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THE

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COMPRISING TEN VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY

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UNDER THE GENERAL EDITORIAL CHARGE OF

GUSTAVUS P. HEAD, M. D.

PROFESSOR OF LARYNGOLOGY AND RHINOLOGY, CHICAGO POST-GRADUATE MEDICAL SCHOOL

VOLUME IX

PHYSIOLOGY, PATHOLOGY, BACTERIOLOGY, ANATOMY, DICTIONARY

EDITED BY

W. A. EVANS, M. S., M. D.
ADOLPH GEHRMANN, M. D.
WILLIAM HEALY, A. B., M. D.

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PATHOLOGY AND ANATOMY.

W. A. EVANS, M. S., M. D.

Professor of Pathology, College of Physicians and Surgeons, Chicago; Pathologist to Cook County Hospital and Columbus Medical Laboratory.

PHYSIOLOGY, BACTERIOLOGY AND HYGIENE.

ADOLPH GEHRMANN, M. D.

Professor of Bacteriology, College of Physicians and Surgeons, Chicago; Bacteriologist to Columbus Medical Laboratory.

DICTIONARY OF NEW WORDS.

WILLIAM HEALY, A. B. (HARV.), M. D.

Instructor in Gynecology, Northwestern University Medical School.

TABLE OF CONTENTS.

SECTION I.

PHYSIOLOGY.

Cardio-Respiratory Phenomena and Roentgen Rays. Abrams[1] reports personal observations and says that many statements will have to be changed. The diaphragm does not flatten with each inspiration but its curve remains unaltered and during respiration it plunges piston-wise up and down. The excursion of the diaphragm was found greater on the left side than the right. The heart is not stationary but moves up and down, following the inclined plane of the central tendon. The aphorism of Fouler, "The position of the apex beat is the key to the diagnosis of nearly all the affections of the heart and chest" is no longer infallible. The rays prove that the apex beat may be palpated at a point remote from the anatomic apex and may be due to the impact of the ventricle against the chest wall. The disappearance of the apex beat during deep inspiration is shown to be due to a recession of the beat from the chest wall and not to a dilated right ventricle pushing the left aside. There was no dilatation found under the circumstance described.

The Heart Reflex. The writer states that his view, that myocardial contraction, resulting from cutaneous irritation in proximity to the precordium, is the beneficial factor in balneotherapeutics, is strengthened by later study. The accompanying chart shows the variation in heart size: Fig. 1.

Lung Reflex. In studying the phenomena of Chercheosky's sign of early arteriosclerosis, the rays show that

(1) American Medicine, Jan. 3, 1903.

there is a misinterpretation of the conditions observed. A percussion blow on the chest wall causes contraction of the lung in the adjoining area which will be seen to become bright again in a few seconds. This is what occurs in trying for the sign, and the blow on the epigastrium causes the lung to dilate again.

There is no contraction or dilatation of the aorta under

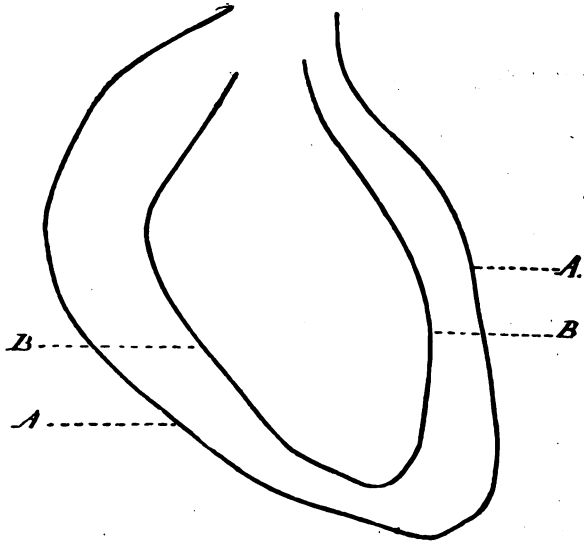


Fig. 1. Heart reflex in a boy aged 8. Duration of reflex 2½ minutes.

these circumstances. The cut shows the movement that takes place during these reflexes. Fig. 2.

Blood Pressure. Jackson[1] has used the Gaertner blood pressure machine for three years with success. He remarks that the apparatus should be tested from time to time against a mercurial manometer to insure accuracy of the readings. The name of the instrument should accompany all reports of the readings of blood pressure. With the Gaertner instrument the finger should always

(1) *Boston Med.* and Surg. Jour., Feb. 26, 1903.

be at the level of the heart. In teaching blood pressure to students the writer has used the method to advantage. He finds that frequently the blood pressure is as high as 150 mm. of mercury in young men against the usual 100 to 130 mm. In young women the pressure is lower, 90 to 110. A pressure of 140 to 150 may be normal for a man, but is abnormal for a woman. Elderly people always have

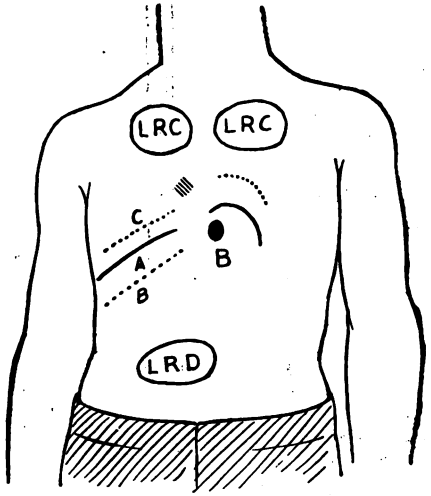


Fig. 2. The lung reflexes of dilation and contraction: A, normal areas of heart and upper liver border respectively; LRD, region for eliciting lung reflex of dilation which, when struck, causes lung border to descend to B, and cover heart area almost to obliteration; LRC, regions for eliciting lung reflex of contraction which causes lung borders to recede to C.

an elevated pressure due to changes in the arteries; in a person of 50 years it may be 175. The importance of the method in clinical work is urged.

Clifford,[1] writing on the same subject, concurs in this opinion that increased blood pressure follows arterial changes. There is, however, no evidence to show that it actually follows, or is an antecedent etiologic factor.

(1) The Lancet, March, 1903.

A *modification of the Gaertner method* is suggested by Hampela.[1] The tonometer is arranged as usual. One hand grasps the arm above the elbow and compresses the brachial artery either with the thumb direct or aided by

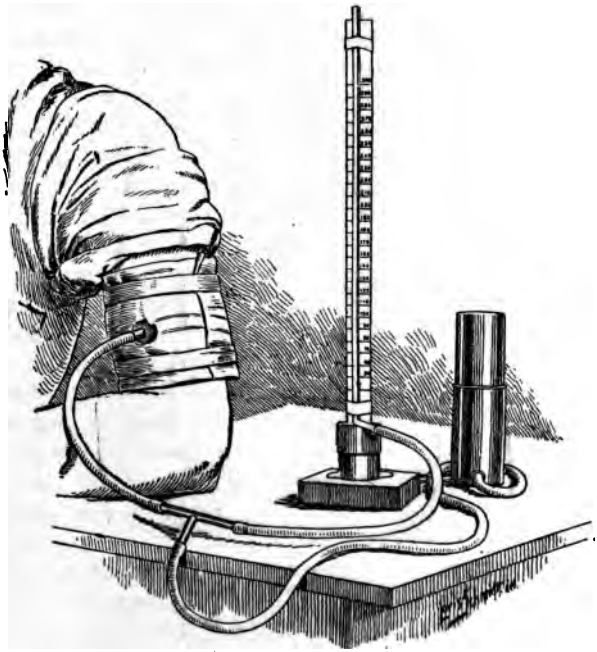


Fig. 2a. Stanton's blood pressure device.

a small pad. The other hand is on the pulse and it can now be exactly noted when the pulse disappears, and when it reappears, and at the same time the manometer will indicate the change in pressure.

In commenting on methods for determining blood pressure Stanton[2] remarks that it is necessary in order to get

(1) St. Petersburger med. Wochen., Jan. 4, 1903.
(2) University of Pennsylvania Medical Bulletin, Feb., 1903.

a complete idea as to arterial pressure in man that *both systolic and diastolic pressures be estimated.* It is only in this way that we obtain information as to the work of the heart, the peripheral resistance and mean pressure. He has also devised a simplified instrument that has been used with entire success. The following is a brief description of it: The armlet of rubber is three and three-fourths inches wide and sixteen inches long and closed at both ends. Outward expansion is prevented by canvas and metal supports. To the center of the rubber cap is cemented a rubber valve stem. Through this passes a piece of glass tubing which in turn is connected by pressure tubing to the mercurial manometer. A small air pump is interposed and connected by soft tubing. The armlet is put on over the relaxed arm and arranged at the level of the heart. The pulse is taken with the left hand and air is forced in by the pump until the pulse disappears. The fingers of the right hand now compress the soft tubing making a valve. Air is now allowed to slowly escape until the pulse returns and this point is mentally noted as the systolic pressure. The pressure is now gradually reduced and the pulsations in the mercury column will be seen to gradually increase in size until a maximum is reached and then to subside. The base line of these maximum pulsations is to be noted as the diastolic pressure. The mean pressure is that which will be found by taking the mathematical mean of the pressures obtained. By this means it is possible to determine the different phases of arterial pressure within so short a time that variations are unlikely.

The Control of Blood Pressure. This is the subject of an address by Geo. Crile[1] in which he reports results of experiments, and states some deductions that are of great practical bearing. Many times the control of blood pressure is synonymous with the control of life itself. Surgical shock is exhaustion of the vasomotor center. Involvement of the cardio-inhibitory, cardio-accelerator, or res-

(1) Boston Med. and Surg. Jour., March 5, 1903.

piratory centers is secondary. Collapse is due to suspension of the vasomotor mechanism. Under such circumstances the writer states, as practical points, the following: in shock, therapeutic doses of strychnia are inert, physiologic doses are dangerous or fatal. There is no practical distinction to be made between external stimulation of this center, and internal stimulation by vasomotor stimulants. Such stimulants are contraindicated. In shock cardiac stimulants have little usefulness and may be injurious. In collapse stimulants may be useful because the centers are not exhausted. Saline infusions in shock have a limited usefulness. In collapse they may be effective. The blood tolerates but a limited dilution with saline solution. Saline infusion in shock raises but it cannot sustain the blood pressure.

In his experiments with adrenalin the writer found that its effect is to raise blood pressure in the normal animal and all degrees of shock. Its effects are fleeting and it should be given continuously. By this means the circulation in a decapitated dog was maintained 10½ hours. By the combined use of artificial respiration and adrenalin injections into the jugular vein animals which were apparently dead for as long as fifteen minutes were resuscitated.

Variations in Blood Pressure During Work. Masing[1] finds as the result of his investigations that: If the same amount of work is done by an older as by a younger person the blood pressure generally increases more in the old person than in the young. The increase in blood pressure is usually less if the experiment is repeated. The increase in blood pressure is usually greater if the work is done with one leg than if both legs are used changing from one to the other,—intermittent. As a rule muscular work brings with it an increase in blood pressure which is proportional to the amount of work done. With the same amount of muscular work the blood pressure is greater or smaller according to the amount of mental energy

(1) Deutsch. Archiv für klin. Med., Nov., 1902.

needed to do the work. With the same amount of muscular work the mental energy gets less as experience increases. In the young man the blood pressure seems to be, within wide limits, independent of the length of time —working time—or in other words the blood pressure in the young man remains at the same high mark during the whole time of work. In the old person the pressure as a rule gradually decreases if the work lasts some time.

A comparison of the behavior of young and old persons shows that the older become exhausted quicker under the influence of muscular work. The heart of the old does not react to stimulation as the heart of the young.

These functional changes in the activity of the senile heart are due, according to Dehia and the author, to anatomic change leading to a senile myofibrosis.

The senile heart in consequence of a partial loss of its contractile substance is no longer in a position to do the work which it did when it was young. It easily gets insufficient if an increase in activity is asked.

Adrenalin Glycosuria. Herter and Wakeman.[1] In this extended study of the effect of adrenalin on animals, the fact that the painting of adrenalin extract over the surface of the pancreas produced glycosuria, induced the authors to look for the specific reason for this effect. Certain experiments made by others would seem to show that this effect is not induced by absorption and transfer to other organs, or by indirect action on the liver. It was found that other substances were capable of inducing the same condition, and that the fact as to whether the bodies were reducing or oxidizing agents was of chief importance. It was found that substances which reduce Fehling's solution occurred in the urine after the application of the following poisons to the pancreas: Sulphurous acid, sulphureted hydrogen, carbon monoxid, benzyl alcohol and pyrogallol. The oxidizing agents that were tried, yielded negative results, except in the case of nitrobenzol.

The appearance of sugar was not induced by the use of

(1) American Journal Medical Sciences, Jan., 1903.

chlorin water, bromin water, potassium chlorate, hydrogen peroxid, chromic acid or nitric acid in appropriate dilution. Sodium salicylate and potassium cyanid which have little or no reducing action, produced no glycosuria. It is therefore apparent that the reducing properties of the substances in question are not alone the cause, but that there must be some obscure toxic action upon the pancreatic cells. Division of the pancreas longitudinally or crosswise did not particularly change the result when either half was treated. The action of adrenalin and other substances on the islands of Langerhans the authors leave as a disputed point. In studying the relation of the adrenal glands and carbohydrate metabolism, the writers made the very interesting observation that extirpation of these glands entirely overcomes the glycosuria that follows intraperitoneal injections of adrenalin. It was further found that if the adrenals were removed or ligated the pancreas could be extirpated without the occurrence of the usual glycosuria that results after this operation, or, if it did appear, it was only for a short period. The effect of squeezing the adrenal gland in causing glycosuria is thought possibly to have its origin in the accompanying nervous disturbance. The experiments indicate a close relation between the adrenals and carbohydrates in the blood, but so far they do not give us any clear explanation of the facts.

The Sugar Controlling Function of the Suprarenal Glands. Herter[1] in a further note states that the fact that when adrenalin is applied to the pancreas it increases the sugar in the blood, suggested to the author that the organism has an hitherto undiscovered mechanism by which sugar metabolism is controlled. He put his hypothesis to an experimental test and obtained the following results: Massage if on both suprarenal glands is followed by glycosuria. Removal of the suprarenal glands or ligature of their vessels is followed by a rapid decline in the sugar of the blood. By total extirpation of the pancreas

(1) Medical News, Oct. 18, 1902.

and previous exclusion of the suprarenal glands, the urine was shortly after the operation free from any reducing substance. The author shows that codein forms a chemical union with the secretion of the suprarenal glands and suggests this as the cause of the well known fact that codein diminishes the excretion of some diabetic patients.

Nephrectomy and Absorption. Miltzer and Salant[1] found, during some experiments on rabbits, that removal of the kidney does not induce edema nor cause a relative plethora. All theories on the formation of edema seem to agree that it is due to increased transudation of fluid from the blood to the lymph spaces, caused at least in part by the failure of the kidneys to remove water from the blood. In many nephrectomies on rabbits and guinea-pigs the writers found that this was not the case. In fact, as shown by injections into the peritoneal cavity, the absorption was increased in marked degree over that in the control animal where the kidneys were present. This status of absorptive ability is due to the increased osmotic pressure of the blood in the absence of kidney excretion. This fact would explain the absence of edema in persons in whom anuria is present sometimes for four or five days.

The Adrenal Gland and Its Active Principle in Their Relation to Cytolysins and Antitoxin Production. A. C. Abbot[2] immunized four rabbits by injecting them intraperitoneally with a sterile emulsion of adrenal gland in .85 per cent salt solution. An average of 31 glands were injected during three months. There was little appreciable immediate effect. The rabbits usually lost about 25 per cent of their weight. The blood serum of these rabbits was injected in doses of .5 to 1 c.c. subcutaneously into guinea-pigs. The pigs settled upon the belly; the temperature fell; there was profuse urination and some bowel looseness; sometimes there were bloody urinations and blood in the serous cavities; the hemoglobin sometimes fell to 10 per cent, erythrocytes going to pieces. Paren-

(1) American Medicine, Jan. 24, 1903.
(2) Jour. Medical Research, June, 1903.

chymatous degeneration was quite marked, but not specifically prominent in the adrenals. The hemolytic action was not due to the rabbits' blood *per se*. When a rabbit had been prepared by injection of guinea-pig blood, the serum produced exactly the same effect in guinea-pigs as those noted with adrenal serum. However, Abbot thinks this is not due to the contained blood, but is due to the adrenal compounds chemically akin to blood. It is to be noted that the effect was on the blood. The adrenals did not suffer especially.

Transformation of Uric Acid. Croftan[1] reports some observations upon the fate of uric acid in the body that add much to our understanding of this complicated chemistry. He was led to this primarily through a study of uric diathesis and was in search of the reasons for the excess of this body in certain cases. Given by mouth it is known that uric acid appears only in small amount in the urine. This does not necessarily mean that it is destroyed, for it may be retained in the tissues, not absorbed from the tissues, or that it may be transformed into other bodies. By the use of intravenous injections with the aid of a special diuretic mixture of salts (S. A. Mathews) it was demonstrated that from 82.7 to 89 per cent of uric acid infused intravenously into rabbits is actually destroyed in passing through the body. The author now proposed three questions: Where is uric acid destroyed? How is it destroyed? What becomes of it? In answer to the first question extracts of all important organs were made and to these known quantities of uric acid were added. At the end of 48 hours another determination was made and the loss of uric acid noted. It was found that the uric acid destroying power of the liver is great in carnivora (cat, dog), while that of the kidney is slight. In herbivora (cow, rabbit) the reverse was shown. In omnivora (hog, man) both the liver and kidney can destroy much uric acid. All organs appear to have a more or less developed property in this regard.

(1) Medical Record, July 4, 1903.

On the second point it was now attempted to find if some certain constituent of the organ extracts was the active agency. Individual bodies did not possess the property but uric acid was destroyed by a combination of a nucleo-proteid having the power of dissociating peroxid of hydrogen, an albumose that does not have this power and the salts required for solution. The specific activity is thought to be due to a loosening of the affinities of the uric acid molecule by the albumose-like body and that the oxygen carrying nucleo-proteid completes the oxidation. The third question Croftan says is not answered entirely to his satisfaction. He finds that urea and oxalic acid can be formed from urea in certain organs. In none of the organ extracts and uric acid mixtures was allantoin found as an intermediary or end product. It would seem that in this diathesis the derangement consists in an inability to carry on proper uric acid destruction.

