœa), chilliness soon followed by high fever, thirst and hot skin.

DYSENTERY.

Seat of inflammation is in the large intestine.

Usually begins with painless, slight diarrhœa, followed by chill, slight or no fever, sense of weight near the anus. No colic.

ENTERITIS.

Pulse at first tense and full; soon becomes small, wiry, quick.

Pain paroxysmal, local tenderness marked, greatly increased by pressure.

Stools mucous, rarely blood, very rarely pus. No scybala. No tenesmus.

Aortic pulsation felt by the patient on the right of the umbilicus.

DYSENTERY.

Pulse often little excited; or if fever is high, full and rapid.

Pain more moderate, usually distinctly over the colon, moderate tenderness.

Stools scanty, bloody, contain pus, scybala, little feces. Marked tenesmus.

Aortic pulsation not noticed by the patient.

DISEASES OF THE LIVER.

Previous to an examination of the liver, the patient should have a free action of the bowels, as fecal accumulations are a constant cause of diagnostic errors. He should lie on his back on a firm bed, with his knees drawn up and the abdominal muscles relaxed. Palpation should be upon the skin directly, not on the clothing. The physician, seating himself on the patient's right side, should apply the tips of the fingers of the right hand just below the free border of the ribs, and request the patient to make full inspiration and expiration. He will thus be able to feel the upper edge and surface of the liver and ascertain the *condition of its surface*, whether smooth or nodular. By percussion, which should be made while the patient is in the same position, the *size* of the liver can be quite accurately mapped out. These two facts are the first steps to a diagnosis; as most hepatic diseases can be assigned to one of these classes—

1. Liver enlarged, with smooth surface.
2. Liver enlarged, with nodular surface.
3. Liver atrophied.

Pain in the hepatic region should be examined; whether dull or acute, persistent or intermittent, etc. The condition of *jaundice* is ascertained, in light cases, by examining the under surface of the tongue and the conjunctiva of the eye, which will display the icteric discoloration when the general surface does not. A still more delicate test of the presence of jaundice is derivable from examination of the urine. The following three tests are employed by Prof. HARDY, of Paris:—

1. *Chloroform*. When this is poured upon normal urine it sinks, by reason of its great density, to the bottom of the test-glass, exhibiting there a crystalline transparency. If we pour it on the icteric urine, and having shaken the test-tube plugged by the thumb, leave it quiet for a moment, the chloroform deposit contrasts strongly by its dull color with the yellow of the superficial layers—the yellow color being deeper in proportion to the quantity of bile in the urine. It is an excellent test of icteric urine.

2. *Iodine*. When the iodine is poured upon the icteric urine the mixture must not be shaken. At the upper part of the tube three very distinct colors are observable—the first layer formed by the tincture is violet; below this is a kind of diaphragm of sea-green color; and the third layer, consisting of the urine, and occupying the lowest part, is yellow.

3. *Nitric Acid*. When this agent has been poured in, the mixture after shaking assumes a bottle-green color, passing into an olive. This is an entirely special and very characteristic appearance.*

With these hepatic symptoms determined, a study of the following tables will in most instances readily supply a correct diagnosis.

THE SIGNIFICANCE OF PAIN IN THE LIVER.

Pain having its source in the liver is divided by Dr. CHARLES MURCHISON† into three varieties, each of diagnostic significance:—

CHARACTER OF PAIN.

I. Pain severe, paroxysmal, with distinct intermissions; little or no local tenderness; no fever; often associated with jaundice.

II. Pain moderate, continuous, slightly increased by pressure, often associated with pain in the right shoulder, slight febrile symptoms and jaundice.

III. Pain severe, constant, greatly increased by pressure, motion, coughing, etc. More or less fever; perhaps jaundice.

DISEASES FOUND IN.

Obstruction of the bile duct by gall-stones, etc. (hepatic colic); hepatic neuralgia (when jaundice is absent, probably the latter).

Congestion and commencing inflammation of the organ; catarrh and partial obstruction of the bile ducts; acute atrophy.

Always indicates inflammation of the capsule (peri-hepatitis), which may supervene in various diseases (cirrhosis, hydatids, etc.).

* *Revue de Therapeutique*, August, 1878.

† "Lectures on Diseases of the Liver."

Hepatic pain may be simulated by various other conditions. The principal ones, with their characteristic differences, are as follows:—

1. *Pleurodynia*. The pain is strictly localized to a small spot. Absence of hepatic disturbance.

2. *Intercostal Neuralgia*. Tender points along the course of the intercostal nerve. Chiefly referred to three points in the course of the nerve: (1) The vertebral groove; (2) The axillary region; (3) The termination of the nerve in front. Co-existence of neuralgia elsewhere. Absence of hepatic symptoms.