Lymphotoxic Serum. Ricketts[1] injected emulsions of the mesenteric lymph glands of guinea-pigs into rabbits. He got a serum which contained a leucotoxin, an endotheliotoxin, a leucoagglutinin and a precipitin. The leucotoxin affected the macrophages, microphages, and polymorphonuclears at about the same time. Ricketts does not say whether the lymphocytes were attacked or not. This is rather opposed to the opinion of Ehrlich that the granular forms spring only from the bone marrow, and supports the view of Dominici that the granular forms can be produced by the lymph structures. When the serum was injected intraperitoneally, or subcutaneously, there was marked leucocytosis after a short time. Ricketts attempted to determine the relation of these facts to immunity. He found that if the culture is injected one hour after the serum, immunity was decreased. If injected 24 hours after the serum, the immunity was moderately raised. This was due to the leucocytosis, though the increase in immunity was not proportionate to the raise.

(1) Trans. Chicago Path. Soc., Dec. 8, 1902.

The Large Intestine a Siphon. Leftwich[1] presents a number of experimental data which would prove that the force operating in the movement of the contents of the large intestine is siphonage. This part of the intestine has every character of a siphon. In order to find out if a siphon would act if its contents were partly fluid and partly solid a mixture of glycerin and air was experimentally tried in a siphon. It was found that it siphoned very actively and that the presence of air in the ascending arm by reducing weight accelerated the flow while in the descending arm the reverse was true. The balance point is the sigmoid flexure and under circumstances there can be back siphonage as well as the normal. Siphonage explains the single daily evacuation. At night when horizontal the siphon does not act. With a continuous flow and no sphincter above the rectum one would expect frequent evacuation as is the case in such quadrupeds as maintain the horizontal position. The force used to move the column of feces and start the flush is hydraulic and not muscular.

Vermiform Appendix. Considerable discussion has been brought out on the point of the functional activity of the appendix. Craig, Eccles, Sajous have presented arguments. A summary by Sajous[2] states the question clearly. Leaving aside the question of the embryologic relations of the organ, the anatomical construction of the appendix probably offers a very interesting reason for its existence. Sajous has interpreted the finding of lymph follicles and glands as being structures whose function is to guard the body against infection by their cytogenic action. These follicles asepticise the materials absorbed by the surrounding villi: they also supply leucocytes to the intestinal cavity, and these become important in the destruction of pathogenic bacteria. If such is actually the function of the appendix an increase in fermentation, at least temporary, should be noted in the cecum after removal. Some of the

(1) Edinburgh Med. Jour., Dec., 1902.
(2) *Medical* Sentinel, May, 1903.

symptoms described by patients may be thus ascribed; but it must be remembered that the organ may have been past physiologic functionation for a long time before excision. Eccles in the Hunterian lectures decides from post mortem dissections that the orifice to the appendicular cavity has a valve arrangement that prevents the entrance of substances into the organ, and at the same time allows the contents to escape freely. In addition muscular action is such that the contents are propelled towards the cecal cavity. As to the fluid Corpe[1] reports it as being about four ounces per day, and he terms it "a powerful germicide." If these views are sound the appendix performs functions similar to those of lymphatic glands elsewhere in the intestines. To consider the appendix as a useless organ is therefore an error, and further to remove a normal appendix during the course of other operations is unscientific and unsurgical. In the presence of inflammation the state of affairs is very different, and our course is clearly outlined.

Rigor Mortis. Folin[2] brings forward some facts regarding rigor mortis which tend to disprove the current theory that it is due to coagulation of certain proteids in the muscles. Although this theory is widely accepted, there has been little gained to strengthen it since its original inception. The fact that the onset of rigidity may be prevented and rigidity already produced may be made to disappear by simply bending or pressing a muscle would seem more in harmony with a contraction than a coagulation theory. Such disappearance of rigor should be due to a redissolution of the myosin, a point that has entirely failed of verification. To prove that rigor mortis is not a coagulation phenomenon the writer produced rigor by subjecting muscles to a temperature of —15° to —20° C. This is a fact long ago reported by Brücke. Frog muscle can be cooled to —7° C., frozen solid, without losing its irritability and contractility when carefully thawed. If

(1) Medical Sentinel, May, 1903.
(2) Amer. Jour. of Physiology, August 1, 1903.

the cooling is carried to —15° C. the power to recover contractility is lost and on thawing the muscle will be found to be in rigor. Under this circumstance the chemical change from a potentially living to a dead and rigid muscle must be very small. Few, if any, other changes occur; there is no shortening, the muscle is neutral or faintly alkaline and shows no sign of cloudiness which could point to coagulation. This perfect translucency of muscles in cold rigor as compared to fresh muscle would seem to disprove that it is due to coagulation or at least would make one think that the coagulum must be translucent. To further prove the point, muscles were frozen and macerated with cold normal salt solution and compared with similar extracts from simple preserved muscle. It should be clear that the two saline solutions obtained from these different muscles should be quite different if in one the coagulation of dissolved protein was the cause of rigor. This was not found to be the fact. The solutions were tested as follows: When set aside at room temperature both showed about the same amount of coagulum. Other tubes when heated side by side coagulated at the same time at 40°-42° C. and the amount of coagulum was practically equal.

The titration of samples from both specimens shows the same degree of acidity. The total nitrogen in 5 c.c. of each sample was found to be identical in both cases. This, the author says, proves to him that muscle rigor is independent of protein coagulation.

SECTION II.

PATHOLOGY.

INTRODUCTORY.

Few workers in pathology have devoted much of their time during the past year to questions of structural pathology. In consequence of that fact, not much has appeared in the medical literature on pathologic anatomy or pathologic histology. In the German journals, prolific in every field, there is still a fairly considerable proportion of the literature devoted to alterations in structure as seen with the naked eye and with the microscope. In American and in English journals, questions of pathologic chemistry, vegetable and animal parasites and their products have largely displaced articles on pathology of structure.

Pathologic anatomy and histology are effects, and search has gone nearer the fountain head. In times past symptomatology was the ultima thule; then came Morgagni, and later Virchow, and in the train of the latter a medical public sentiment that demanded that the structural basis for symptoms be investigated. Now the question is, what is responsible for the structural changes? Bacteriology was just such a study. But between bacteriology and pathology there remained a gap. Much of the recent work has been in an effort to fill this gap, and at the same time solve similar questions. For example, the relation of yellow fever to Sanarelli's bacillus has been cleared by the failure of yellow fever serum to agglutinate this bacillus. The paracolon and paratyphoid fevers would still be unknown country were it not for our knowledge of the

agglutinating properties of sera. Hand in hand with the multiplication of information on questions of this character go data which have led and which will lead still further to specific therapy. This phase of the year's work it will be difficult to classify: some part we have considered under physiology, and some part under bacteriology. This does not mean that nothing more remains to be found in structural pathology. With the old methods and procedures not very much was left besides statistical studies. But the present investigations are furnishing newer methods—are throwing light from different angles.

In every bacteriologic process two factors are concerned: the invading cause, and the host. The host impresses his personality on the structural findings of the resultant disease, and also on its chemistry. The study of the host factor in etiology and pathology has been relatively fruitless. But new methods, new facts and new use of old facts are becoming available for this purpose. The larger application of physics and chemistry to pathology is bearing fruit. In a few years structural pathology will be better studied and better understood by reason of its temporary neglect.

In addition to the epoch-marking work on the chemistry of bacteria, and of physiologic chemistry in general, much has been done of value during the year on parasitology. The work on animal parasites has been of great value. The discovery of the wide distribution of uncinaria, and in lesser measure of strongyloides, is of major importance. It is probable, as Stiles thinks, that many of the malarial cachexias are in reality uncinaria infections. The "pot-bellied dirt eaters" amongst the "poor white folks from the sandy lands" are an economic as well as a hygienic equation. It is too early yet to know of what importance trypanosoma infections may be in this country. In any event they will serve to direct our attention still more to the field of protozoa.

Probably the best piece of purely scientific work of the year was the probable discovery of a protozoon cause for

yellow fever. Yellow fever is an American problem, and the lasting work in its investigations has been done by American investigators.

The Shiga bacillus seems to be a world-wide parasite. Its relation to infantile diarrheas promises to clear a tangled question.

This sketch would not be complete without reference to two forces that are producing results in pathology in America. The first of these is the Rockefeller grants; the second the work of the Public Health and Marine Hospital Service. The American mind is broad and far-seeing, and has a "nose" for values. By reason of these admirable qualities it has always neglected certain fields. Just these deficiencies are being eliminated by these grants and by the work of the Public Health Service. The significance of these forces is greater than the mere discoveries that may result. The people are coming to know that to discover an antitoxin is of more importance, commercial and otherwise, than to discover a star, build a railroad, or buy an island.

METHODS.

Ultra Microscopic Objects. Siedenkopf and Zsigmonty[1] announce the discovery of a procedure by which objects too small to be seen by older methods come within the range of microscopic vision.

The method used is to use a heliostat to throw a ray of bright sunlight into a darkened room. The heat rays are eliminated by quartz lenses. The intense cone of light is brought to a point on the field of the microscope. The light is directed at right angles to the microscope. The illumination of the barrel of the draw tube is by radiation from the object. The most powerful combinations of the best lenses in use before this discovery did not allow us to see objects less in size than 0.5 mikron. It is claimed that

(1) Annales d. Physik, 1903. Quoted by Medicine Aug., 1903, and Journal American Medical Association July 11, 1903.

this will allow us to see objects that are .005 mikron in diameter.

Both the Journal of the American Medical Association and Medicine approve of the suggestion of Mouton[1] that this principle can be made use of in discovering bacteria. As is understood, the principle involved is not increased magnification, but is refraction. The light that it will furnish will be of aid in determining the presence of bacteria; not much of detail of structure can be added.

There is the possibility that the same principle can be made use of with lower powers in the study of larger objects. Such an attachment might differentiate different parts of the same cell, or different cells from each other. By this method of study, some of the necessity for staining tissues and fluids may be removed.

A New Blood Stain. Laporte[2] has described a stain for blood which is a modification of the Jenner method, and should give a better nuclear staining. He uses two solutions. The first a .5 per cent solution of Jenner's powder (Grubler) in wood alcohol (Merck's highest purity); this is not to be filtered. Second. Unna's polychrome methylene blue solution is to be diluted with 150 parts of distilled water. To the blood spread, without any heating, about 5 drops of solution No. 1 is added. This is allowed to act for one minute. To this he then adds 10 drops of solution No. 2; the two fluids are made to mix on the blood spread. This is left on for five minutes; it is then washed off with distilled water and the water is allowed to remain on the specimen for one minute. It is then carried into a dilute acetic acid solution (1 drop of 50 per cent acetic acid in 10 ounces of water). When the spread becomes reddish in color, it is washed in water and dried in the air. No heat is to be applied and no filter paper.

Results. Red cells a pale rose; all nuclei a carmine purple; eosinophile granules a bright coppery red; mast-

(1) Bulletin d. l'Institute Pasteur, 1903, 197.
(2) Medical Record, June 27, 1903.

zellen granules, metachromatic blue; protoplasm of lymphocytes pale light blue; bodies of the blood plates light blue; body of malarial plasmodia bright blue. Granular degeneration of red cells, fine blue specks.

Whitney[1] has made use of a combination of pyronin and methylene green for staining specimens which for one reason or another do not have to be treated with alcohol after being stained. Pappenheim in his work on plasma cells first suggested this stain, but Whitney is responsible for this use of it. A 1 per cent distilled water solution of each stain is made. At the time of use they are to be mixed in the proportion of 4 parts of pyronin to 1 of green.

Application. Pus, Gonorrheal. Smear and dry as usual; pour on the stain; heat; wash thoroughly. The nuclei of the cells are bluish-green. The neutrophilic protoplasm is unstained. Lymphocytes, mast cells, endothelial cells and epithelial cells, each stains to a different degree of purple. The bacteria stand out brilliant red in sharp contrast.

Blood. This combination used in this way does not stain red blood corpuscles, and therefore, if we wish to show the cells, Whitney suggests that the process be modified as follows:

Fix either with heat, ether-alcohol, wood alcohol or Zenker nitric acid. Stain with 1 per cent aqueous solution of orange G. Wash well. Stain with the pyronin methyl green mixture.

The red corpuscles will be a light yellow; the leucocytes will stain as indicated above, except that the protoplasm of the neutrophilic leucocyte will stain yellow with red granules.

As a Counter-Stain for Gram. After the usual procedure, stain with a 1 per cent watery solution of pyronin for one or two minutes; the contrasting elements will be a bright red.

Fresh Tissues, such as scrapings, membranes, etc. Add

(1) Boston Medical and Surgical Journal, May 7, 1903.

a drop of the mixture to the fluids or to the scraping suspended in water. Stir well with needle. Draw off excess with filter paper; wash with water, using a filter paper for drainage.

Frozen Sections can be stained with this mixture and mounted in glycerin. They do not hold their color. Whitney has never succeeded in mordanting the stain with resorcin, as Pappenheim suggested.

New Method of Hemo-Alkalimetry and a New Hemo-Alkalimeter. The principle of this new instrument devised by Dare[1] is the use of a spectroscope as an indicator. When blood is examined with a spectroscope, it is found that the hemoglobin bands disappear when the blood is neutralized. Dare adds his acid solution, watching for the disappearance of the bands with a spectroscope.

The instrument consists of a specially graduated mixing tube, in which 20 cubic millimeters of blood is mixed with a standard solution of tartaric acid. With a hand spectroscope the point at which the bands disappear is noted, the amount of tartaric acid is read off, and the degree of alkalinity is calculated and expressed in figures as milligrams of NaOH.

Funke made 75 determinations with this instrument from which the following conclusions can be drawn: In typhoid, alkalinity is decreased. In the primary anemias, alkalinity falls lower than color index. In chlorosis it keeps pace with it. There seems to be relation between alkalinity and color index.

A New Coagulometer. Evans[2] has devised a modification of the Wright coagulometer. It consists of a small copper bucket wrapped in some poor conductor of heat. The bucket is 8 cm. in height and 6 cm. in diameter. It holds 150 c.c. To the cover of the bucket a cell 7x2.5 cm. is attached. This cell is lined with felt. It is equipped with a dozen glass tubes; these are 5.5 cm. long and .3 cm. in diameter and have a lumen of .025 cm. The

(1) Johns Hopkins Hospital Bulletin, July, 1903.
(2) Read before Chicago Med. Soc'y, 1903.

bucket is filled with warm water, the tubes are placed therein. The finger is properly prepared and puncture made. Blood is rapidly drawn into six or more tubes, and these are instantly placed in the cell within the bucket. Six tubes can be thus filled in 15 seconds. In 2 minutes one tube is removed and the blood is blown into a porcelain dish; this is repeated every half minute or minute until the blood is found to be clotted.

The advantage of this over the Wright coagulometer consists in the greater uniformity of the temperature and the greater speed with which it can be used, each a point of considerable importance in this procedure.

Coagulation Time of Blood. Pratt[1] has studied the variation in the time of coagulation, largely from the standpoint of accidental influences. He made use of the apparatus of Brodie and Russell,[2] or rather of a modification of that apparatus devised by himself with the aid of Grützner. The Brodie and Russell method consists in directing a current of air against a drop of blood suspended in a moist chamber. The resultant flow of the corpuscles is watched under the microscope. When coagulation begins, movement ceases. Pratt found that the time varied in the same blood according as the blood was drawn directly from a vein into a glass tube, or was from a deep wound, or a shallow wound, or was pressed out. The time varied from 2 to 9½ minutes. Pratt is of the opinion that for these reasons, estimation of the time of coagulation is not of value. He concludes that the third blood corpuscles or blood plates have little or no relation to coagulation, and that estimation of their number is both difficult and valueless. He, however, inclines to believe that they are not derivatives of the red blood cells nor of the leucocytes.

A Modification of the Widal Test. Wolff[3] has suggested the following modification of the Widal test in the diagnosis of typhoid fever. By the use of his method he has been able to demonstrate typhoid bacilli in the feces

(1) Journal of Medical Research, August, 1903.
(2) Journal of Physiology, 1897.
(3) American Journal Medical Sciences, April, 1903.

very early in the course of the disease. He proceeds as follows:

A loop of the feces is smeared on an agar slant. From the growth, one or more bouillon cultures are made. The bouillon must be 1 to 2 per cent alkaline, as shown by N/10 acid, using phenolphthalein as an indicator. These bouillon cultures are allowed to grow for 12 hours. It is now treated with blood from the patient and from known typhoid subjects. If groups of the bacilli in the culture are agglutinated by known typhoid blood, it proves the presence of typhoid bacilli. Colon bacilli are unaffected.

Controls can be made by agglutinating with the patient's own blood serum; but this cannot be made use of until the second week, as prior to that time the serum will not agglutinate.

The author believes that the statement that typhoid bacilli do not appear in the feces until the eleventh day is inaccurate. While the 36 cases in which he has applied the test are too few for final conclusions, he thinks that the test gives promise.

Drigalski and Conradi[1] offer a culture medium on which typhoid bacilli will grow with so much greater rapidity than will other bacilli, that they can be differentiated. The medium is made as follows:

First Solution. Take 3 pounds of chopped beef, 2 liters of water, 20 grams peptone, 10 grams salt, 20 grams nutrose, 60 grams agar. Make a solution according to the usual plan.

The second solution is 30 grams of milk in 260 c.c. of litmus solution.

Add No. 2 to No. 1 and render alkaline. Then add 20 c.c. of a .1 per cent solution of crystal violet B. On this medium, typhoid bacilli grow with great rapidity.

Test for Albumins in Urine. Lindsay and Gies[2] report comparative tests with Pollacci's[3] reagent for albumin testing. This reagent is composed of

(1) Zeitschr. f. Hygiene u. Infc. Krank, 39-283-1902.
(2) American Medicine, Jan. 31. 1903.
(3) Schweiz. Wochenschrift f. Chemic. u. Pharm. XI., 1901, p. 168.