3. *Pleurisy*. Presence of pyrexia and physical signs of the disease.

4. *Gastrodynia*. Comes on with relation to food (stomach always either full or empty). Pyrosis.

5. *Intestinal Colic*. Pain referred to the umbilical region. No jaundice. Blue line of lead poisoning. Errors of diet.

6. *Renal Colic*. Pain chiefly referred to one kidney, when it shoots to the testicle and down the thigh. No jaundice. Hæmaturia and renal calculus.

Little or no hepatic pain is felt in—

1. The waxy, lardaceous, or amyloid liver.
2. The fatty liver.
3. Simple hepatic hypertrophy.
4. Hydatid tumor.

THE SIGNIFICANCE OF JAUNDICE.

The common and obvious symptom of jaundice results either (1) from obstructions of the common bile duct; or (2) independent of any obstruction of the duct. The diagnosis of these two conditions may be presented as follows:—

JAUNDICE FROM OBSTRUCTION.	JAUNDICE WITHOUT OBSTRUCTION.
When persistent, speedily becomes intense. The stools are clay-colored. Tumor in the region of the gall-bladder often present. May appear suddenly in a person in good health.	Persists and continues slight. The stools are natural. No tumor there. Appears gradually, unless there is a history of shock.

JAUNDICE FROM OBSTRUCTION.

Intermittent jaundice in advanced life signifies gall-stones.

Pain, usually, in severe paroxysms.

Co-existence of ascites, pregnancy, pyloric cancer (obstruction from without).

JAUNDICE WITHOUT OBSTRUCTION.

Intermittent jaundice in youth signifies catarrh of the duodenum.

Pain usually more or less constant.

Preceding severe mental emotion, pyæmia, malarial fevers, phosphorus poisoning, epidemic prevalence.

The principal diseases which are associated with these varieties of jaundice are the following:—

JAUNDICE FROM OBSTRUCTION MAY BE DUE TO

DISEASES.	DIAGNOSIS.
1. GALL STONES.	Biliary colic present. Pain acute, paroxysmal, referred to the gall bladder, and from this round to the right scapula. Tenderness absent or slight. Irregular rigors. No fever. Severe vomiting. Jaundice appears after a day or two. Pathognomonic; the presence of gall stones in fæces.
2. HYDATIDS.	Liver enlarged and altered in form but painless. Biliary colic with fever, quick pulse and high temperature. Pathognomonic; hydatid vesicles in the fæces.
3. CANCER AND TUMORS.	Antecedent history of visceral cancerous disease. Pain and nausea after taking food. A hard and sensitive tumor in the epigastric or right hypochondriac region. Hemorrhage from the stomach or bowels.

JAUNDICE WITHOUT OBSTRUCTION MAY BE DUE TO

DISEASE.	DIAGNOSIS.
1. MALARIAL FEVERS. Yellow Fever, Pyæmia.	History of malarial or specific poisoning, or actual presence of one of the diseases named.
2. EPIDEMIC JAUNDICE.	Gastric catarrh; stools pale; epigastric soreness; nausea or vomiting; loss of appetite; often commences with a chill after exposure. Most epidemics of jaundice seem to have been due to malarious poison or vitiated atmosphere. Infantile jaundice is of the latter character.

JAUNDICE WITHOUT OBSTRUCTION MAY BE DUE TO—(Continued)

DISEASE.	DIAGNOSIS.
3. NERVOUS JAUNDICE.	History of severe mental emotion, great suffering or sudden shock. Onset rapid; often cerebral symptoms.
4. JAUNDICE FROM CONGESTION.	Feeling of weight and soreness over liver. Bad breath; poor appetite; furred tongue; vertigo. Right decubitus. Urine scanty and high colored. Slight dyspnoea. Bowels sluggish.

Acute atrophy, mineral poisons (especially by phosphorus), and very obstinate constipation, are other occasional causes of this form of jaundice.

CLASSIFICATION OF HEPATIC DISEASES WITH REGARD TO THE SIZE OF THE LIVER.*

1. LIVER ENLARGED, SURFACE SMOOTH.

SIMPLE HYPERPLASIA.	° Liver enlarged, smooth, painless; absence of other symptoms.
LEUKEMIC H Y P E R - P L A S I A.	Liver enlarged and smooth. Spleen enlarged. Pallor of the skin. Pathognomonic; presence of a marked increase of the white blood globules, 1:20 and upward.
CONGESTION.	Enlargement moderate. Tenderness; conjunctiva jaundiced; stools pale; bowels irregular; tongue coated; low spirits; headache; vertigo; noises in the ears. No jaundice or dropsy.
(a) Simple.	Liver enlarged, smooth. Slight jaundice. Some dyspnoea. Dropsical effusions. Mitral or aortic disease. Emphysema or induration of the lungs.
(b) From cardiac disease.	Enlargement slight. Enlarged spleen. History of malarial disease. Pathognomonic; the malarial pigment in the blood.
(c) From malaria.	Enlargement considerable, uniform, of slow growth, borders sharply defined, feel firm. Pain slight. Patient emaciated and cachectic. Splenic enlargement common. Diarrhoea and dyspepsia. History of phthisis, syphilis or protracted suppuration.
WAXY DEGENERATION.	

* Partly taken from E. J. JANEWAY, "Diagnosis of Hepatic Affections." N. Y., 1877.

CLASSIFICATION OF HEPATIC DISEASES WITH REGARD TO THE SIZE OF THE LIVER.

I. LIVER ENLARGED, SURFACE SMOOTH—(*Continued*).

FATTY DEGENERATION.	Enlargement considerable, borders rounded, feel doughy. No tenderness nor pain. Spleen small; jaundice slight or absent. Diarrhœa. A pale, smooth, greasy skin. History of intemperance, phthisis or indolent life.
HYDATID TUMORS.	Enlargement considerable, irregular, painless; usually of the left lobe of the organ. Feel elastic or fluctuating. Jaundice rare. Increase of size slow. No constitutional symptoms.
SIMPLE ATROPHY.	Liver small, surface even. Preceded by ascites, dyspnœa, serious disease of heart or lungs, or signs of congestion.
ACUTE YELLOW ATROPHY.	Rare. Jaundice always present, though rarely intense. Pain considerable. Tenderness. Generally vomiting; splenic dullness. Pulse irregular. The typhoid state common. Urine dark, acid, sp. grav. 1.012-1.024; absence of urea, uric acid and the chlorides; presence of leucine and tyrosine (pathognomonic). Intestinal hemorrhage and hæmatemesis common.

II. LIVER ENLARGED, SURFACE NODULAR OR IRREGULAR.

ABSCESS OR TROPICAL HEPATITIS.	Liver enlarged, irregular surface bulging. Dull, heavy pain. Jaundice rare. Pyrexia and chills. History of residence in a warm climate.
CANCER.	Enlargement often very great, progressive, irregular; nodular excrescences often to be felt. Feel hard and resistant. Pain lancinating and tenderness acute. No febrile symptoms. Jaundice. "The co-existence of enlarged liver with persistent jaundice ought always to raise the suspicion of cancer" (MURCHISON). Dyspepsia, nausea, vomiting, constipation, or diarrhœa, short, dry cough, ascites. Patients over 40. In suspected can-

CLASSIFICATION OF HEPATIC DISEASES WITH REGARD TO THE SIZE OF THE LIVER.

II. LIVER ENLARGED, SURFACE NODULAR OR IRREGULAR—(*Continued*).CANCER (*Continued*).

cer of the liver the urine should always be examined; half a drachm of strong nitric acid should be added to half an ounce of the urine. If the fluid changes to a dark or black hue, and especially if no albumen is present, and the liver is either increased or diminished in size, the diagnosis of *melanotic cancer* is rendered very probable. (Dr. EISELT, of Prague.)

SYPHILITIC LIVER.

Liver enlarged, surface nodulated, lobes irregular, separated by deep fissures.

III. LIVER DIMINISHED IN SIZE.

CIRRHOSIS, OR CHRONIC ATROPHY.

Liver small, sometimes only half size, surface granular or nodulated; "hob-nail liver." Outset insidious, with signs of disordered digestion. Dull pain and slight tenderness in hepatic region. Ascites common. Spleen often enlarged. Superficial veins of the abdomen enlarged. Hemorrhoids frequent. Jaundice rare or slight. Progressive emaciation and debility. History of spirit drinking almost invariably.

HEPATIC ABSCESS.

It has lately been shown* that an obscure and chronic form of hepatic abscess is a far more common disease in the United States than is generally supposed, and that it is often exceedingly difficult of diagnosis.

These abscesses may exist without any local symptoms or such general disturbance of the system as is commonly regarded as indicating their presence, and are a very common concomitant of prolonged malarial poisoning. The pathognomonic sign of their presence is the *discovery of pus on aspiration of the parenchyma of the liver*. This operation is not dangerous, and there need be no hesitation in its performance. The place of election is one of the intercostal spaces. The rational symptoms may be collated as follows:—

* TAUSCKY, *Med. Record*, April 20th, 1878; HAMMOND, *St. Louis Clin. Record*, June 1878; BYRD, *N. Y. Med. Journal*, July, 1878, etc.