(a) 1 gram tartaric acid.
5 grams mercuric chlorid.
10 grams sodium chlorid.
Dissolved in 100 c.c. of water.

(b) Solution *a* plus 5 c.c. 40 per cent formalin.

They find the reagent very delicate, but it is not a differential test for albumin, as other proteids are coagulated by it, such as globulins, proteoses, mucinoids, mucus proteids, and even gelatin. They conclude that it is a delicate test for various proteids. The test is too delicate for ordinary clinical purposes, since normal urinary proteids may be mistaken for albumin. The reagent has no advantages over the fluid proposed by Spiegler. The reagent is applied by the contact method.

Urea. Long[1] uses the following method for urea determination: With mercuric nitrate 25 c.c. of urine are mixed with an equal volume of baryta solution, and filtered. Of the filtrate, 20 c.c. are taken for the titration. This must be neutralized with dilute nitric acid; a piece of litmus paper is placed in the filtered fluid, and with a glass rod the amount of nitric acid is added. Titration with standard mercuric nitrate solution is then made. This solution is so adjusted that 20 c.c. correspond exactly to 200 mg. of urea in 20 c.c. The end reaction is determined as follows:

Spread a number of drops of normal sodium carbonate solution (53 gm. Na_2CO_3 to the liter) on a glass plate with dark background, and as the end of the titration is neared bring a drop of the mixture in the beaker alongside one of the carbonate drops, and cause the two to mix on one edge. Part of the beaker drop is thus drawn over the carbonate, giving a precipitate; as soon as this appears distinctly yellow, consider the process finished and dilute the mercury solution so that exactly 20 c.c. should be required to produce this reaction, it being assumed that the standard solution in the beaker contained exactly 200 mg. of urea in 20 c.c.

(1) Journal American Medical Association, May 30, 1903.

The following *deductions for ammonia, uric acid and creatinin* were determined by the writer's experiments:

6 mg. of ammonia in 20 c.c. require 1 c.c. of mercury sol.

7.5 mg. of uric acid in 20 c.c. require 0.15 c.c. of mercury sol.

14.0 mg. of creatinin in 20 c.c. require 0.85 c.c. of mercury sol.

A valuable table, showing the amount of urea corresponding to the mercuric nitrate, is given.

Tests for Sugar in Urine. Vasey,[1] appreciating the difficulty of determining accurately the turning point of Fehling, has modified Fehling's method as follows: To 10 c.c. of Fehling properly diluted, he adds 2 teaspoonfuls of finely powdered calcium carbonate, or barium sulphate. This is raised to the boiling point while gently stirring with a glass rod. The urine is run in. The red suboxid of copper goes down with the powder, and the fluid above is not contaminated.

Riegler's Test for Sugar. Riegler's[2] test for sugar is performed as follows: Twenty drops of urine are placed in a large test tube. To this is added 10 c.c. of water and as much phenyl hydrazin oxalate as can be taken up on the point of a knife blade. Boil until all is dissolved. Shake well while boiling. Then add 10 c.c. of 10 per cent solution caustic potash. Stop the tube well and shake. If sugar is present, the fluid turns purple in less than a minute.

To make oxalate of phenyl hydrazin, dissolve 20 grams of phenyl hydrazin hydrochlorate in 300 c.c. of water with heat. Then add a solution consisting of 10 grams of ammonium oxalate in water. Cool, collect the crystals on a filter.

Safranin Test for Sugar. Christopher[3] has made an extensive study of the excretion of sugar in the normal urine, especially in children. The point that he wished

(1) The Lancet, June 20, 1903.
(2) Deutsche med. Wochenschrift, April 9, 1903, s. 266.
(3) Journal American Medical Association, June 20, 1903.

to determine was whether certain people who are physiologic so far as the ordinary conditions are concerned, excrete larger quantities of sugar than do other people. He also addressed himself to the determination of variations in the amount of sugar at different periods of life, and especially at those periods when the body is under considerable developmental stress. It is to be understood that the sugar excretion referred to is physiologic, and it became necessary for him to make use of some reagent which was more delicate than the copper or polariscope tests. A reagent which he made use of was safranin, and the method employed was that of Allen, somewhat modified.

Method: Take 6 to 12 test tubes. Into each put 1 c.c. of urine and 1 c.c. of normal sodium hydrate solution. To these add a 1 to 1,000 watery solution of safranin. In tube I put 1 c.c., in tube II, 2 c.c., and so on. These are then placed in glycerin in a beaker, and so carefully heated that not a bubble forms. If sugar is present, the mixture loses its color. Under no circumstances can the tubes be agitated, lest the safranin oxidize and the color return.

The index is the amount of safranin decolorized. For example, if 1 c.c. of urine decolorizes 5 c.c. of safranin, the index is 5. In Christopher's opinion, nothing found in the urine decolorizes safranin except glucose, and possibly some of the related acids like glycuronic acid. As the result of examination in which he had the assistance of Eichberg and Herzog, he concludes that as a test for sugar in urine, the safranin test is more delicate and more reliable than Fehling. An index of 5 was about the smallest amount that would react with Fehling. The normal index for children is 1.81; a child with 2, or over, Christopher thinks has a saccharin diathesis. He cautions repeatedly against conclusions drawn from a single examination.

Acid Index of Urine. In the article above quoted

Christopher[1] lays some stress upon the acid index. It is desirable that some acid index should be more widely accepted than is the condition of affairs at the present time. The index proposed by Christopher is the number of c.c. of N_{10} soda solution required to neutralize 10 c.c. of urine, using phenolphthalein as an indicator. It would seem, however, that it would be better to multiply this by 10, and in this way get a result figured on the basis of 100, the accepted basis of percentage estimations.

Chemical Test for Adrenal Tumors. Croftan[2] has devised a rapid chemical means of differentiating hypernephromas from other tumors. It is based upon the diastatic capacity of the adrenal gland. The method is as follows: A small quantity of the tumor is ground with sand and extracted with about five times its volume of water. This is filtered through gauze. Two test tubes of a watery solution of starch are taken; to one some hypernephroma extract is added; now a drop or two of an alcoholic solution of iodin is added to each tube. Each tube turns blue, but the one containing the hypernephroma is rapidly decolorized.

Staining of Sputum. Smith[3] recommends the following method: As thin a cover glass preparation as possible should be made from the sputum by means of a sterile platinum loop. This thinness is the more essential if the sputum is at all bloody, as is so frequently the case in pneumococcus pneumonia. Fix the preparation in the ordinary way, by passing two or three times through the flame. Cover the specimen thus fixed with anilin oil gentian violet, holding it well above the flame, and allow it to steam. Avoid burning.

Wash off the excess of anilin oil gentian violet with IKI solution; cover with IKI solution, and allow to steam as before. Decolorize as much as possible with 95 per cent alcohol. Wash a few seconds in alcohol-ether solution. Wash in water. Stain a few seconds in saturated aqueous

(1) Journal American Medical Association, June 20, 1903.
(2) Journal American Medical Association, Jan. 10, 1903.
(3) Boston Medical and Surgical Journal, Dec. 18, 1902.

solution of eosin. Wash off excess of eosin with Loeffler's blue; cover with Loeffler's blue, steaming as before. Decolorize slightly with 95 per cent alcohol; wash in absolute alcohol; follow with xylol; mount in Canada balsam and examine with an oil immersion lens.

The appearance of a specimen stained by this method is as follows: the protoplasm of the leucocytes, lymphocytes and other cells takes the eosin stain, as do the red blood corpuscles; the nuclei of the cells stain with the Loeffler's blue. Eosinophiles appear as in stained blood preparations. Gram staining organisms appear black or deep violet, while Gram decolorizing bacteria take the Loeffler's blue stain. Capsules about organisms, when present, are stained by the eosin, as are ciliæ.

Elastin Staining by Weigert's Method. Fisher[1] calls attention to the value of Weigert's method:

In showing advance of a tumor and breaking through the elastic fibers of the tissue.

It also helps to outline the tissue.

It is of advantage in showing the breaking of malignant growths into vessels.

In determination of tissue element where extreme disintegration has occurred.

A Rapid Method for Hardening and Embedding Tissues. Bolton and Harris[2] fix and imbed tissue at one step by the following method: The fresh tissue is placed at once in a hot 2 per cent solution of agar to which 10 per cent formalin has been added. This is kept at 70° C. for one to several hours. It is then attached to blocks with 5 per cent agar to which 10 per cent formalin has been added. The blocks are placed in 95 per cent alcohol for two to four hours; they are then ready to cut.

Freezing Agent. Katz[3] recommends the use of anasthol, sold by Speier and Kasper, Berlin, as having great advantages over ethyl chlorid in the preparation of frozen sections of tissue. The tissues are placed for a short

(1) Muench. med. Wochens. No. 43, 1902.
(2) American Medicine, May 23, 1903.
(3) Deutsche med. Wochens., June 11, 1903.

time in formalin and then fastened to the microtome and cut under the freezing action of the anasthol. This substance is cheaper and more volatile than ethyl chlorid. It is also sold in tubes.

Romanowski's Stain for Tissue. Feinberg[1] uses this stain as a tissue stain, especially for tumor sections. Following his studies of blood parasites by use of this stain, he concluded to test it on tissue specimens. In order to accomplish this he found that alcohol alone must be used for fixation and hardening. In order to dehydrate after staining, it was found that alcohol was entirely unsuitable because the colors were partially dissolved and caused simply a diffuse staining. To avoid this, the author uses ether and follows with xylol and mounts the sections in balsam. Sections stained in this way show distinct color differentiation of protoplasm and nuclear structure. The tissue cells of tumors show (1) the plasma stained blue; (2) the nuclear structure as also the nuclear membrane, reddish-violet, and (3) the nucleolus intense blue. This was found general. Comparing these results with the staining effect of the reagent on blood parasites, the marked difference in the effect on the nucleus is noted. In these this structure is red. The author finds bodies staining in this latter way in tumor sections and considers them as parasitic organisms. These bodies have a red stained point at about their center, around which in many instances there is a clear space, while the rest of the structure is stained blue.

Museum Specimens. Littlejohn[2] has made use of a dry method for the preservation of anatomic specimens for the last five years. The experience of these years convinces him that the method has advantages which are not offset by its disadvantages. The advantages are that the cost of the specimen is materially less because of the lessened cost of the container and of the fluids; that the specimen is less heavy and therefore more easily handled; that the element

(1) Berliner klinische Wochenschrift, No. 45, 1902.
(2) Journal of Pathology and Bacteriology, September, 1902.

of spoiling from evaporation does not have to be contended with.

He prepares his specimens by Kaiserling's method. After the methyl alcohol treatment, he soaks them for from one to four weeks in 50 per cent glycerin and water, to which a little formalin has been added. The specimen is then placed in a glass jar or box, a little glycerin is poured over it or placed elsewhere in the box, the glass cover is placed on and fastened hermetically, preferably with putty. As is the case with all museum specimens, and especially those in which an effort is made to preserve the color, it is necessary that these specimens be kept in the dark. Littlejohn has some specimens prepared in this fashion that he has had for five years; they have retained their color and their moisture.

The Melnikoff-Razewednikoff method for the preparation of museum specimens is as follows:

1st step—In

Formalin	10.
Water	100.
Potassium chlorate	0.5
Potassium acetate	3.

2d step—

In weak alcohol.

3d step—

In 95 per cent alcohol.

4th step—

Finally, after a few days, in

Potassium acetate	30	by weight
Glycerin	60	
Water	100	

The method of Riche and Gothard for the same purpose, is as follows:

1st step—In

Formalin	150.
Aq. dist	1000.
Potassium nitrate	10.
Potassium acetate	30.

The specimen is to remain in this one or two days, then transfer to

2d step—

80 per cent alcohol. After 12 hours.

3d step—

To 95 per cent alcohol, in which it remains 2 hours. It is then kept in

Glycerin	100.
Aq. dist	100.
Potass. acetate	30.

To preserve anatomic specimens with permanent retention of their color, Claudius[1] employs the following method: The specimen is put into a jar which is about one-third full of a saturated, watery solution of ammonium sulphate. The jar is securely sealed and illuminating gas is run in. By this method, the hemoglobin of the specimens is converted to a carbon-monoxid hemoglobin, in which condition it will retain its color indefinitely.

Preservation of Sediments. In order to preserve the sediments of urine, sputum and other clinical material, Rohnstein[2] employs the following means: Urine which is rich in sediment is allowed to settle in a conical glass, a few crystals of thymol having been added. The supernatant fluid is siphoned off, water is added to the sediment, the mixture is thoroughly shaken and set aside again

(1) Hospitalstidende, Copenhagen. Abstract in Journal Am. Med. Assoc., 1903, Vol. XLI.
(2) Fortschritt der Medicin, XX, No. 2, p. 41.

with the addition of a few grains of thymol. After the sediment is settled, remove the supernatant fluid and add at least twice as much 2 per cent formalin as there was sediment. This is thoroughly mixed and again sedimented. Pour off half the fluid and add an equal amount of the following mixture:

Formol	20
Glycerin	125
Distilled water	200.

If the urine contains but little sediment, he modifies his method of procedure as follows: A filter paper is properly placed in a funnel and washed with a little water. Through this he filters a considerable quantity of urine. The sediment is washed by filtering two waters through it. Now the filter paper is removed and the sediment adhering to it is washed off with a 2 per cent formalin solution. This is then sedimented, the formalin solution is drawn off and the formol-glycerin solution, indicated above, is added.

[The Editor cannot see why the same results could not be more expeditiously accomplished by making use of centrifuged specimens.]

Gastric Contents. The stomach contents are filtered until about the thickness of porridge. This is then mixed with an equal quantity of the glycerin formol mixture above indicated.

Sputum. The sputum is to be filtered until it is as thick as gruel. The portion which has not passed through the filter is then mixed with an equal quantity of the formol glycerin mixture.

Feces. The method to pursue with feces depends upon the degree of concentration. If the feces are quite fluid, the water is to be filtered out until the remainder is about the thickness of porridge. This is then diluted with formalin and glycerin. Should the consistence not demand the filtration, the mixture of formalin and glycerin can be added as the first step in the procedure.

It is needless to say that these sediments are to be kept in well stoppered vials.

LABORATORY DIAGNOSIS.

Practical and Scientific Value of Bacteriologic Examinations of Blood During Life. Hektoen[1] thinks that bacteriologic blood examinations have proven of value both scientific and practical, in the septicemias, and especially in pneumonia, typhoid and paratyphoid fevers.

Perhaps it would be wise to describe the usual procedure at this point, even though it be fairly well known. The only apparatus required is any syringe holding a half ounce. Hektoen prefers a glass syringe (Luer patent). The proper culture medium is about 5 flasks of bouillon. For every culture except pneumococcus, each flask should contain 100 to 150 c. c. of bouillon. Rosenow thinks that pneumonia grows better with a smaller quantity of bouillon. Any prominent vein will answer; the one usually employed is the median in the elbow. The skin is to be sterilized with soap and water and alcohol. The vein is compressed above. The syringe point punctures with a quick slanting stroke, and about 15 c. c. of blood is to be drawn. The pressure on the vein is to be relieved just before the needle is withdrawn. One c. c. of blood is then mixed with each flask of bouillon and the flasks are incubated. At the end of 24 hours the bouillon clouds if a bacillus is growing. If we are examining for typhoid or paratyphoid, we test the bacillus with known sera, according to the Widal method. If we are examining for other bacteria, we examine in the usual way.

Hektoen believes with Petruschky[2] and Bertelsman[3] that in septicemia the finding of bacteria in the blood is of no prognostic value. In pneumococcus infection there may be no pulmonic localization, sometimes there is no local manifestation anywhere. In these cases blood examination alone is diagnostic. Bacilli in the blood in pneu-

(1) Illinois Medical Journal, July, 1903.
(2) Zeitsch. f. Hyg. u. Infekt., XVII., p. 59.
(3) Verh. d. Geschell. f. Chirurgie, 1902, xxxi.

monia have no prognostic significance. In typhoid, Kerr and Harris have shown that blood examination in the first week is one of the best diagnostic agents. It was their opinion that bacilli in the blood was of no prognostic significance. Schottmüller and Rudiger think that the finding of bacteria in the blood is of some prognostic significance in typhoid, especially late in the disease.

Longcope[1] and Lion and Negel[2] describe paratyphoids without intestinal lesions. Blood examination is a necessity in such cases.

In a communication to the Pathological Society of Philadelphia, Rosenberger[3] gave a statistical study illustrating in very graphic fashion the *value of bacteriologic blood examinations in various diseases.* In a collection of 535 cases of typhoid fever, positive results were secured in 80 per cent of the cases. This, of course, is additionally valuable by reason of the fact that the typhoid bacilli are found more readily in the blood in the first few days of typhoid than at any other time. These are the days when the serum reactions are of but little service. He reports 46 per cent of positive results in 176 cases of sepsis; 53 per cent of positive results in 58 cases of pneumonia; and 69 per cent of positive results in 423 cases of plague. In this connection it is proper to note that Woolstein was not able to get cultures from the blood in her cases of infantile diarrhea.

Rosenow[4] has *studied the blood by culture and smear methods in 83 cases of pneumonia.* One case was examined 12 hours after the initial chill, the others at various times up to the 24th day. He found the pneumococcus in 77 cases. In two cases he was able to find the diplococcus in smears, but not in cultures. His earlier work was done with higher dilutions.

Pneumonic Endocarditis. Wells[5] believes pneumonia to be a frequent cause of endocarditis. Of 517 fatal cases of acute endocarditis, 22.3 per cent were associated with

(1) American Journal Medical Sciences, 1902, cxxiv.
(2) Centralb. f. Bakt., 1902, xxxii.
(3) American Journal Medical Sciences, June 20, 1903.
(4) Medicine, June, 1903.
(5) Journal American Medical Association, Oct. 18, 1902.

pneumonia. In 1,213 fatal cases of pneumonia, endocarditis was reported as present in 4 per cent. This figure, Wells thinks, is far below the truth. In 40 fatal cases the mitral alone was involved in 14, the aortic alone in 10, the mitral and aortic in 7, the tricuspid in 2; the tricuspid alone in 2; the pulmonary alone in 2; the left valves in 1.

The pneumococcal lesion is usually a massive vegetation, with a marked tendency to ulcerate.