1. Gastric and intestinal derangements ; dyspeptic symptoms of various kinds.

2. Slight jaundice, conjunctivæ yellow ; complexion sallow.

3. Depression of spirits, hypochondria or melancholy. This is a very usual symptom, and so important that Dr. HAMMOND recommends that in all cases of hypochondria or melancholia the region of the liver should be carefully explored, and even if no fluctuation be detected, or any other sign of abscess be discovered, aspiration, with proper precautions, should be performed. If pus be evacuated, the operation may be expected to be followed by a cure of the mental disorder, as well as by the preservation of the life of the patient from the probably fatal consequences of hepatic abscess.

4. Sense of weight or pain in the right side ; more or less tenderness on pressure (all local symptoms often absent).

5. Circumscribed fluctuation over the hepatic region. This is a positive sign, but is by no means always to be discovered.

6. Cerebral symptoms, as vertigo, cephalalgia, insomnia hysteria and hyperæmia.

7. Slight rigors, and feverishness, simulating some of the more chronic forms of intermittent fever.

INTERNAL PARASITES.

The symptoms to which parasites in the intestinal canal and other organs give rise are numerous, but by no means specific or definite. The following tabular arrangement sets forth the more prominent :—

TAPE WORM.

Tænia Solium.

Tænia Saginata.

Pain and discomfort in the belly ; variable appetite ; constipation and diarrhœa alternating ; *itching at the nose or anus* without local cause, low spirits, loss of flesh, nervous seizures. Stools unusually dark or light.

Pathognomonic : The discovery of joints in the stools, or about the anus, or of eggs in the fæces (microscopic).

HYDATID CYSTS.

Tænia Echinococci.

These occur chiefly in the lungs and liver. (See Diseases of the Liver.) They begin with a rounded, tense, smooth, elastic swelling, painless until inflammation begins, and without other symptoms than those caused by their size. They are often attended with the "hydatid thrill." This may be felt by placing the left hand flat and closely upon the tumor, then percussing sharply with the fingers of the right hand. A long sustained tremor is observed, "like that experienced on an iron railway bridge during the passage of a train."

Pathognomonic: Echinococci or microscopic hydatids in the contained fluid, which may safely be drawn by aspiration.

ROUND WORMS, LUMBRICI.

Ascaris Lumbricoides.

Symptoms of intestinal irritation. Capricious appetite. Pain of a gnawing or griping character. Tenderness on deep pressure over the abdomen. Tumid condition of the belly. Alternate constipation and diarrhœa. The tongue pale, flabby, indented by the teeth, and often has a peculiar shiny appearance. Pupils generally dilated. Squinting, nervous twitchings, or even convulsions. Sleep is restless, with grating of the teeth and waking with sudden starts. Fever may appear, often of a remittent type (worm fever, vermin fever).

Pathognomonic: Worms found in alvine evacuations.

THREAD WORMS.

Ascaris Vermicularis or Oxyuris Vermicularis.

Violent itching and irritation at the anus and vagina, increased at night. Tendency to strain. Itching at the nose. Leucorrhœa.

Pathognomonic: Worms found upon examining the parts, also seen in patient's bed and his under-clothing.

TRICHINOSIS.

(Trichinæ in the blood and muscular system).

Trichina Spiralis.

First Stage: Gastro-intestinal disturbances; thirst; loss of appetite; nausea; colicky pain in the abdomen; constipation or diarrhœa; coated tongue; feverishness. *Second Stage:* Swelling and stiffness of the muscles; muscular soreness; œdema of the subcutaneous tissue; copious sweating; debility and increased

TRICHINOSIS (*Continued*). fever; dyspnoea; hoarseness and loss of voice; dropsy commencing in the eyelids and face, and proceeding to the extremities; difficulty of motion and respiration.

Pathognomonic: Presence of trichinæ in the fæces; or in the muscular structure.

The differential diagnosis from *rheumatism* is in the soreness being in the muscles and not the joints; from *typhoid fever* in the unusual pain and stiffness; the early swelling, dropsy, etc.

Trichinæ do not colonize equally throughout a muscle, but in groups here and there. It is best, therefore, to dissect out a muscle lengthwise in order to judge of their number.

The very large number of symptoms attributed to the presence of worms in the intestinal canal is the irritation they cause, implicating the general nervous system. This, occasionally, extends so far as to produce a "worm fever," which in many respects resembles a mild remittent with unusually pronounced nervous symptoms. The tongue is pale and flabby, and often has a peculiar shiny appearance (DATE). The pupils are generally dilated. Squinting sometimes occurs, and nervous twitchings of a choreic character. The fever is often high, with great heat of skin, and the cerebral manifestations being marked, may lead to the suspicion of hydrocephalus. From this it can be distinguished by the mere direct remissions; by the previous history, showing the primary symptoms to be referable to derangements of the alimentary canal; by the less obstinate constipation; and by the expulsion of worms.