Gonorrheal Endocarditis. Harris and McLeod[1] reported a case of gonorrheal septicemia in which there was an atypical murmur, a terminal partial consolidation of the lung, and a violent secondary anemia. The first four bacteriologic examinations were negative. In each of these tests 12 c. c. of blood was drawn. On two occasions this amount was placed in four flasks of hydrocele bouillon in the proportion of one part blood to fifty of bouillon. On two occasions 12 c. c. of blood was plated with 25 c. c. of agar. Successful cultures were made at the fifth effort. This was 24 hours before death. The post mortem showed very large vegetations on the mitral cusps. Elsewhere the endocardium was smooth. There was a splenic infarct. The kidneys showed parenchymatous nephritis. The lower urinary organs showed no trace of the original infection. In the alveoli of the lung were some masses of fibrin showing beginning organization and partial covering of the bands with epithelium. Harris and Johnstone are of the opinion that a moderate quantity of blood diluted with a small quantity of plain agar and plated is the best method. A large percentage of blood and plenty of exposure to air seem to be required. They quote Prochaska[2] as having had a similar experience with pneumococcus. They give Longcope's method for pneumococci as one part blood to 2½ parts agar in plates. Using this method, Longcope is able to get a culture from the blood in nearly every case of pneumonia.

(1) Johns Hopkins Hospital Bulletin, Oct., 1902.
(2) Deutsche med. Woch., May 22, 1902.

Bacillus Aerogenes in the Blood. Cole[1], in examining a case of crushing injury in which emphysema developed, found *Bacillus aerogenes capsulatus.* He then examined blood taken from a vein situated in another part of the body than that in which the gas was found in the tissue. About .5 c. c. of blood was placed in a tube of litmus milk. Several of these tubes were incubated anaerobically and *Bacillus aerogenes* grew abundantly. Twenty-five c. c. of the blood was run under a column of water and kept under observation for a few minutes. No gas was seen to escape.

Cole concludes that however much of a gas producer *Bacillus aerogenes* may be, it can grow in the blood stream without producing any gas.

Paratyphoid and Paracolon Fevers. As greater accuracy in diagnosis in cases of typhoid fever is secured by the Widal reaction and bacteriologic examination of the blood, it becomes apparent that there are fevers somewhat like typhoid in their general history which are due to infection with other bacilli of the colon group. That such is the nature of the so-called "third fever" of the South is suggested by Happel, who, however, merely offers this as a surmise. The question is of academic rather than of practical interest in all probability, since the hygienic precautions that are necessary in typhoid fever are just as necessary in this set of fevers. It is unfortunate that the possibility that a given infection was of this nature has been responsible for a general hygienic laxness in some instances. DeFeyfer and Kayser[2] reported an epidemic at Eiberg which consisted of fourteen cases. The course was that of a mild typhoid. Occasionally there were minor intestinal hemorrhages. The blood never caused any agglutination of the bacillus typhosus. It did not agglutinate the paratyphoid bacillus of the type B of Schottmüller. In the cases collected by Schottmüller, Kurth and Brion and Kayser, the type Schottmüller B

(1) Johns Hopkins Hospital Bulletin, Oct., 1902.
(2) Münch. med. Wochenschr., 1902, Vol. XLIX.

was by far the most common of the two types of paratyphoid bacilli known to cause fever.

H. W. Allen[1] adds three new cases of paratyphoid in which the bacillus was quite well worked out. Two of the cases would be classed clinically as mild typhoid. The third case would have been mild had it not been for an infection of the gall-bladder requiring operative procedure. The serum of each agglutinated typhoid in dilution 1:10, but failed where the dilution was greater. They each agglutinated a culture of the other two, and two agglutinated a culture from Gwyn's case, and also one from Johnstone's case. Allen does not enter upon the question of the type. The disposition to hemorrhage in these paratyphoid infections noted by Allen, and observed by Longcope, would indicate that hemorrhage cannot be regarded as a pathognomonic sign of typhoid fever. Possibly this opinion as to hemorrhage may permit a different interpretation of Libman's case.

J. W. Walker[2] reports the case of a physician who had a continued fever associated with more sweats and chills and with fewer abdominal symptoms than belong to a typical typhoid. There was a thrombus in a leg vein. The blood never agglutinated Bacillus Typhosus. The blood cultures were made by Harris and were studied by G. B. Smith.[3] The bacillus was clumped by serum from the patient, by serum from another case reported on by Smith, which we will designate as No. 2, and by serum from an animal made immune to Buxton's paratyphoid. It would not clump with hog cholera serum nor with enteriditis serum. Comparative gas measurements made with the different carbohydrate media, and also its growth in litmus milk, convinced Smith that it was paratyphoid of the group Müller of Schottmüller, and corresponded with Gwyn's, two of Johnstone's, Hewlitt's, Buxton's, and possibly Cushing's bacillus. Smith reports a second case: This did not give a Widal, was not clumped by typhoid,

(1) American Journal Medical Sciences, Jan. 3, 1903.
(2) Journal American Medical Association, Vol. xli.
(3) Journal American Medical Association, Vol. xli.

hog cholera, nor by enteriditis serums. The limit of the clumping with Buxton's serum was a dilution of 1:100, as contrasted with 1:2000 for the first bacillus. Its gas measurement did not correspond with that of Walker's case (No. 1) nor with Buxton's bacillus. This was another of the cases of paratyphoid in which there was hemorrhage. Smith has collected the following cases of paratyphoid with hemorrhages: Gwyn,[1] Strong,[2] Hume,[3] Feyfer and Kayser.[4] Rudiger,[5] examining thirty cases of clinical typhoid fever, found three cases of apparent paratyphoid. In No. 1 the patient's blood did not agglutinate the culture made from his blood in a dilution 1:200. It was not agglutinated when mixed with an immune serum made by immunizing a goat with typhoid. In the two remaining cases the failure to be agglutinated by typhoid serum proved that the bacilli were not typhoid, and the fact that the serum from the third case agglutinated the bacilli from all three cases proved that they were of the same type. Beyond that the question of types was not investigated.

Scarlet Fever. Weaver[6] examined the secretion from the throats of 18 cases of scarlet fever. The bacteriologic examination of the skin gave nothing, except that streptococci were occasionally found. In addition, there was a fair flora of air bacteria. He secured a rich culture of streptococcus from each of the 18 throats. He places some stress upon the necessity of removing the swab without touching the tongue, for swabs that have come in contact with the tongue always show contamination by the large saprophytic cocci found on that organ. Weaver was not able to discover any differences between the streptococci grown from the tonsils in scarlet fever, and other streptococci. As to the frequency of the finding of streptococcus, in addition to his own observation, he quotes

(1) Johns Hopkins Hospital Bulletin, 1898.
(2) Johns Hopkins Hospital Bulletin, 1902.
(3) Thompson-Yates Lab. 1902.
(4) Loc. cit.
(5) Transactions. Chic. Path. Soc'ety, Jan. 12, 1903.
(6) Transactions, Chicago Pathological Society, Feb., 1903.

Baginsky and Sommerfield as having found streptococcus in 695 out of 701 cases. Weaver could not find evidence that the Class coccus bore any relation to scarlet fever.

Weaver[1] investigated the *vitality of bacteria from the throats of scarlet fever patients,* with especial reference to streptococci. His study was made on 95 cases of scarlatina. He made 145 swabs; from 87 of the swabs he cultivated streptococci—from 58 he failed. He concludes that streptococci are almost always present in the throats of scarlet fever patients; the number is greatest in the earlier stages of the disease. The streptococci from scarlet fever throats resist death from drying as long as do any other bacteria; in one case this was 90 days. They live a long time in milk. A small amount of sugar increases the value of a medium for streptococci.

Forbes[2] makes a report on the *frequency and importance of diphtheria bacilli in the ear discharges of scarlet fever patients.* His report embraces 34 observations on 20 patients. These were cases of scarlet fever in which there was otorrhea. In some of the cases the otorrhea was so slight that there was no external discharge. The observations were made in the convalescent ward of Monsull Fever Hospital. Some of the cases had been in contact with faucial diphtheria. In all 20 of the cases, diphtheria bacilli were found. In two it was found in one ear, but not in the other. The bacilli were frequently in pure culture. In any event it is easy to recover the bacillus, as no other bacillus grows so rapidly on blood serum. He concludes that infection occurs through the nose. While he thinks that plugging the ear with antiseptic cotton and covering with a bandage makes the patient a fairly safe companion, yet such liberty is not wise for either the patient or the community. In none of his cases did a faucial diphtheria follow the otorrhea, however. The yearly statement of the hospital shows 1.4 per cent of cases of scarlet fever having symptoms of diphtheria. The bacilli

(1) Journal of Medical Research, May, 1903.
(2) Journal of Pathology and Bacteriology, May, 1903.

were found from the 2nd to the 61st day of the otorrhea in the cases examined by Forbes.

Otitis of Measles. Albesheim[1] reviewing the literature of this subject, says that Schribe has always found pyogenic bacteria. Streptococcus albus and aureus are present, in frequency, in the order named. Sweigerhofer, Moos, Zaufal and Netter had practically the same result. Wolff found pneumococci once. Williams and Councilman found diphtheria bacilli twice.

In Albesheim's 5 cases, he found, in one case, Streptococcus pure; in one, Staphylococcus albus pure; once, streptococcus with a bacillus; twice staphylococcus with a bacillus. In one of these cases he studied the bacillus and concluded that it belonged to the group of ungula.

Small Pox. Councilman, Magrath and Brinckerhoff[2] announce the discovery of *small, probably protozoan bodies,* that they believe to be the cause of variola. These bodies were found by them in the deep epithelial cells during the early stages of variola. The body is first seen as a small structureless mass, 1 to 4 mikrons in diameter. This increases in size, becomes reticular and granular and possibly ameboid. No nucleus was found. Segmentation occurs. This is the asexual cycle, and is to be seen on the edge of variola nodules. The epithelial protoplasm and nucleus do not seem much affected. About this time the sexual cycle begins in the nucleus as a small vesicle or ring body. These soon form several vacuoles arranged around a central vacuole. These enlarge and the nucleus disappears. Now this vacuole becomes a definite body containing multiple circular bodies with a central dot. The small circular bodies are 1.5 mikrons in diameter. Tyzzer, examining vaccinated rabbits and calves, found in the vaccinia nodules, the intracellular or asexual cycle only. In vaccinated monkeys, both cycles were found.

Councilman, Magrath and Brinckerhoff believe that the variola protozoon in all probability passes through the double cycle, the first cycle being that evolved in the proto-

(1) Archiv f. Ohrenheilkunde, Bd. liii. S. 87, 1901.
(2) Journal of Medical Research, May, 1903.

plasm outside of the nucleus and the second cycle comprehending the spore-forming body that is found within the cell nucleus. In vaccinia it is their belief that the organism proceeds through but one of these cycles, namely, the extra-nuclear. The protozoon is seen with great difficulty, not only by reason of its small size, but also by reason of its similarity to the host structures, and this is probably the reason that it has not been discovered by other workers in this field.

Schamberg[1] in an effort to solve the *etiology of small-pox* made cultures from 82 small-pox lesions. In 64 cases the results were negative. In the other 18, infection with Streptococcus pyogenes, with a bacillus possibly that of diphtheria, and with Staphylococcus aureus was found. He thought that these infections were accidental. In his opinion, the *suppuration* of variola is not due to a secondary infection. He quotes Ewing as having examined 23 cases in the same way. In 17 he obtained streptococcus pure, and the same coccus mixed with other bacteria in two other cases. Ewing thus found streptococci present in 87 per cent of his cases. Bergey, quoted by Schamberg, found streptococcus present in 5 of 12 cases. Both Schamberg and Bergey think that streptococcus is not of direct etiologic importance in variola. The writer found the Renault-Guarnieri bodies in the fluid of the vesicle in every case examined.

Cyto Diagnosis of Small-Pox.—Ferguson[2] has studied variola with a view to determining if there is a leucocytosis; if so, the relation of the varieties of leucocytes each to the other; and evidence, if any, in the leucocytosis bearing upon the etiology of the suppuration in the suppurative stage of variola.

He divided his cases into three groups:

I. Mild cases that had been previously vaccinated.

II. Grave cases terminating favorably.

III. Most severe cases (hemorrhagic or confluent), usually dying.

(1) *Jour. Am. Med. Assoc.*, Feb. 14 1903.

(2) *Journal of Pathology and Bact* 1903, p. 411.

In all, there is leucocytosis. This begins on or before the onset of the rash; in the mildest cases it is somewhat delayed. It attains its maximum in eight or nine days, after which it wanes. However, there is some leucocytosis throughout the disease. In Group III there is some leucocytosis, but as the case approaches death the leucocytosis subsides. The polymorphonuclears are about normal or slightly increased absolutely (decreased relatively). These show marked degenerative changes. The striking point is the increase in small mononuclears, occasionally going to 50 per cent. There is a slight large mononuclear increase. In severe cases there are neutrophilic and eosinophilic myelocytes. These sometimes mounted to 10 to 12 per cent. Eosinophiles were increased early, but decreased late, though this was not a uniform observation.

The author quotes Weil[1] and Courmont and Montagard[2], with whom he agrees, except that he has found more leucocytosis in the grave cases.

Cyto Diagnosis. A somewhat broader view of the relations of the cells in inflammatory exudations is given by Beattie[3], who has studied experimentally produced peritonitis. The results would indicate that the number of phagocytes and the prominence of the different varieties is dependent both upon the variety of the infecting microorganisms, and the length of time after infection. He concludes that the polymorphonuclears are the principal phagocytes. The eosinophiles have no phagocytic capacity. He thinks that the large mononuclear phagocytes are derived partly from the endothelial cells, partly from the large mononuclears of the blood current and partly from the lymph tissues. These cells are both ameboid and phagocytic. The plasma cells, which Mallory suggests are the source of the antibody in typhoid, Beattie thinks, are derivatives of the large mononuclears. It was Mallory's view that they came from the small lymphocytes.

Bunting[4] has studied the *question of pleurisy a frigore*

(1) Comp. Rend. Soc. de biol. Paris, June 23, 1901.
(2) Ibidem, June 30, 1900.
(3) Jour. Pathol. and Bacter'ol., Vol. iv., June, 1902.
(4) Johns Hopkins *Bulletin*, July 19, 1903.

from an etiologic standpoint. Incidentally he writes of cyto diagnosis as advocated especially by the French School. He dwells upon the insufficiency of the usual methods of determination of the causes of such pleurisies. Autopsy reports have to be taken *cum grano salis* because death seldom occurs until there has been large opportunity for intercurrent infections. An ultimate death from tuberculosis is not proof that Koch's bacillus caused the primary infection. The pleurisy may have paved the way for a later tuberculosis. Direct examination of the effusion, cultures, biologic tests, all are faulty. In the Practical Medicine Series for 1902, Vol. IX, can be found the Widal-Ravant formulæ for tubercular and other serous effusions. Bunting thinks that probably cyto diagnosis is of value. He found the fluids to have a specific gravity of about 1018. There was a high albumin percentage. At the end of the first week the effusion showed polymorphonuclears 85 to 88 per cent. During the second week the polymorphonuclears fell to 3 to 5 per cent. Such a showing indicates tuberculosis. In pus processes polymorphonuclears formed 80 per cent. In pneumonic pleurisies they formed 96 per cent. In 3 transudate cases the specific gravity was 1005 to 1015. Endothelial cells single and in placques were abundant.

Polymorphonuclears were few. Sweet[1] thinks that in tubercular cases, within the first 24 hours, the mononuclear forms constitute 10 per cent of the cells. In 3 or 4 days they will have risen to 30 or 40 per cent.

Weir[2] continues the work of Howard and Perkins[3] on the presence of eosinophiles in the tissues. The best routine method of fixing was in Orth's fluid. He usually stained with eosin and hematoxylon, carbol toluidin blue or polychrome methylen blue. He found large numbers of eosinophiles in the tissues in acute and subacute inflammation of the pelvic viscera. In the mucosa of the

(1) Univ. of Penn. Medical Bulletin Sec., 1902.
(2) American Journal Medical Sciences, Jan., 1903.
(3) Johns Hopkins Hospital Reports, Vol. X.

appendix, both normal and inflamed, large numbers of eosinophiles were seen. Weir accepts the statements of Howard and Perkins that eosinophiles can develop from smooth muscle fibers, from plasma cells, and from large mononuclear leucocytes. He has noticed plasma cells become faintly granular until the mast cell type was reached. Examination of the blood did not show circulatory eosinophilia in the cases of pathology in the pelvic viscera.

Pietrowicz and Daniels'[1] report a case of trichinosis. Their attention was directed to the case by a leucocytosis of 10,500 to 17,900 and an eosinophilia of from 21 to 37 per cent.

Capps[2] case of uncinaria was also accompanied by eosinophilia. Eosinophilia is fairly constant in people infected by intestinal parasites. However, it is not a *sine qua non.*

Investigations of the origin of leucocytes have been made by Brinkerhoff and Tyzzer.[3] It was Ehrlich's[4] idea that the granular leucocytes originate in the bone marrow, and the non-granular forms in the lymph system. Dominici[5] has shown that under certain abnormal circumstances granular forms can originate in the lymph system. Brinckerhoff and Tyzzer seemingly have shown that the so-called polymorphonuclear neutrophiles are produced in the bone marrow. That they do not spring from, or change to either the eosinophile or the hyaline forms. They give the cytology of the bone marrow as follows:

First—Undifferentiated cells,

Second—Cells of the erythroblast series,

Third—Cells of the leucoblast series, which are subdivided into: *a,* mast cells; *b,* eosinophiles; *c,* amphophiles or neutrophiles,

Fourth—Giant cells.

From the indifferent cells result the leucoblastic series, and from these the amphophilic series by changes, chem-

(1) Trans. Chicago Pathological Society, Jan., 1903.
(2) Jour. Am. Med. Assoc., Jan. 3, 1903.
(3) Journal Medical Research, Dec., 1902.
(4) Ehrlich & Lazarus Nothnagel Series, Bd. VIII.
(5) Memoire de la Societe de Biol., 1900.

ical and physical in the protoplasm, and in the nucleus. The myelocyte is an intermediate stage. The nucleus first elongates and then doubles. No mitosis ever occurs. New cells are supplied to the peripheral blood by differentiation and division in the earlier forms, and by wandering into the circulation, through chemotaxis, of the mature forms. An interesting point is that after the cells once reach the leucoblastic "fork in the road" they move on to become either eosinophiles or amphophiles. Muir[1] has shown that leucocytes that have wandered from the circulation do not return.

Shaw[2] removed the spleen of an animal and injected cinnamate of soda. He then made differential blood counts. As the result of his experiments he concludes that the neutrophilic leucocytes are produced from the hyalines. This probably occurs in the circulating blood though some of it may occur in the bone marrow. None of it occurs in the spleen.

The author thinks that probably eosinophilia is a less active form of neutrophilia.

The conclusions of R. Stockman and F. J. Chateris[3] are based on the study of bone marrow in some cases of arsenic poisoning resulting from beer drinking. Muir[4] has also reported some cases from the same group. The changes in bone marrow consisted in the disappearance of the fat, increased vascularity, and increased cellularity. The increase of the cells was in the leucoblasts. Late results of the arsenic poisoning were hyaline degenerations. The authors believe that there is nothing specific in the changes due to arsenic. The same changes were found in various poisonings, and in several emaciating diseases. They conclude that arsenic increases neither the hemoglobin nor the red cells of the circulating blood, and that such building up of the blood in conditions such as pernicious anemia, as is accomplished, is due to destruction of the morbific agent.

(1) Jour. Path. and Bact., Vol. vii., 1901.
(2) Jour. Path. and Bact., May, 1903.
(3) Jour. Path. and Bact., May, 1903.
(4) *Journal Path.* and Bact., Vol. VII., 1901.

Blood Differentiation by Means of Specific Precipitins. Austin[1] finds in certain experiments, he reports, that the differentiation of human blood from that of other animals can be made only within certain limits. Spontaneous precipitates must be recognized as a possibility, and can only be avoided by very careful control tests. He also notes the difficulties, as stated by earlier writers, in selecting a suitable solvent for the dried stain, and makes the point that the salt solutions may induce molecular changes in the hemoglobin present. A previously noted fact is also shown, in that an immune serum for a certain blood will also give a precipitum with serum from the same species of animal that was immunized. Thus anti-human rabbit serum may show a precipitum with human serum and also with rabbit serum. To avoid this, the antiserum in question should be prepared by using several animals. The writer found that serum preserved on filter paper did not retain its efficiency any length of time. This is quite contrary to the reports of a number of observers.

Contrary to the report of Myers[2] and others, Michaeles[3] *could not obtain an active serum for peptones or albumoses.* He concludes that the change from native albumin to these bodies destroys the side chain group that is present in the former. However, he makes the point that they may be acted upon by the specific serum, but do not precipitate after the manner of albumin.

In order to observe slight precipitations and also their early appearance, Robin[4] uses a *microscopic method.* Hanging drops made after the usual manner on concave slides are prepared from the serum to be tested. The serum in these drops must be clear. To some of them the antiserum is added, and the observation continued. Granular and refractive masses will be seen in the positive reactions.

(1) Boston Medical and Surgical Journal, March 12, 1903.
(2) Centralb. f. Bact., 1900.
(3) Deutsch. med. Wochens., No. 41, 1902.
(4) Philadelphia Medical Journal, Dec. 20, 1902.

Gall-Bladder Fluid in Cystic Obstruction. Sollmann[1] after a careful chemical and physical study of fluid in two cases, concludes: The contents of the human gall-bladder in cases of obstruction of the cystic duct, whether this is congenital or through a gall-stone, consist of a dilute solution of true mucin, of the molecular concentrations of the serum devoid of bile acids and pigments, of ferments and of sugar. Traces of coagulable proteid may be present. No nucleoalbumin was found. The gall-stones producing the obstructions consisted essentially of cholesterin.

Cerebrospinal Fluid. Sollmann[2] has collected data as to analyses of the cerebrospinal fluid. This table shows the following to be the mean and extremes of results:

	Specific Gravity.	Depression of Freezing Point.	Total Solids. Per cent.	Total Ash. Per cent.	Coagulable Proteid. Per cent.	Reducing Substance. Per cent.	Clorin as NaCl. Per cent.	Urea. Per cent.
HUMAN.								
Number of analyses.....	9	3	14	15	20	.....	11	2
Extremes.	1.006-1.0137	0.535-0.591	9.6-19 23	7.25-9.48	0.2-5.8	0.0-0.555	4.48-8.0	0.044-0.098
Mean..	1.0079	0.541	10.75	8.5	0.9	.....	6.6	0.071
Proportion of cases within 10 per cent of the mean..	6/9	3/3	9/14	13/15	5/20	.....	7/11	.....
Proportion of cases within 25 per cent of the mean..	8/9	3/3	12/14	15/15	8/20	.....	11/11	.....
DOGS.								
Number of analyses.....	.....	.....	8	.....	.....	.....	.....	.....
Extremes..	.....	.....	13.91-20.24	.....	.....	.....	.....	.....
Mean.....	.....	.....	17.5	.....	.....	.....	.....	.....
CALVES.								
Number of Analyses.	12	.....	12	12	12	.....	.....	.....
Mean.....	1.0076	.....	13.14	8.136	0.221	.....	.....	.....
HORSES.								
Number of Analyses.....	5	.....	.....	5	13	.....	.....	.....
Mean.....	1.0082	.....	.....	8.15	1.01	.....	.....	.....

Cryoscopy. Tinker[3] has investigated cryoscopic examination of the blood and urine as an indicator of the condition of the kidneys, and in relation to various pathologic conditions. He considers such examination of great

(1) American Medicine, March 14, 1903.
(2) Journal American Medical Association, June 6, 1903.
(3) Johns Hopkins Hospital Bulletin, June, 1903.

value. He makes use of the Heidenhain apparatus. This consists of a thermometer 45 cm. long, with a large bulb, and, in addition, a reservoir bulb at the upper end. It is graduated from + 1°C. to —4°C. and is marked so that one can discover variations as small as 1-200 of a degree. The fluid to be examined is placed in a central test tube in which is placed the thermometer and a small stirring rod. This test tube is held in a large test tube by a rubber ring. Twenty c. c. of urine or blood is required. The smaller test tube is submerged in salt and ice until the mercury of the thermometer quits the reservoir at the top. Then this test tube is placed within the larger one, which from the very beginning has been snugly packed in ice. The mercury falls below the freezing point; it then rises to the freezing point and remains there for some while, after which it again falls. [The Editor has frozen very satisfactorily, and much more quickly by placing a nest of test tubes within a wooden jacket (a mailing case in which a hole has been made on the side). Through this hole a stream of liquid CO_2 was delivered against the outer test tube.] Tinker finds the freezing point of the blood to be —0.56°C, with a physiologic variation of .02°C. The freezing point of the urine varies physiologically between —0.9°C and —2.°C. Above the former point danger lurked. His observations extended to 25 cases. Three fair illustrations of gross pathologic conditions gave; blood —0.71°C—0.69°C and —0.63°C. The urine in the same cases gave —0.53°C.—0.47°C, and —0.87°C. Tinker thinks the test reasonably easy, and, in case of blood, of remarkable exactness and value.

Kümmel[1] has reported 265 tests on 170 patients, and he is emphatic in his endorsement.

Dresser found the normal freezing point of urine to be —1.2°C, to — 2.3°C. The variations noted by him in pathologic states ranged from —0.16° to —4.72°C.

In case we wish to estimate the freezing point of urine

(1) Centralblatt f. Chir., 1902, XXIX.

or blood *without its sodium chlorid,* the method is as follows: take the freezing point of the urine or blood: estimate the percentage of sodium chlorid with a standard silver solution. Multiply the numeral representing the percentage of sodium chlorid by .613, subtract the result from the freezing point estimated above. The result will be the freezing point of the chlorid free fluid. For example let us say the freezing point of a urine containing 1 per cent sodium chlorid is —2.5°C., 1×.613=.613. 2.5—.613= 1.887° C. The freezing point of the chlorid free urine would be —1.887° C. Koranyi insists that repeated examination of the 24 hour specimen must be made and that the body weight and diet must also enter into the calculation. Thus environed Koranyi thinks the test a valuable index.

Claude and Balthazar take the freezing point, multiply it by the number of c. c. of urine passed in 24 hours: the product is multiplied by 100 and divided by the weight of the patient. McCaskey says that the urine must be fresh. He found that a urine which froze at —1.5° C. when fresh, froze at —3.5°C when full of bacteria. He says that one c. c. of formalin in 1 liter of urine raises the freezing point .05° C. D. S. Gwin thinks that the cryoscopic examination of urine is of great value when checked by (1) repeated examinations, (2) cryoscopic blood examination, (3) methylen blue and phloridzin tests.

Phloridzin Test for Renal Sufficiency. Watson and Bailey[1] report the results of experiments with this test on 70 patients. The test depends upon the production of glycosuria by subcutaneous injections of phloridzin. As glucose is separated from the blood by the epithelial structures of the glomeruli and tubules, and in no other part of the body, it can be used to show the relative activity of these structures. It seems clearly established that the phloridzin test is harmless and that in health it shows that the two kidneys have an equal functional activity.

The technic of the phloridzin test is exceedingly simple. It consists of the administration of a subcutaneous in-

(1) *Boston* Medical and Surgical Journal, Dec. 4, 1902.

jection of a sterilized preparation of the drug to which an equal quantity of $Na_2 CO_3$ has been added, the latter being used to hold the phloridzin in solution. The dose of phloridzin is five milligrams for persons of moderate size and weight, and twice this quantity for those of large size and unusual weight. The bladder should be emptied just before the injection is given. Half an hour after the administration of the drug, sugar should appear in the urine if there is normal sufficiency of the renal function. Serious disease of the kidneys is indicated if no sugar is present. And if its appearance is delayed, or only a small percentage is found, it is taken as an indication of renal insufficiency. The quantity of sugar in these tests that shall be taken for the standard by which to determine renal sufficiency or the reverse is a somewhat arbitrary one. The authors took for the point of division between the normal and abnormal performance of the renal function, the sugar percentage which stood midway between the average percentage of the cases in which the kidneys were believed to be normal, and the same of those in which renal disease was known to be present, which was found to be .30 of one per cent, all percentages of sugar falling below this number being taken as an indication of renal insufficiency in proportion to the degree in which they were less.

After giving the details of the results obtained in their seventy cases the authors state the following conclusions:

1. That the average quantity of sugar eliminated in the first half hour after the administration of the drug subcutaneously in the doses stated, and when the kidneys are normal, is about .45 per cent; and that the first half hour's elimination is greater than the second half-hour's by about .06 per cent.

2. When renal disease exists, the first half hour's quantity of sugar eliminated is, for a series of cases, about one-half as much as that when the kidneys are normal, and there is but little more in the first half hour than in the second.

3. The effect of ether anesthesia is to stimulate the kidneys to greater functional activity, but the renal function is not, if judged by the phloridzin test, in any way impaired by the anesthesia. This is true of the kidneys when normal only; when they are diseased, ether fails to stimulate their function, and there is, moreover, relatively much less sugar eliminated in the first than in the second half hour after phloridzin has been given than is the case when the kidneys are sound.

If judged by the average results of a series of cases taken collectively, the phloridzin test gives accurate indications of the condition of the renal function and of the existence of renal disease. In a certain number of individual cases, it even appears to be more delicate and discriminating than the ordinary urinary tests, but the relatively large number of instances in this series of observations, in which the reverse of this is true, shows it to be too variable to be trustworthy, or to incline one to urge its adoption in preference to the former methods of urinary analysis for estimating the functional capacity of the kidneys.

Casper[1] reiterates his faith in the *phloridzin test of kidney capacity.* He has operated on kidneys 72 times without a casualty, depending for his decision upon data furnished by this test. In 88 cases analyzed by him the test was found to be reliable. In 9 cases death occurred. In 7 of these autopsy was allowed and they showed that the test was accurate. In 3 cases the test indicated that the other kidney was involved. The autopsies so showed. He is somewhat skeptical of cryscopy. In the first place the blood must also be examined; secondly, account must be taken of the total quantity of solvent.

Conductivity of Urine. Long[2] has come to the conclusion that the electrical conductivity of urine is of no clinical importance at the present time. He quotes Steyrer[3] as thinking it of value as a check on cryoscopic and chemic

(1) Deutsch. med. Woch. XXIX., No. 25.
(2) Trans. Chicago Pathological Society, Feb. 9, 1903.
(3) Beitrage zur. chem. Phys. u. Path., 1902.

examination. [It is difficult to accept this, as electrical conductivity is dependent wholly upon the inorganic salts, and especially the sodium chlorid, whilst it is not affected to any material degree by urea, uric acid, the xanthin bodies, or any of the toxic organic products of tissue waste.]

GENERAL PATHOLOGY.

Yellow Fever. Parker, Beyer and Pothier[1] constituted a commission sent by the Public Health and Marine Hospital Service to study the problems of yellow fever. They report the following conclusions:

1. That the bacteriologic examination of the blood of cases of yellow fever during life, and the blood and organs at autopsy performed immediately after death in uncomplicated cases, is negative.

2. That Stegomyia fasciata, when contaminated by feeding on a case of yellow fever forty-one and a half hours after the onset of the disease and subsequently fed on sugar and water for twenty-two days one and a half hours, can, when permitted to feed on a non-immune individual, produce a severe attack of the disease.

3. That Stegomyia fasciata, contaminated by feeding on a case of yellow fever and after varying periods killed, sectioned, and appropriately stained, presents with regularity a protozoan parasite, the Myxococcidium stegomyiæ, that can be traced through a cycle of development from the gamete to the sporozoite.

4. That Stegomyia fasciata fed on blood from a case of malarial fever, on normal blood, or artificially fed, does not harbor the parasite indicated in conclusion 3.

Vera Cruz was selected as the base of study, for several reasons. In the first place, owing to the practical recognition of the mosquito agency in transmitting yellow fever, this disease has ceased to exist in the possessions of this

(1) Yellow Fever Institute Bulletin, No. 13, Public Health and Marine Hospital Service.

country. In the second place, Vera Cruz is infected with yellow fever at all seasons of the year, and by reason of the high interior, yellow-fever-free country is not remote from the city.

They found Stegomyia fasciata abundantly present in

Fig. 3. Stegomyia fasciata Fabr. Female.

Vera Cruz, and they learned that it was there at all seasons of the year. This mosquito is a day flyer, seldom biting after the oncoming of the evening. The noonday nap of the Mexican gives stegomyia more opportunity for

feeding than it would otherwise have. It breeds in quiet waters, and especially in the water tanks and cisterns that

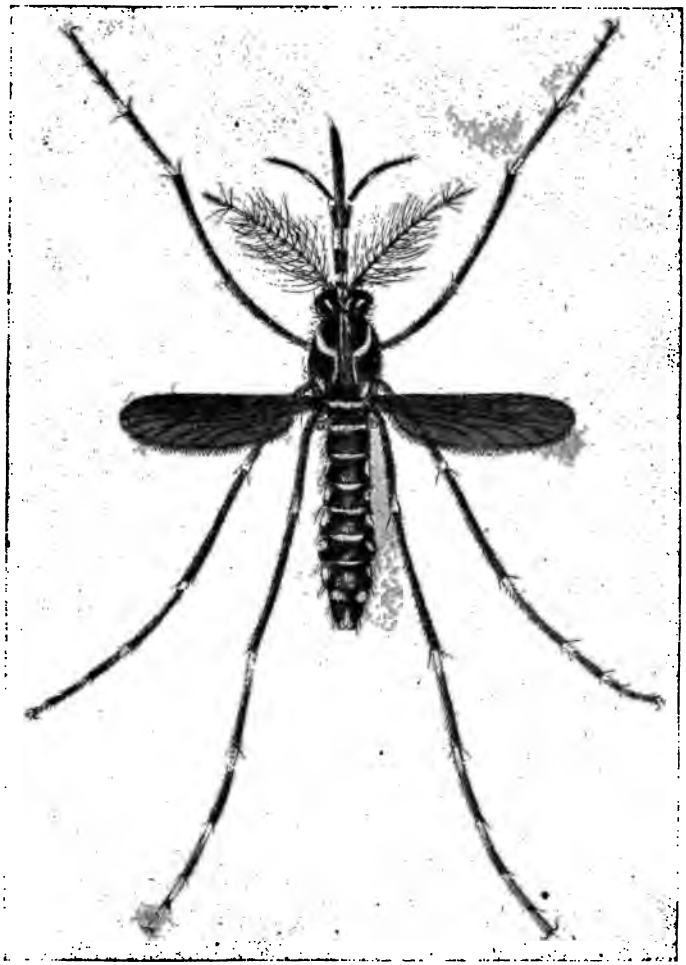


Fig. 4. Stegomyia fasciata Fabr. Male.

are so frequent in semi-tropical cities. They believe that this mosquito is carried from town to town in ships. On

account of the prevailing conditions, wooden ships are very much more liable to transfer it, than are iron ships. They found this mosquito on one of the ships—the Channon—in which it had been discovered in an American port by Grubbs.

Ordinarily stegomyia larvæ can be told from culex by the shorter respiratory syphon. This larva is much lighter in color than is culex, being nearly milk-white when matured. When 8 to 10 days old, the larvæ change to dark brown pupæ. The adult mosquito escapes from the pupa case in from one to two days. This means that all developmental stages have passed in from eight to ten days. The adult is a very persistent biter—that is, it is driven off with difficulty. After feeding, the female becomes quiet, deposits her eggs in a few days and then dies.

Parker, Beyer and Pothier repeated some portion of the work of Reed and his colleagues; they produced yellow fever in a man not otherwise exposed by allowing an infected mosquito to bite him. Having failed to find in the blood, or in any of the organs any bacillus or any other organism which they believed could be held responsible for yellow fever, they turned to a study of the histology of infected stegomyiæ.