It has also been confounded with tubercular disease. Here the most important diagnostic point is the temperature. This in tubercular disease is always high; but when the irritation is from worms it is either normal or but temporarily elevated above the normal standard.

CHAPTER V.

DISEASES OF THE URINARY SYSTEM.

The Early Signs of Bright's Disease—Comparative Diagnosis of the Different Forms of Bright's Disease (Acute Parenchymatous Nephritis, Chronic Tubal Nephritis, Yellow Fatty Kidney, Secondary Contraction of Kidney, Interstitial Nephritis or Renal Cirrhosis, Albuminoid or Amyloid Renal Degeneration, Parenchymatous Renal Degeneration)—Diabetes Mellitus and Glycosuria—Diabetes Insipidus and Hydruria—Bile in the Urine—Urinary Calculi.

General methods for the examination of the urine, and the chemical reagents and manipulations required in its analysis, are to be found in so many text-books and treatises that we may omit them here, and confine ourselves to the differential symptoms of some of the most prominent and frequent renal diseases.

THE EARLY SIGNS OF BRIGHT'S DISEASE.

The early progress of Bright's disease is often remarkably insidious, and readily escapes recognition. Nor is it to be detected by the familiar and easy plan of testing for albumen; for this substance is by no means invariably present in the urine, even in advanced and well marked cases. FOTHERGILL justly observes that the progress of interstitial nephritis is often without the albuminous secretion for long periods.

On the other hand, it has been abundantly shown that albumen is occasionally and transiently present in the urine of persons who present no traces of nephritis; who, in fact, may be in excellent health.

Hence the value of other means of determining the existence of these forms of renal disease becomes manifest. Of these the presence of *hyaline casts* has recently been urged as pathognomonic of renal hyperæmia and inflammation, and invariably present.* These must be sought for with considerable care, as from their transparent character, and the fact

* Dr. B. A. SEGUR, Proceedings of the Medical Society of King's Co., 1878, p. 241.

that they do not form a sediment, they are readily overlooked. The directions given for their search are that the urine to be examined is placed in a tall, conical glass; after three to six hours it is inspected; from the visible deposits, whether floating or sedimentary, with the pipette a quantity is taken sufficient to fill a concave slide or a shallow cell.

This little pool is first searched with a four-tenths objective, and in a little time any cast or other microscopic object it contains is found. A more careful observation is made of the object thus found with the one-fifth. When the examination of deposits has been made in this way, the conical glass of urine should be set aside (a little chloral may be added, to prevent decomposition), and after twelve hours more the examination should be repeated. Of course, it will be remembered that the hyaline cast may be found when the condition of the kidney is only one of transient hyperæmia.

The effort has also been made to call in the aid of the ophthalmoscope. The presence of minute white exudations in the retina, principally around the maculæ luteæ, are believed to point to the presence of Bright's disease, and to be found in its early stages (retinitis albuminurica). The appearance of the retina in these cases is characteristic. It consists in the grouping of small white spots, the outline of each being clearly defined; they are invariably circular, of extremely small dimensions, and present the appearances of a pearl of an intensely bright color, and stand out from the retina in a marked manner. The grouping of the spots is symmetrical in each eye, and is generally in the form of a crescent. Often the urine will only yield signs of the minutest quantities of albumen—sometimes none at all; but hyaline casts and these white spots may be detected by the processes here described.

We shall now proceed to classify the diagnostic points in the differentiation of the seven forms into which the varieties of Bright's disease are now divided, premising that more than one form may exist in the same patient.

COMPARISON OF THE DIFFERENT

	ACUTE DESQUAMATIVE NEPHRITIS.	CHRONIC TUBAL NEPHRITIS.	YELLOW FATTY KIDNEY.
History.	Sudden onset after scarlet fever or exposure to wet and cold; œdema of the face the sign first noticed; headache, feverishness, pain in the loins, gastric disturbance.	Symptoms of more than six weeks' duration. Often history of acute nephritis. Uræmic symptoms; abnormally low temperature. Serous inflammations. Cardiac hypertrophy.	Often follows alcoholism.
Appearance.	Dropsical, more or less swollen about the face; skin generally dry.	More or less œdema, and general anasarca. A pale, almost characteristic, waxy look.	Dropsy considerable and persistent; renal cachexia often marked.
Urine.	Scanty, smoke colored, dark when acid, red if alkalized. Highly albuminous. Specific gravity high, 1.025-1.030. Reddish brown sediment of epithelial, blood and hyaline casts.	Generally scanty, though variable. Pale, albumen about one-fourth, specific gravity low, 1.005-1.015; white sediment of hyaline and epithelial casts. No blood casts.	Scanty, pale, low specific gravity, with abundant sediment of oil casts and cells filled with oil. Albumen abundant.
Prognosis.	Recovery frequent. May lead to chronic tubal nephritis.	Recovery not likely.	Almost certainly fatal.
Pathology.	Kidneys enlarged, congested, vascular; cortical substance increased. Tubules dark and dense.	Kidney enlarged, cortical substance increased, capsules easily separated. "Large white kidney."	Kidneys enlarged, fatty, mottled, the tubes full of fat and oil cells.