They found a protozoan parasite in infected mosquitoes, and they were able to work out the life cycle of this parasite. For the first two or three days after the mosquito had fed, her stomach was found filled with disintegrating blood. On the third day a small fusiform-shaped protozoon was seen, and on a few occasions they noted terminal conjugation. The parasite then wanders through the stomach wall and into the esophageal dilatation (suction stomach), where it passes through an oöcyst and sporoblast state. The oöcysts rupture and the sporoblasts penetrate the wall of the esophogeal dilatation and enter the salivary glands. They are to be found within the epithelial cells of these glands. Soon after the sporoblasts come to rest, they form large numbers of minute bodies—sporozoites. Parker,

Beyer and Pothier propose the name "Myxococcidium stegomyiæ." (Parker, Beyer and Pothier, 1903.)

This is the sexual cycle of the parasite. They found it in the mosquito that had been fed on yellow fever patients, in those that had transmitted yellow fever and in those which had fed on patients suffering from the experimentally produced fever. Under no other circumstances

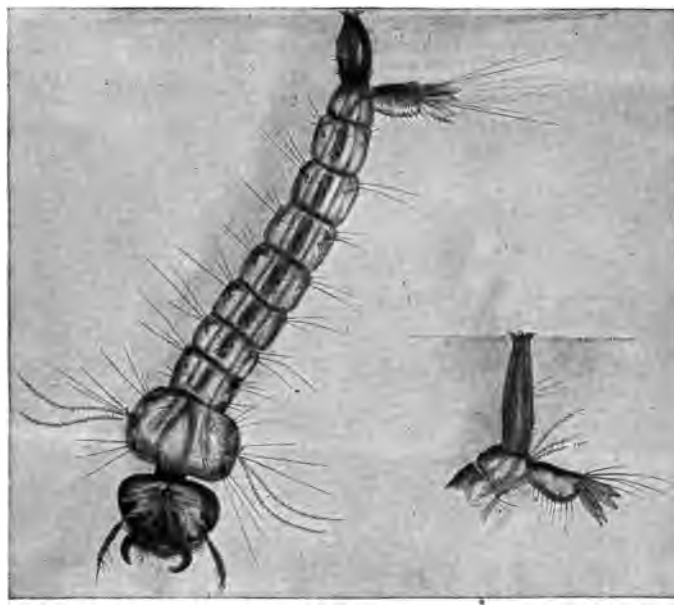


Fig. 5. Mature Larva of Stegomyia fasciata. Respiratory siphon of Culex to the right.

was it found. It is their opinion that somewhere in the human body, the asexual cycle of this protozoon is passed, and they hint at the possibility that this may be in the third blood corpuscles—blood platelets.

This parasite is quite like that of malaria, differing only in the point that the oöcyst forms sporoblasts and then sporozoites, whereas, in malaria, the oöcyst forms

sporozoites direct; the former is the usual plan with the protozoa. It also differs a little from the mosquito existence of malaria, in that it stops awhile in the esophogeal dilatation whilst traveling from the stomach to the salivary gland; the malarial protozoon goes direct.

The authors furnish valuable light on several minor questions connected with the etiology of yellow fever. As we have already said, they were not able to find any bacterium in the blood or in the organs of people dying with yellow fever. They tested the agglutinating capacity of blood serum from yellow fever patients with the Sanarelli bacillus, Bacillus typhosus, Shiga bacillus and the Bacillus coli communis; they found that none of these were agglutinated. Another interesting point noted by them was the gradual spread of yellow fever from Vera Cruz to the interior, and the relation of railway trains to this spread. In years past there was no yellow fever in Cordoba or in Carasal. It occasionally happened that a patient with yellow fever was carried to one or the other of these towns, but the disease never spread. At that time there were no stegomyiæ in these places. Railroads have been built now, and probably as the result of transportation in cars, the stegomyiæ have been spread until they are found abundantly as high as an elevation of 4,200 feet. The consequence is that yellow fever now prevails in these cities. Jalapa and Palmar, stations a few miles farther inland and a few hundred feet higher, are not infested with stegomyiæ, and in consequence yellow fever does not spread, though cases go to these villages every year. Another point of considerable practical importance is that whereas the female Stegomyia fasciata, which has fed on blood, is expected to deposit her eggs and then die within ten days, those that have fed on yellow fever blood and have become infected thereby, live a very much longer time. Parker, Beyer and Pothier noticed that one effect of this infection was that the eggs atrophied and disappeared. Were it not for this fact, yellow fever would be more easily combated than it is at the present time.

[It is to be hoped that before the termination of another season some one will find this protozoon inside the human body and will work out its asexual or sporulating life cycle there. However, we are to remember that from the time that Laveran discovered the plasmodium of malaria in 1880, until the discovery of the sexual cycle of that organism by Ross in 1899, the etiologic relation of the plasmodium to malaria was accepted, although we only knew of its asexual cycle. It is true that the asexual cycle is passed in the patient, and therefore, its relation to the disease is more patent, but that relation was no nearer the accepted views in parasitology in 1880 than is this in 1903. If we are to accept the bodies seen by Councilman, Magrath and Brinckerhoff in variola as protozoa, we are struck with the possibility that protozoa may lie around in tissue sections, missed of observation by reason of close resemblance to host tissue. On reading a work on protozoa and disease—for instance, Clarke—one is constantly struck with the difficulty of distinguishing protozoa from host cells. In this connection, we call to mind the controversy over Plimmer's bodies and Gaylord's protozoon for carcinoma.—ED.]

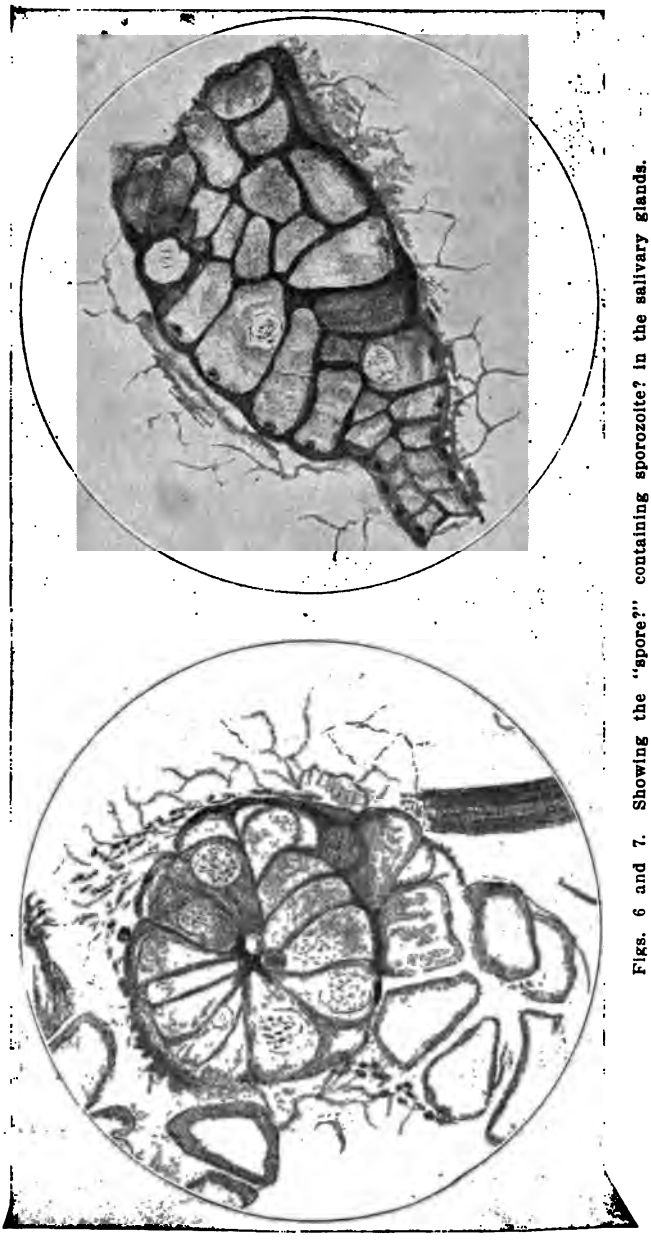
Malaria. Very little that is new has been recorded concerning malaria during the past year. The relation between the mosquitoes of the Anopheles family and this disease has found considerable substantiation. There still remains considerable controversy as to whether mosquitoes constitute the sole means of transmission of the disease. That there is a fair degree of biologic independence of the two stages of the existence of the parasite, namely its asexual and sexual stages, is recognized; but that the asexual parasite can be passed from a sick man to a well man, without the intermediary of the mosquito has not been proven. I know of no material facts relative to the asexual cycle that have been developed during the past year. The sexual cycle of the protozoon is so well known at the present time that it requires but little consideration.

We quote a few points from Clarke[1] placing some emphasis upon certain points of difference between this parasite and that claimed by Parker, Beyer and Pothier, to be the cause of yellow fever: The macrogamete having been fertilized in the body of anopheles changes to a mobile spindle-shaped oökinet. These go into the epithelial wall of the true stomach as contrasted with the esophageal dilatation or suction stomach, the developing place of the yellow fever parasite. Deep in the wall the oökinet loses its mobility and becomes an oöcyst. As the oöcyst grows in size its nuclei divide, and as observed by Ross, sporozoites develop directly without the intermediary sporocyst formation. The oöcyst ruptures in the tissues of anopheles, and the sporozoites move by vermicular motion to all parts of the body, but noticeably to the salivary glands. In yellow fever the oöcyst divides into sporoblasts, each with a nucleus, and then the oöcyst ruptures into the tissue liberating its sporoblasts. These wander into the tissue, noticeably the salivary glands, where they enter the epithelial cells, come to rest and form sporozoites.

Moore[2] watching aestivo autumnal parasites was able to verify the statement of MacCallum, relative to the sexual function of the flagella. He saw a flagellum break away from a hyaline body and travel to a pigmented body. The pigment granules had previously become wildly active. The flagellum entered the pigmented body and was lost to view. The pigment became quiet.

Craig[3] described the microscopic lesions in latent malaria infections, based on seven autopsies. The seven cases were selected because in none of them had any one found malarial organisms in the peripheral vessels during life. The findings were practically the same in tertian, and in aestivo-autumnal malaria. The spleen was darker and larger than normal; otherwise to the unaided eye no pathology was demonstrable. Under the microscope plasmodia were found in fair abundance. Craig concludes that all

(1) Protozoa and Disease, Wm. Wood & Co., 1903.
(2) Johns Hopkins Hospital Bulletin, October, 1902.
(3) American Medicine, July 25, 1903.



Figs. 6 and 7. Showing the "spore?" containing sporozoite? in the salivary glands.

stages of the asexual life of the plasmodium can be passed in the spleen. This is equally true of each variety of malaria. The liver spaces contained a few degenerated plasmodia and pigmented leucocytes. The blood cells contained no parasites, neither did the liver cells. To the unaided eye the liver was negative. Bastionelli and Bignami have reported somewhat similar observations.

Mosquitoes. The interest in mosquitoes increases each year. In Vol. IX., Practical Medicine Series for 1902, we quoted Pothier and Beyer at considerable length. Two of the writers of that article have participated in the discovery of a protozoon in Stegomyiæ fasciata that have fed on yellow fever bloods.

Parker, Beyer and Pothier tell us that Stegomyia fasciata is to be found in all cities and towns south of Maryland, though it is possibly still more widely spread. More important than the present location of stegomyia, is—what towns and cities store their water supply in small above-ground cisterns? The following description of stegomyia is taken by the authors from Theobald:[1]

The eggs of Stegomyia fasciata are laid in any uncovered collection of water. They are usually to be found near dwelling places in cisterns and barrels of water, grindstone water, etc. They are seldom found in underground cisterns or other rather dark places. About 150 boat-shaped eggs are laid; they are dark brown in color and flattened on one side, 0.04x0.75 mm. The larvæ hatch in 10 to 24 hours, growing to pupæ in 8 to 10 days. They have a short, barrel-shaped, black siphon; they are whitish-gray in color, being much lighter in color than Culex. If the water vessel is jarred, the larvæ go right to the bottom; they cannot be poured out except with the last of the water contained in the vessel. Handling or drying is about as fatal to them as is kerosene.

Adult Female. Head densely clothed with broad, flat scales, black and gray on each side, a white patch in the

(1) Theobald's description, quoted on p. 21, Yellow Fever Institute Bulletin No. 13, Public Health and Marine Hospital Service.

middle, in front, extending back to the neck, a white patch on each side, a thin white border to the eyes; the scales at the back of the crown with an ochraceous tinge in some lights, long black bristles projecting forward;

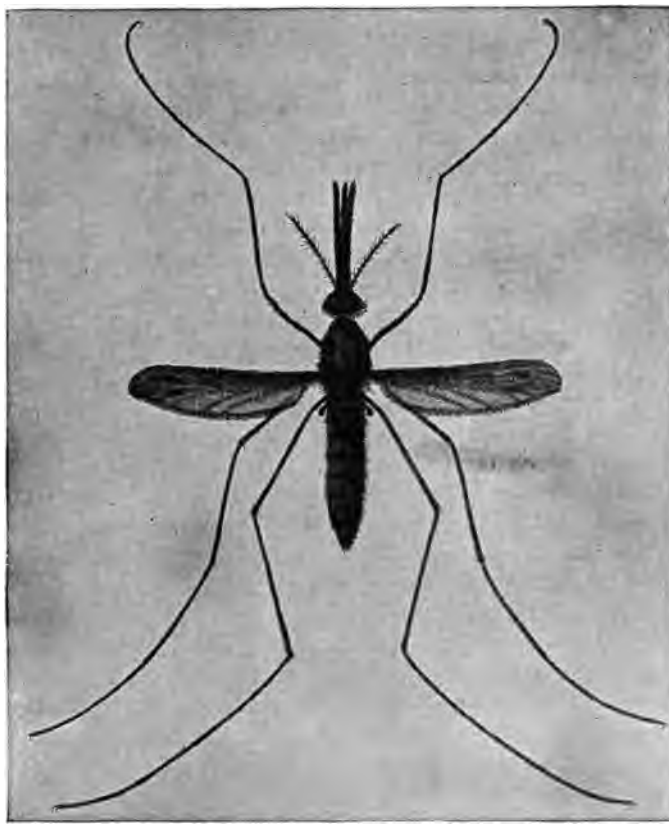


Fig. 8. Anopheles maculipennis Meigen.

eyes black with silvery patches in some specimens; antennæ blackish, with narrow pale bands; basal joint black, with a patch of white scales on the inside (appearing as

two small white spots with a lens); second joint sometimes pale testaceous at the base, pubescence and verticils dark brown; palpi black scaled, the last joint with pure silvery white scales inside and on the tip, sometimes entirely white.

Thorax dark brown, covered with reddish-brown, pale golden and creamy curved scales ornamented as follows: A pure white, broad curved band on each side, curved inward about the middle of the mesonotum and continued back as a thinner pale line to the scutellum, two thin parallel pale scaled lines between, extending about halfway across the mesonotum and more or less onto the scutellum, a short white line in front between these two, a white spot on each side of the thorax in front near the neck; scutellum with a thick row of white scales and with three tufts of bristles; metanotum brown; pluræ dark brown with several patches of silvery scales.

Abdomen dark brownish black, with basal bands of white scales; first segment densely clothed with creamy scales and edged with pale hairs; sides with patches of white scales forming more or less flat triangular patches.

Legs with the femora with the bases yellowish, dark scaled toward the apex, extreme tip pure white, ventral surface partly covered with white scales; tibiæ black; metatarsi with basal white bands; fore tarsi with the first joint basally white, rest black; mid tarsi the same; hind tarsi all basally white, except the last joint, which is pure white, penultimate joint mostly white with black apex; fore and mid ungues both toothed, and hind without teeth.

Wings with the veins clothed with very long, narrow, brown scales and short median, broad dark-brown ones; first submarginal cell longer and but slightly narrower than the second posterior cell, base of the former a little nearer the base of the wing than the latter; posterior cross vein about one and a half times to twice its length distant from the mid cross vein.

Halteres ochraceous, sometimes the knob is slightly fuscous.

Length: 3.5 to 5 mm.

Male. Darker than the female.

Head black with white scales in front and in the middle; antennæ brown with paler brown bands, sometimes almost white, basal joint jet black with a large tuft of pure white scales, plumes brown; palpi black with four white basal bands; proboscis black; thorax marked as in the female, but much darker, and the white scales clearer and more silvery.

Abdomen with the first segment with creamy scales, bases of the second to the fifth segments white, fifth to eighth with white lateral spots; these spots also occur on the front segments, but quite at the sides.

Legs as in the female. Fore claws unequal, the larger one with a short blunt tooth, smaller one untoothed; mid ungues unequal, untoothed; hind equal, untoothed.

Length: 3 to 4.5 mm.—(Theobald.)

In reporting recent progress in our knowledge of malaria, Wright[1] gives the following tabulated comparison of anopheles with culex:

ANOPHELES.	CULEX.
Selection of water on which to lay eggs: Clear, quiet, slow running streams.	Selection of water on which to lay eggs: Artificial pools, receptacles in which water is left standing, grassy streams.
Larvæ: Head small and breathing tube short.	Larvæ: Head large and breathing tube long.
Do not descend to bottom until late in larval stage.	Feed on the bottom, ascending to surface to breathe.
Larvæ lie just below and parallel to the surface, with breathing-tube and portion of head protruding above it.	Larvæ while breathing assume a position of 45° to the surface, breathing-tube protruding.

(1) American Journal Medical Sciences, October, 1902.

ADULT MOSQUITO.	ADULT MOSQUITO.
Palpi of both male and female the same length as the proboscis.	Palpi of female much shorter than the proboscis.
Certain arrangements of spots upon the wings.	No spots upon the wings.
When clinging to horizontal surfaces, the body is almost at right angles to the surface.	When clinging to horizontal surfaces, the body is parallel to the surface.
Vertical surfaces; body at angle of about 35°.	Vertical surfaces; body parallel.

Ship-borne Mosquitoes. Grubbs[1] examined 82 vessels with a view to determining the presence of mosquitoes, and when present, the circumstances of their arrival and their propagation. In 65 he found no mosquitoes; in 14, culex was found, and 9 of the 14 had become infected while off shore, possibly as much as twenty miles; in 3, he found stegomyia. This mosquito had come on board while the vessels were one-half mile off shore. One of these vessels was found infested by Grubbs at Ship Island, Hodgson at Vera Cruz and Parker, Beyer and Pothier at Vera Cruz all working independently. He thinks that mosquitoes can live on board ship as much as 17 days, even when they cannot propagate. They breed in any bit of still water. During rough weather at sea, larvæ do not fare well. Giles and Parker have recorded transportation of mosquitoes by ships. Parker, Beyer and Pothier describe the transference of these mosquitoes by railroad trains.

Carter[2] proves that yellow fever can infect the crew of a ship through stegomyia on board the vessel.

A parasite round worm in American mosquitoes. Stiles[3] has collected the following facts relative to the parasites of mosquitoes:

(1) Bulletin No. 11, Public Health and Marine Hospital Service.
(2) Bulletin No. 9, Yellow Fever Institute, Public Health and Marine Hospital Service.
(3) Public Health and Marine Hospital Service Bulletin, No. 13.

Ross (1895) found gregarines in the intestines of mosquito larvæ. Perroncito (1899) found a filamentous phyto-parasite in anopheles, near Turin. Lavaran (1902) described a pathogenic yeast in the abdominal cavity in Anopheles maculipennis in Spain and he found several acarines on the bodies of culicidæ. Leger (1902) found a parasitic flagellate in the intestine of Anopheles maculipennis. Marterano[1] has described a trematode in the same mosquito. Stiles adds a nematode Agamomermis culicis found in the abdominal cavity of Culex sollicitans. He found a similar worm in Saxony and the mosquitoes infected were definitely sick. [The greatest importance of these discoveries attaches to the possibility of solving the mosquito question by infecting them with parasites. The malaria mosquitoes can never be adequately destroyed in any other way. The yellow fever mosquito is more easily made way with.—ED.]

A protozoan parasite of mosquitoes. Johnson[2] has made a study of the mosquitoes near Roxbury, Mass. Three hundred and fifty-eight were proportioned as follows: Anopheles maculipennis, 248; Anopheles punctipennis, 25; Culex pungens pipiens and Conso brunis, 85. The principal object of his study was to determine the percentage of these that were infected with malaria. Not one was so found. He found 20, or 5.6 per cent, contained a protozoan parasite very similar to malaria. This was found as an oöcyst on the stomach wall. Once he found one in the salivary gland. This oöcyst had but a single nucleus. It contained bodies that might be sporoblasts, but only the oöcyst stage was found. Johnson thought that it was gregarine and he thought it belonged to the acephalina. The principal point of contrast with malaria was the single nucleus, but this point was not demonstrable unless the parasite had been fixed in sublimate and then stained. Johnson thinks that there is great probability that these parasites will be confounded with the

(1) Centralb. f. Bakt., Vol. 30, Dec., 1901.
(2) Jour. Med. Research, March, 1903.

oöcysts of malaria. The illustration indicates more than a probability that this mistake will be made.

Mosquitoes as hosts. Mosquito hosts have been claimed with some show of reason for the following parasites:

Anopheles for malaria.—Ross and Manson.

Stegomyia fasciata for yellow fever.—Reed, Carroll, Lazear, Agramonte, Finlay, etc.

Culex ciliaris or Culex pipiens for Filaria Bancroftii.

Culicidæ of different varieties for dengue.—Graham.

Stegomyia fasciata for Filaria Demarquai.—Low and Vincent.

Culex Fatigans for Filaria Nocturna.—Quoted by Low.

Trypanosomes. Incited by the great activity of those working in diseases due to animal parasites, much attention has been directed during the past year to a division of the protozoa, known as the flagellata. A sub-class of flagellata, namely, the trypanosoma, has been the subject of very fruitful study during recent years. The better known of these pathologic varieties are:

Trypanosome brucii, which is responsible for nagana.
" evansi, responsible for surra.
" equiperdum, which is responsible for douraine.
" lewisi, which is the trypanosoma affecting the common brown rat.
" equinum, which is responsible for mal de caderas.

During the past year Dutton, Manson, Castellani and others have demonstrated a trypanosome in the human subject; in fact, it seems certain that Kruse of Bonn is right, in his statement that the human subject can be infected with more than one trypanosome, as the parasite described by Dutton, Manson and others, presently to be referred to as the cause of an intermittent fever, does not seem identical with that described by Castellani as the cause of sleeping sickness.

By reason of the large death rate among horses in the Philippines, Salmon and Stiles[1] prepared an emergency report on surra containing, in addition to the large amount of information on this disease, a bibliography of trypanosome diseases prepared by Hassell. Francis'[2] statements, on Trypanosoma lewisi, give us information that in all probability will be of value to us. Sherwood[3] has reported to the Chicago Medical Society a case of intermittent fever, accompanied by urticaria, and lasting four months, which was identical, clinically, with the cases of fever due to trypanosome infection reported by Dutton, Manson and others. Sherwood's case was due to the bite of an ordinary wild rat. Evans[4] refers to the case seen by Sherwood, and two others seen by him in the far South, each of which had been bitten by a rat. All three of these cases were clinically of the same type as that in which trypanosome was found in the blood, by English and African workers. He has collected about 25 similar cases in the American literature, 2 in the European literature, and has made use of the 45 cases collected from the Japanese literature by Myaki.[5]

The following description of the parasite is given by Francis: The parasite is always extra-cellular. It swims with its flagellum end forward, its blunt end backwards. Its motion is wavy and rotary. It pushes its way here and there among the red corpuscles, which however it never enters. It measures from 13 to 25 mikrons in length, with a flagellum from 7 to 15 mikrons in length. Along one side is an undulating membrane. The parasite can divide by: (1) longitudinal division, (2) transverse division, and (3) segmentation; and all three kinds of division have been found in the same specimen. In addition, when the nutritional conditions become bad, for any reason, the parasite agglutinates in rosettes, which

(1) Bulletin No. 42, Bureau of Animal Industry, 1902.
(2) Hygienic Lab. Pub. Health and Marine Hospital Service Bulletin, No. 2.
(3) Chicago Medical Recorder, Vol. XXV.
(4) Transaction of the Chicago Pathological Society, Vol. V.
(5) Mittheil. aus den Grenzegebiett der Medicin und Chir., 1898.

PLATE 9. Magnified 1000 times.

Figs. 1 and 2. Adult trypanosomes.

Fig. 3. Parasite undergoing longitudinal division.

Fig. 4. Transverse division.

Fig. 5. Beginning multiplication.

Fig. 6. Same, later stage. The mother parasite is still visible.

Fig. 7. A. multiplication rosette.

Fig. 8. Auto-agglutination.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

PLATE 9.

bear some rough resemblance to the segmenting stages of malaria. Agglutination can be brought about by treating the blood with a normal serum from some non-susceptible animal, or by placing it awhile in an ice-chest. The agglutinated parasites do not lose their mobility. As the animal becomes immune the parasites begin agglutinating, giving the condition which Francis terms, perhaps incorrectly, "auto-agglutination."

The duration of the infection in the rat does not seem to be long, and after one infection the animal seems to be immune. In this opinion Francis is sustained by Rabinowitsch and Kempner[1] and Laveran and Mesnil.[2]

In Francis' rats the duration of the infection ranged from seven to fourteen days. His, however, was artificial infection. Jurgins[3] found that his animals remained infected for from one to two months.

The parasite by reason of its large size, and its rapid movement, pushing the blood cells to either side, is easily found. It is not easily confused with any ordinary condition. In dried specimens it stains very nicely with Romanowski, Jenner, Goldhorn, Wright and Leishman. It has not been determined that the parasite has any other cycle save that in the blood, and we do not know how it is transferred under ordinary circumstances. Experimentally it has been transferred by intraperitoneal inoculation, by subcutaneous inoculation, by intrastomachal injection and by feeding. Rabinowitsch and Kempner[4] were able to transfer infection by fleas, and Laveran and Mesnil[5] found the parasite on the bodies of lice which were caught on infected rats. Durham has found trypanosome in Stegomyia fasciata. Flagellata, as a general proposition, do not have a double life cycle.

Novy and McNeal[6] at the 1903 meeting of the Ameri-

(1) Zeitschrift f. Hygiene, Vol. 30.
(2) Ann. del 'Inst. Pasteur, Vol. 15.
(3) Archiv fur Hygien, Band. 42, heft 3, 1902.
(4) Loc cit.
(5) Loc cit.
(6) Jour. [illegible]; also contribut[illegible]

can Medical Association reported the growth of a pure culture of the common rat trypanosome, Trypanosoma lewisi, in the water of condensation of a mixture of rabbit's blood, serum and agar.

Trypanosome Infection in the Human Subject. In December, 1901, Dutton[1] recognized a trypanosome in the blood of a patient who came from the Gambia River in West Africa. The patient had a mild intermittent fever which lasted about two years, producing death in January, 1903. The fever was accompanied by erythema multiforme and a more or less general edema. Dutton suggested the name Trypanosoma Gambii by reason of the geographic location of the infection. The same case was seen by Forde.[2]

Adams[3] gives some further notes and speculations on the Dutton case. The patient had an atypical pneumonia on shipboard which Adams thinks was probably due to trypanosome. He thinks that the infection is transmitted by rat bite. In the Dutton case the man had been so bitten. Baker[4] reports three additional cases. In none was there a history of a bite.

Manson[5] saw a woman with a peculiar intermittent fever coming every ten days, and lasting for two or three days. It was accompanied by an erythematous rash. There were many rheumatic pains. The fever and erythema persisted for over a year. Eventually after 18 months Broeden of the Congo found trypanosoma in the blood. The patient was very certain in this instance that the infection was from a tick bite; Manson thought that it was due to the bite of a tick, Argus moubata. He quoted Dawson and Daniels as having seen a similar fever due to the bite of a tick. He quotes Broeden as having seen two other cases of trypanosomiasis in the Congo.

Manson and Daniels[6] report another case of trypano-

(1) Thompson-Yates Laboratory Reports, Vol. 4, Part 2.
(2) Journal of Tropical Med. Vol. 5, September, 1902.
(3) British Medical Journal, March 28, 1903.
(4) British Medical Journal, May 30, 1903.
(5) British Medical Journal, March 28, 1903.
(6) British Medical Journal, May 30, 1903.

some infection. It was that of a lady who became sick with an intermittent fever, a rash and an edema about two weeks subsequent to a bite. These symptoms had continued, though in a mild form, until May, 1903. In October, 1902, the blood was examined for trypanosome with positive results. The parasites continued in the blood up to the date of the article. The average number of parasites found in counting 500 leucocytes was 20. Hewlitt injected this blood diluted with a solution of sodium citrate into rats, mice, guinea-pigs, rabbits, dogs, monkeys, horses, and pigs. The results were negative. The conclusion of Manson and Daniels is that the human trypanosome is not that of rats, of surra, of nagano, mal de caderas, nor of douraine. Working on the idea that the horse was immune to trypanosome they injected their patient with horse serum, hoping to kill the parasite. It was without beneficial result.

Leishman[1] says that he believes that the disease is very common in India. In the *British Med. Jour.* for March 28, 1903, there is a short note to the effect that the Liverpool School expedition sent to the British Colony reports that in examining 1,000 natives they had found the disease very prevalent. They also find it very common in horses. In a second note[2] they record that they have succeeded in inoculating a horse with human trypanosome. They also found some apparent conjugating forms in the stomach of a fly which had fed on a horse.

[The Editor feels very certain that the fever following a rat bite, reported occasionally in this country and more frequently in Japan, and in which bacteriologic blood examinations have been uniformly negative, are due to trypanosome infection.]

Sleeping Sickness. For a long time there has been much speculation as to the nature of sleeping sickness. Castellani[3] reports finding, on November 12, 1902, a trypanosome in the cerebro-spinal fluid in a case of typical

(1) British Med. Journal, April 18, 1903.
(2) British Medical Journal, April 18, 1903.
(3) British Med. Jour., May 23, 1903.

sleeping sickness. As the number of parasites is small it is necessary to draw off considerable fluid, about 15 c.c. This is centrifuged, and the sediment examined. He found the parasite in 20 per cent of 34 cases. In 12 controls he never found it in the cerebro-spinal fluid. Three of these controls were cases of fever described by Manson, Dutton, Forde and others. He says this fever is very common in Uganda. He thinks that his trypanosome may differ from that of Dutton in having the nucleus nearer the extremity, a larger vacuole and perhaps a slower movement. Col. Bruce continuing Castellani's work found trypanosome in the lumbar fluid in each of 38 cases, and in 12 of 13 bloods examined by him.

Castellani[1] was formerly of the opinion that this disease was due to a diplococcus which he found in the cerebro-spinal fluid. Berrencourt, Kopke, Rezende, and Mendes[2] believe that this diplococcus is identical with a diplococcus previously described by them. Their diplococcus they called hypnococcus. They found it usually in the cerebro-spinal fluid, and rarely in the blood.

Filaria Perstans. Low[3] finds this filaria very abundant in the human subject in equatorial countries. Seemingly it produces no ill health. It is assuredly not the cause of sleeping sickness. The adult worm is 34 to 50 mm. in length and is to be found in the mesentery. The larvæ inhabit the blood. He found some evidence that the intermediate host was the mosquito, Tæniorhynchus fuscopennatus (Theobald). Incidentally, Low says that he and Vincent found some evidence that Filaria demarquai could be transmitted by Stegomyia fasciata, also that Filaria nocturna is found in Culex fatigans.

Human and Animal Tuberculosis. Dean and Todd[4] have experimented along the same lines as Koch. They conclude that human tubercle bacilli will cause tuberculosis in pigs and various other animals when injected sub-

(1) London Lancet, May 14, 1903.
(2) London Lancet, May 23, 1903.
(3) British Medical Journal, March 28, 1903.
(4) Journal of Pathology and Bacteriology, May, 1903.

cutaneously or when fed. Furthermore, when animals have been infected with human tubercle bacilli, and the lesions from those animals are used to infect calves, the calves become tubercular. Human tubercle bacilli seem to lose some of their pathogenic power for calves when passed through an intermediary animal. That they are not more positive about this last point, is due to the fact that the rôle of the accidental bacteria in sputum, in determining infection with tubercle bacilli, remains an unsolved problem.

The sources of error in the work of Koch they believe to have been several; they do not think that his work was extensive enough to draw conclusions from. They think that Koch's use of a culture of tubercle bacillus was an error, since the culture may have been attenuated by growth, or it may have been a rather mild bacillus primarily. Furthermore, they do not think that the factor of the other bacteria of sputum can or should be ignored.

Transmission of Hereditary Syphilis. Schalek[1] thinks that the routes by which syphilis may be transferred to the fetus may be any of the following:

1. Father's sperm cells may contain the virus.
2. The maternal generative organs may contain the virus.
3. Placental circulation may allow infection to travel either way.
4. All of these factors may combine.

The probability of transference through the mother is greater than that of the reverse route for reasons that are easily understood. Such greater probability of transference comports well with the clinical history of hereditary syphilis.

Actinomycosis Hominis. Erving[2] reports six cases of actinomycosis hominis. This brings the total number of cases reported in America up to 100. In four of these cases the location was the abdominal region: one was in

(1) Journal American Medical Assoc., May 16, 1903.
(2) Johns Hopkins Hosp. Bull., Nov., 1902.

the thoracic region, and one in the cervicofacial. Erving, reviewing Ruhrah's[1] collection of American cases, and those since added, thinks the probability that a given infection is through the mouth is always great. In 18 cases the infecting route was definitely proven to be a carious tooth. [The Editor in three cases of actinomycosis in park deer has found the cavities in the maxillary bones filled with grain.] Erving says, however, that there is generally a lack of definite history of the route of infection. Illich (quoted by Ruhrah) gives the point of primary involvement in 421 cases as follows: Head and neck, 55 per cent; pulmonary, 15 per cent; abdominal, 20 per cent; cutaneous, 2.5 per cent; unknown, 6 per cent. Erving thinks that the method of spreading is (1) directly through the tissues, and (2) metastasis, along the blood route. In the Ruhrah collection the liver involvement was secondary in five cases to intestinal lesions, and in two cases to lesions in the lungs.[2]

Anvray[3] has collected, from the literature, 31 cases of *actinomycosis of the liver.* In actinomycosis of the liver the route of infection may be either from the intestinal tract or from the lungs directly through the diaphragm. In a few cases that Anvray has collected, infection of the liver seems to be the primary focus.

Change in Carcinoma Cells Due to X-rays. Ellis[4] has studied two superficial squamous-celled carcinomas, one mammary scirrhous carcinoma, and one malignant endothelioma that had been X-rayed. He is of the opinion that the *primary process is a necrosis of the cells.* The cell protoplasm becomes granular, vacuolated and sometimes fatty; occasionally it fuses with neighboring protoplasm. The nucleus undergoes fragmentation. There is increase of elastic tissue and some lymphocytic infiltration. There is some tendency to occlusion of vessels. [This coincides in most points with the observations of the Editor. In

(1) Annals of Surgery, 1899 and 1900.
(2) Fütterer, N. Y. Medical Journal, 1901.
(3) Revue de Chirurgie, July 10, 1903.
(4) American Jour. Med. Sciences, Jan., 1903.

the early cases that he has examined the striking phenomenon has been the cell degeneration of the type described by Ellis. However, in his observation there has been great polymorphonuclear infiltration. A peculiarity occasionally noted has been the presence of islands of deeply staining and well nourished epithelial cells in the midst of degenerated fields. In cases in which X-raying had been long continued there were heavy bands of mature fibrous tissue and obliterating arteritis.] Codman[1] believes that the influence is exerted on the trophic nerves. He found arteritis present.

Pusey[2] thinks the degenerative process primary as does Scholtz.[3] Rinehart[4] endorses this opinion. McCaw[5] and Beck[6] describe a colloid degeneration which Ellis failed to demonstrate in his specimen. Ellis disagrees with Beck in that he does not believe that the arteritis bears an etiologic relation. It is quite understandable that the chronicity of X-ray burns is due to the connective tissue changes in the blood vessel walls around the nerves and elsewhere, but this has but little to do with the explanation of the acuter changes in carcinoma tissue that has been treated with X-ray.