FORMS OF BRIGHT'S DISEASE.

SECONDARY CONTRACTION OF KIDNEY.	INTERSTITIAL NEPHRITIS. RENAL CIRRHOSIS.	ALBUMINOID OR AMYLOID RENAL DEGENERATION.	PARENCHYMATOUS RENAL DEGENERATION.
Symptoms of more than a year's duration. Headache. Coma or convulsions. Cardiac hypertrophy. Epistaxis.	Symptoms few and faint. Often the arthritic diathesis. Exposure to cold and fatigue. Sense of weariness. Frequent headaches. Amaurosis. Cardiac hypertrophy.	Antecedent syphilis, phthisis or osseous disease of chronic suppuration. Enlarged liver or spleen. Chronic diarrhœa.	Pregnancy, diphtheria, or acute fever.
Generally some dropsy, but not very extensive. Face sallow.	Little or no dropsy. Nerve implications, as paralysis, loss of sight or hearing, etc.	Dropsy generally amenable to treatment. Emaciation. Face sallow or pallid. Dyspnœa.	Generally no dropsy.
Scanty, pale, specific gravity about 1.015. Albumen moderate. Sediment of pale casts, dark granules, fatty cells and waxy products.	Largely increased, pale; albumen trifling; sediment little, of finely granular casts, or minute oil drops. Specific gravity low.	Largely increased. (50-60 oz.) pale or golden; albumen considerable, perhaps one-half. Specific gravity 1.007-1.015; little or no sediment; casts hyaline and waxy.	Normal in amount. Albumen $\frac{1}{10}$ to $\frac{1}{4}$ bulk.
Generally fatal, but of slow progress.	With care, not immediately dangerous, but predisposes to uræmic attacks from exposure.	Incurable, though the patient may live for years.	Recovery frequent.
Kidneys contracted, dense, capsule adherent; atrophy of the tubules.	Kidneys at first enlarged, later contracted; connective tissue increased; capsule adherent, diminished and corrugated. "Chronically contracted" kidney.	Kidney enlarged, smooth, waxy looking.	Kidney enlarged. the parenchyma more or less hypertrophied.

In the form of *amyloid degeneration* the difficulties of diagnosis are considerable, as not only has it been generally recognized that albumen may be absent for considerable periods while the disease is steadily advancing, but it has been abundantly shown that it may never appear at all in fatal cases.*

It seems, therefore, certain that we possess at present no sure diagnostic sign of amyloid degeneration of the renal vessels; that on the one hand, it is likely to be confounded with, or mistaken for, chronic parenchymatous nephritis arising under identical etiological conditions; on the other, it runs a great risk of being altogether overlooked. But both of these evils may be avoided with a little care. BARTELS points out that the differential diagnosis between amyloid disease and chronic parenchymatous nephritis depends upon the distinguishing characters of the urine, which, in the former, is clear, with little sediment and few casts, mostly hyaline, and scarcely ever blood-corpuseles; in the latter it is always more or less turbid, with considerable sediment, is dirty colored, contains many casts of every variety, and not uncommonly blood-corpuseles. In those cases in which no albumen was present, there have been signs of amyloid disease in other organs; and, in order to escape error, it will be enough to know that the absence of albumen from the urine does not exclude a slight degree of amyloid disease of the kidneys.

Cystic kidney is not considered worthy of special remark, since ordinary cysts are not to be recognized with any certainty during life, nor can they always be distinguished from the chronic varieties of Bright's Disease, in which they frequently are developed (DA COSTA).

DIABETES MELLITUS AND GLYCOSURIA.

The presence of *sugar in the urine* is characteristic of both these conditions. The most convenient simple test is caustic potash (MOORE'S test), either in solution or small fragments. Heated with urine containing sugar, this substance immediately produces a more or less yellow or brown color, the intensity of which is in proportion to the quantity of sugar present, and a peculiar sweet smell (melassic acid).

The test usually preferred is TROMMER'S or FEHLING'S, which depends upon the reduction of a salt of copper by the sugar. The Fehling's
*LECORCHÉ, "Maladies des Reins, Paris, 1875; LITTEN, *Berliner Klin. Wochenschrift*.

test may be obtained in a solid form as "cupric test pellets," as suggested by PAVY.*

Apart from this test, the presence of sugar in the urine is revealed by many indications. We may often recognize it by grayish patches on the clothing or linen, which are reduced to powder when scratched with the nail. In women the chemise, from prolonged contact with the urine, may become spotted and stiffened, as if by drops of syrup. Another circumstance indicating the sugary savor of the urine, especially in the country, is the great number of flies or ants that will be attracted around the vessel containing it.

The presence of sugar once determined, it remains to decide whether it arises from simple glycosuria, which is a comparatively common and not dangerous condition, or from saccharine diabetes, which is more rare and a very perilous affection. This distinction has lately been insisted upon by M. GÉRIN ROZES. The contrasting features of the two disorders may be presented as follows:—

DIABETES MELLITUS.

Onset gradual; occurs at all ages, and without reference to known predisposing causes.

The amount of sugar varies very little.

The absence of saccharine food makes little or no change in the urine.

Volumetric analysis by Fehling's method is easy.

Polyuria, polyphagia, polydipsia, and impotence common and well marked.

Nervous complications frequent.

Treatment of little avail; result usually fatal.

SIMPLE GLYCOSURIA.

Onset sudden; more common in the aged; in persons consuming saccharine food; in the insane; in those taking chloral; in the paroxysms of ague; after sudden excitement; blows on the head; cerebral affections.

The amount of sugar varies greatly from day to day (pathognomonic, ROZES).

The withdrawal of saccharine food diminishes the sugar.

Such analysis is obscure, owing to the quantity of creatinine and similar substances present.

All these may be, and generally are, absent, or slightly marked.

Rare.

Treatment efficient; result usually favorable.

* See Article by Dr. NEFF, in *Medical and Surgical Reporter*, for May 10th, 1880.

With the knowledge of the very fatal character of diabetes mellitus, a recognition of its earliest symptoms becomes of immense importance for treatment. Its invasion is seldom sudden, and at the very outset may be curable, which it rarely or ever is when once developed.

Various nervous symptoms are among the earliest noted, and it is a wise rule in all nervous disorders of a doubtful character to examine the urine for sugar. Changes in the character of an individual, an abnormal irritability of temper, insomnia, and extreme feeling of fatigue, *disorders of vision*, itching of the skin, pruritus of the genital organs, especially the vulva, and more or less protracted headache, are often premonitory symptoms. Intense and obstinate neuralgic pains, without obvious cause, especially in the foot and leg, should lead to the suspicion of diabetes. Recurrent boils and carbuncles are well known to accompany the diabetic condition.

Genital impotence is one of the first signs of approaching diabetes; and whenever individuals are met with who, previously virile, become weak and impotent without coinciding disease, especially of the spinal marrow, diabetes will usually be found to be the cause. Valuable information is derivable from the mouth; for besides the insatiable thirst and dry mouth, some patients complain of a disagreeable taste, which is sometimes acrid, and at others faint, or bitter, or sugary; and it is this perverted taste which contributes to maintain the thirst.

The mouth frequently exhibits an aphthous condition, while the edges and tip, and even the whole surface of the tongue, may present a red aspect, as if the aphthæ had been removed. The gums, also, are often softened, fungous or bleeding; while in some the teeth become loose, or fall out without being decayed, and in others become carious. The breath is frequently of a bad, acid smell, and the saliva, on examination, is acid instead of neutral. Another fact which has sometimes led to the diagnosis is the existence of intertrigo at the commissure of the lips. This intertrigo labialis is not exclusively connected with diabetes, but when met with should always lead to an examination of the urine.

With regard to the digestive organs, boulimia on the one hand, and a complete repugnance for food on the other, with dyspepsia, should lead us to suspect diabetes. The unusual thirst of diabetics prompts them to drink large quantities of water at night, and such a habit should suggest

strict inquiry for other symptoms. As a general rule it may be said that whenever there is muscular debility, emaciation and anæmia, without discoverable local cause, the urine should be examined, and will almost always be found to contain either sugar or albumen.

The prognosis in a case of Diabetes Mellitus improves with the age of the patient; occurring in elderly persons, with ordinary care, it does not appear to shorten life (DA COSTA).

DIABETES INSIPIDUS AND HYDRURIA.

The habitual discharge of an excessive amount of urine of low specific gravity, and containing neither albumen nor sugar, if accompanied with progressive emaciation, excessive thirst, and loss of vital power, constitutes *diabetes insipidus*; but under various conditions excessive diuresis may be temporarily present, as in hysteria and other cerebro-spinal and nervous affections, without serious general symptoms, and constitute the condition of *hydruria*. The distinction between the two can be made by noting the coincident disease in the latter form, the slight direct impairment of the general health, the varying amount of urine voided, and by the fact that the quantity, although large, never attains those extraordinary measures—thirty to fifty pints daily—which marked cases of diabetes insipidus present. A large amount of urine is discharged by patients with amyloid degeneration of the kidney.

BILE IN URINE.

The significance of bile in urine is the same as that of jaundice (see page 200), as it indicates the presence of bile in the blood. The tests are those for the bile pigment and those for the biliary salts. The color test usually employed is that of Gmelin; a few drops of urine are placed upon a white plate and nitric acid dropped at its side; if bile pigment be present a play of colors, from grass-green to red, is produced. The same may be obtained by adding sulphuric acid to urine in a test tube, and dropping in a crystal of potassium nitrate. The tests for the biliary salts are so complicated that they are entirely unreliable, as generally applied. For cautions and directions for their use the reader is referred to NEUBAUER and VOGEL'S "Chemistry of the Urine."

URINARY CALCULI.

There are but three forms of calculi which are at all of common occurrence, and which are, therefore, likely to demand analysis. These are *uric acid and its compounds, oxalate of lime*, and the *mixed phosphates*. Calculi of *xanthine* and *cystine* are found, though very rarely.

1. *Uric acid calculi* are the most common. They are either red or some shade of red, and usually smooth, but may be tuberculated. They leave a mere trace of residue after ignition.

2. *Oxalate of lime calculi* are frequently met with. They are generally of a dark brown or dark gray color, and from their frequently tuberculated surface have been called mulberry calculi. They may, however, also be smooth. Considerable residue remains after ignition. The calculus is soluble in mineral acids without effervescence.

3. *Calculi of the mixed phosphates or fusible calculi* are composed of the phosphate of lime and of the triple phosphate of ammonia and magnesia. They form the external layer of many calculi of different composition, and may form entire calculi, but very seldom form the nuclei of other calculi. They are white, exceedingly brittle, fuse in the blowpipe flame, and are soluble in acids, but insoluble in alkalis.

Few calculi of large size are of the same composition throughout. Oxalate of lime is the most frequent nucleus; uric acid may also serve as a nucleus, but phosphates, as stated, almost never. Small collections of organic matter, as blood-clots, frequently form nuclei, and may often be recognized by the odor of ammonia on ignition. It is not uncommon to find calculi made up of concentric layers of different composition.

TO DETERMINE THE COMPOSITION OF CALCULI.*

Heat a portion of the *powdered* calculus to redness upon platinum foil. Note whether there is a residue.

A. *There is a fixed residue.* To a portion of the original powder apply the murexid test. (This is as follows: Dissolve a small portion of the powder in a drop of nitric acid on a porcelain plate, then carefully evaporate over a spirit lamp. When dry add a drop or two of liquor

* The processes here given are taken, with slight alterations, from Thudichum's work on the *Pathology of the Urine*.

ammonia, when, if uric acid is present, a beautiful *purple color* will appear where the ammonia spreads).

I. A purple color results; *uric acid* is present. Observe whether a portion of the calculus melts on being heated.

a. It melts and communicates—

1. A strong yellow color to the flame of a spirit lamp; *sodium urate*.

2. A violet color to the flame; *potassium urate*.

b. It does not melt. Dissolve the residue after ignition in a little dilute HCl, add ammonia until alkaline, and then ammonium carbonate solution.

1. A white precipitate falls; *calcium urate*.

2. No precipitate. Add some hydric sodic phosphate solution; a white crystalline precipitate falls; *magnesium urate*.

II. No purple color results. Observe whether a portion of the calculus melts on being heated strongly.

a. It melts (fusible calculus). Treat the residue with acetic acid; it dissolves. Add to the solution ammonia in excess; a white crystalline precipitate falls; *ammonio-magnesium phosphate*. In case the melted residue is insoluble in acetic acid, treat with HCl; it dissolves. Add to the solution ammonia; a white precipitate indicates *calcium phosphate*.

b. It does not melt. Moisten the residue with water, and test its reaction with litmus paper; it is not alkaline. Treat with HCl; it dissolves without effervescence. Add to the solution ammonia in excess; white precipitate; *calcium phosphate*. Treat the calculus with acetic acid; it does not dissolve. Treat the residue after heating with acetic acid; it dissolves with effervescence; *calcium oxalate*. Treat the original calculus with acetic acid; it dissolves with effervescence; *calcium carbonate*.

B. *There is no fixed residue.* Apply the murexid test (p. 216).

I. A purple color is developed.

a. Mix a portion of the powdered calculus with a little lime, and moisten with a little water; ammonia is evolved, and a

red litmus paper suspended over the mass is turned blue ;
ammonium urate.

b. No ammonia ; *uric acid*.

II. No purple color.

a. But the nitric acid solution turns yellow as it is evaporated,
and leaves a residue insoluble in potassium carbonate ;
xanthine.

b. The nitric acid solution turns dark brown, and leaves a
residue soluble in ammonia ; *cystine*.

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