Stewart[7] is of the opinion that the primary and essential changes in carcinoma cells that have been subject to X-ray treatment are *degenerative in character;* that the beneficial effects are due to this destructive action on the carcinoma cells, or whatever may be contained in those cells; and that the connective tissue processes are secondary thereto. In the epithelial cells he found a marked fatty degeneration, karyokinesis disappearing, and chromotolysis marked. The hyaline infiltration which Stewart found in considerable quantity was akin to the hyaline infiltrations that are usual and to be expected in squamous epithelial cells. Later in the process the nuclei lost their

(1) Philadelphia Med. Jour., March 8 and 15, 1903.
(2) Jour. Am. Med. Assoc., Sept. 12, 1902.
(3) Archiv f. Derm. and Syph. Bd. LIX. Hft. 1, 2 and 3.
(4) American Journal Med. Sciences, July, 1902.
(5) New York Medical Journal, Aug. 9, 1902.
(6) New York Medical Journal, May 4, 1902.
(7) Jour. Amer. Med. Assoc., July 18, 1903.

capacity for staining and the cell protoplasm lost its outline,—fusion occurring. Plimmer's bodies were frequently found. There was a new growth of blood vessels, and of elastic tissue. The tissues were infiltrated with leucocytes, generally, to a very considerable degree. In one case plasma cells were found to be infiltrating in very large numbers. Christian[1] in his discussion of the Stewart paper, refers to the carcinoma cells that have been treated with X-rays as "dead cells." Amongst the infiltrating cells he finds giant cells.

Codman[2] does not think that the therapeutic results of X-rays are due to destruction of the malignant tissue. It is rather due to a *stimulation of the nutrition of the parts.* In at least one case of carcinoma no destruction of the cells could be demonstrated. The shrunken cells sometimes seen are the result of the activity of the granulation tissue.

[The Editor is definitely of the opinion that the primary change is a retrogressive process within the epithelial cell.]

Enzymes in Tumors. Buxton[3] has tried to find ferments in malignant tumors. As the amount of material at his disposal was small, he used the agar diffusion method. That is to say, he made mixtures of various substances with agar and planted the glycerin emulsion of the tumor thereon. It is needless to say that he took proper precautions to prevent the growth of bacteria. His results were rather negative. There seemed to be little or no proteolytic ferments. There was more evidence of glycogen and maltase. There was evidence of lipase, or a similar fat-splitting body, and probably also an oxidizing body. All in all, he is not justified in drawing any conclusions and he draws none.

(1) Jour. Am. Med. Assoc., July 18, 1903.
(2) Johns Hopkins Hospital Bulletin, May, 1903.
(3) Journal Medical Research, January, 1903.

DISEASES OF THE BLOOD.

Pernicious Anemia and Hemo-lymph Glands. Warthin[1] in studying 8 cases of pernicious anemia found that the hemolymph glands were always pathologic. In each case they showed dilatation of the blood-sinuses and evidences of increased hemolysis. In some cases the glands were materially increased in size in others they were not. Warthin found no evidence of hemolysis in his eight cases, in the stomach, intestines or liver. The hemosiderin found in the liver and kidney is transported there. He found the spleen, the lymph glands, and bone marrow giving evidences of hemolysis. The pigment found in the hemolytic organs does not give the reaction for iron: it is only after it has become granular that it does so react. He offers the suggestion that the destruction of hemoglobin forms something (possibly histon), and this in turn is hemolytic—a vicious cycle.

Warthin[2] states that the credit for the discovery of the hemolymph glands belongs to Gibbes, who wrote of these glands in 1884, holding that they were *sui generis*. It is the opinion of Warthin that the hemolymph glands can develop from fat, especially from the fat which is to be found in the prevertebral region. When for any reason whatsoever, there is a greater need for hemolymph glands, the blood spaces dilate, the fat absorbs and the lymph gland elements proliferate. The newly dilated and arranged blood channels may fill up with lymphococytes, in which event the hemal gland becomes a lymph gland. He uses the term hemolytic glands and subdivides them into four groups. The first, splenolymph glands; second, hemal glands; third, hemolymphatic glands and fourth, hemolymph glands. Group four, contains both blood and lymph anatomic elements, and the channels belonging to these two systems anastamose. In group three, both

(1) American Journal Medical Sciences, October, 1902.
(2) Trans. Chicago Pathological Society, Nov. 12, 1902.

lymph and blood channels are found, but there is no anastomosis. The functions of these glands are, first, hemolysis; second, the formation of leucocytes, and blood plasma; third, destruction of leucocytes; and fourth, the production of eosinophiles.

The question of the possibility of the *formation of hemolymph glands in areas of fat* by the growth of tissue, from the blood vessel wall, is discussed in a report of two cases of adiposis dolorosa, made by McCarthy.[1]

Tizzoni and Filetti, 1880, noted the new growth of glands in the midst of and from adipose tissue. This was corroborated by Warthin[2], who said that if the blood channels persisted, a hemolymph gland was formed. If the lymphoid tissue overgrew, it formed a lymph gland.

The author reports two cases of adiposis dolorosa. In one, autopsy showed adenocarcinoma of the pituitary body, acute parenchymatous nephritis and interstitial neuritis of the nerve filaments of the subcutaneous fat. In the subcutaneous fat were many orange-sized, red nodules surrounded by a fibrous tissue capsule. In the vicinity of the large bodies were many small pea-sized bodies, lying free in the fat. Microscopically, these were typical hemolymph glands. The same type of changes was found in the large bodies.

The calendar of changes through which fat is changed to a lymph gland is as follows: First, there is a change in the acid values of the fat as determined by Edsall. The absorption of fat is assisted by large round vesicular cells, similar to the compound granule cells of the central nervous system. As absorption proceeds, the capillaries of the capsule and trabeculæ extend into the fat areas. Here and there small foci of lymphocytes appear. Here and there amongst the lymphocytes, plasma cells appear. He found much free blood pigment, though but little of it gave the iron reaction. The second case was also one of adiposis dolorosa. Post mortem, he found a glioma of

(1) Journal Medical Research, May, 1903.
(2) Journal Medical Research, May, 1902.

the brain, involving the pituitary, a dorso-cervical syringomyelia, chronic thyroiditis, with calcification and acute focal thyroiditis, cirrhosis of the ovary, and interstital neuritis, in the adipose areas. The microscopic findings were similar to those of the first case.

Etiology of Pernicious Anemia. Charlton[1] has studied the effects on the blood of injections of colon bacillus into the peritoneal cavity and into the blood current of rabbits. He received his impetus from the work of Adami, Ford and Blachstein. His culture was a sub-variety of bacillus coli communis cultured from the intestinal content of a rabbit. He found that the rabbits which lived some time became profoundly anemic. The type of anemia was that of pernicious anemia, the drop in red cells being fully as great as the drop in hemoglobin. The secondary changes in shape, size and consistency were those of profound anemia. There were no definite tissue changes.

Etiology of Leukemia. Nichols[1] thinks that there is little evidence that any predisposing conditions influence the oncoming of leukemia except possibly trauma, malaria, severe anemias and pregnancy. The cause must be toxemia, infection or cancer-like hyperplastic processes. He believes that toxins as a cause cannot be eliminated in the present state of medical information. He has tabulated all of the cases in literature in which bacteriologic examinations were made. Of these 96 cases, 75 were adequately examined. That is adequate according to the standards which prevail for known bacteria. These were about equally divided between acute and chronic cases. In two-thirds bacteria of some sort were found. He concludes that, in all probability, all of these infections were secondary and most of them were terminal. He does not accept either Löwit's *Hemameba leukemiæ magna* for myelemic leukemia or his *Hemameba leukemiæ intra-nuclearis* as the cause of lymphatic leukemia. He rather believes with Turk that these bodies are artefacts. Ani-

(1) Journal Medical Research, Nov., 1902.
(2) American Medicine, July 18, 1903.

mal inoculations with human leukemia have been unsuccessful, though Moore, Delbet, Löwit and Bonvicini show that a leukemia-like condition can be transmitted. This is done more easily, however, from one lower animal to another than from man to a lower animal. Especially successful was Moore in infectious leukemia of fowls. Nichols does not believe that contagion possibility has been established for either acute or chronic leukemia. As to the cancer-like theory, so long as nothing is known of the etiology such a theory is not possible of demonstration, even were it correct.

Protozoa of Leukemia. Löwit[1] sets forth further observations in support of the pathologic relations of the protozoan bodies described by him. He reports that he has found by post mortem examination hemameba magnum in its spore stages in the blood forming organs in myelemic leukemia. In a rabbit infected with leukemia he found two forms of the parasite; an amebic form (the principal form in man), and a flagellate form (seldom found in man). He thinks that the flagella are for locomotion, for attaching to the leucocyte, and they, in his opinion, also have sexual functions. He thinks that in the human system the ameba proliferation is by sporulation, and that outside the human body proliferation is by sexual reproduction. Incidentally, he claims to be able to inoculate rabbits with the disease.

The next of his series of articles relates to the staining of the ameba. His method is as follows: He uses a strongly alkaline methylene blue solution: The specimen is heated while staining: it is then washed in acid water, and then stained with an eosin solution. In the nuclei of the leucocytes, in cases of lymphemia, he found long, oval or sickle-shaped bodies. He called these *Ameba parva intra-nuclearis*. His method of staining for amebæ in the organs was as follows: The sections were stained in a strongly alkaline blue, in acid water, then in Lugol solu-

(1) Zeitschrift für Heilkund, Bd. 21 Heft 9, und Ziegler, Beitrage zur Path. Anat., Bd. 33, 1903. Heft 1 and 2. Centraiblatt für Pathologie, Bd. XIV, June, 1903.

tion, then in 0.3 per cent solution of salt in alcohol, then In ordinary alcohol. He found nucleoid bodies in the nuclei of the leucocytes, in the spleen, lymph glands, and in the bone marrow. In a case of pseudoleukemia he found these same bodies very abundant in the spleen, and a very few in the lymph glands. There were none in the bone marrow.

Löwit[1] is more convinced than ever that his ameba is the cause of myelemic leukemia. He thinks that it is a hemosporidien, that it is an obligatory leucocytic parasite, that it is usually a sphere, though it may have a ring form. The extra-cellular form—a sickle-shaped body—was not always found. The sickles, he thinks, are probably the sexual form: he found them frequently becoming oval in shape. His parasite usually destroyed the cell.

Löwit has taken cognizance of the objections raised against his discovery and has endeavored to show the difference between his parasite and the mast cells. He suggests two differentiating methods. His first method is to heat the preparation for from one to two hours, to 200° to 230°C. He then stains in a watery solution of thionin. After such a heat nothing is to be seen except the Löwit ameba. The red cells, white and mast cells have lost their capacity to take a stain. His second method depends upon the staining of mast cells and his ameba with different methylene blues and thionin. He has never found the granules of mast cells taking the same shade of blue as does the ameba. The methylene blue that he used is known as "medicinal of the best quality." The thionin is Muhlheim's.

Unusual Case of Leukemia. Simon[2] reports a case of leukemia in which the symptoms and the course of the disease differ very much from the classical clinical pictures. The earlier symptoms were those of a secondary anemia.

The patient was under observation for eighteen months. The number of leucocytes did not rise above 25,000 until

(1) Contralblatt für Pathologie, Bd. XIV., No. 11, June, 1903.
(2) American Journal Med. Sciences. June, 1903.

within two weeks of his death. The usual leucocyte count was 10,000 to 15,000. The spleen was not palpable below the ribs until within six months of his death. A differential leucocyte count was always suggestive. On this symptom the diagnosis of leukemia was finally made. The percentage of neutrophilic myelocytes was 6 at the first examination; at the end of the first year it had risen to 29. During the last six months of life it ranged from 25 to 53. Ordinary eosinophiles were always absent. Mast cells were always low, being usually about 3 per cent. There was a decided increase in large hyaline or non-granular mononuclears, especially towards the end when these rose to 15 per cent of the total leucocytes.

The author devotes a good deal of space to discussion of the large mononuclears. He thinks that they are lymphoid, that they are from the bone marrow, and that they are the antecedents of the myelocytes. He believes in Reed's 6 forms of myelogenic leukemia. The general tendency of the myelogenous form is towards chronicity, of the lymphatic to acuteness. Nothing except the differential count saved this from being regarded as an ordinary secondary anemia during the earlier months of the disease.

Hodgkin's Disease. Simmons[1] has studied histologically and bacteriologically, nine cases of Hodgkin's disease. Naturally he devotes considerable attention to Sternberg's conclusion, namely, that tubercle bacilli are responsible for this disease. He did not find tubercle bacilli in sections of the glands and inoculation tests were negative. He found no histology to suggest tubercle. There was no focal necrosis. He considered Crowder's cases of intestinal tuberculosis in which tuberculosis was demonstrated to be able to produce extensive tissue hyperplasias without focal necrosis. In the glands a few giant cells were present, but they were not of the tubercle variety. In three of his cases tubercle was present in other organs, as proven by autopsy or by plain clinical signs.

(1) Journal Medical Research, June, 1903.

Weighing all of this evidence, Simmons concludes that tubercle does not stand in etiologic relation to Hodgkin's disease. He is of the opinion that Hodgkin's disease has no relation to leukemia. Histologically, he finds proliferation of the endothelial and other cells of the reticulum and eventually the gland masses become a mass of fibrous tissue.

He is also of the opinion that there is no relation to sarcoma.

Eosinophiles in the glands were quite scanty.

THORACIC ORGANS.

Obliteration of the Superior Vena Cava. Osler[1] reports two cases of obliteration of the superior vena cava. In one the obliteration was a conversion of the superior vena cava, innominate veins, and left jugular, into a fibrous cord due apparently to a tubercular caries of the adjacent vertebra and inflammation of the neighboring structures. The necropsy in this case was performed with a view of determining the collateral circulation, and so the blood vessels were properly injected and the dissection made with this point constantly in mind. In this case the collateral circulation was through the internal mammaries and the lateral thoracics into the azygos veins, and, into the ascending cava through the epigastrics. In the second case the descending cava was obliterated by enlarged mediastinal glands in a case of Hodgkin's disease. Hume[2] collected 29 cases as follows: 8 cases due to thrombus from phlebitis, 1 case propagated thrombus, 1 case thrombus due to endophlebitis, 4 cases due to adjacent tuberculosis, 4 cases due to mediastinitis, 3 cases due to mediastinal syphilis, 2 cases due to periaortitis, 1 to carcinoma and 1 to fibroma. It is probable that there are many such cases, poorly studied, scattered through the literature or not reported at all.

(1) Johns Hopkins Bulletin, July, 1903.
(2) Ibid.

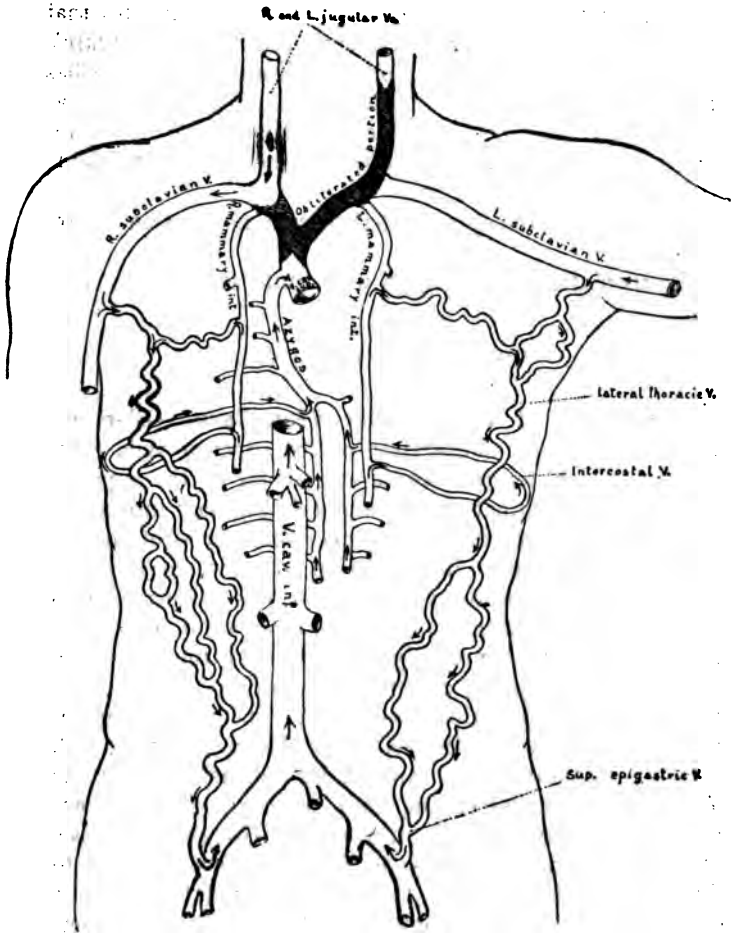


Fig. 10. Osler's case of obliteration of the superior vena cava.

Myer[1] reports a case in which at necropsy Libman found occlusion of the superior cava above the opening of the azygos vein. The occluding area involved the terminal portions of each innominate. The inferior cava was oc-

(1) N. Y. Med. Journal, May, 23, 1903.

cluded at the point of perforation of the diaphragm. There were jaundice, edema of lower limbs, ascites, general adenopathy, marked dilatation of the veins of the trunk, slight dilatation of the veins of head, neck and arms. Adequate demonstration of the collateral circulation was not possible, as the autopsy was made through an abdominal incision. He surmises that collateral circulation is along the channels that were outlined by Thomas.[1] This is not the route of collateral circulation that was demonstrated in Osler's case. Myer is of the opinion that in his case the examination of the ends of the occluded veins indicated no local inflammatory reaction or compression. He cites numerous cases tending to show that gradually acquired obstruction can be of long standing producing a minimum amount of disturbances. Whereas, as is to be expected, a rapidly developing obstruction may produce death in a few hours. The illustration, Fig. 10, shows the obliteration of the vessels and the routes of collateral circulation in Osler's case.

ABDOMINAL ORGANS.

Epidemic Dysentery. Vaillard and Dopter[2] find that the epidemic dysenteries of France are due to the Shiga bacillus. This bacillus has now been identified, not only by its cultural peculiarities, but also by its serum agglutinations, in nearly every country of Europe and in many portions of the United States, and in our island possessions in both the Atlantic and Pacific. Strong and Musgrave[3] report that 766 out of 1,328 cases of dysentery in the Philippines were due to the Bacillus dysenteriæ. Kruse[4] studying the diarrheas of insane asylums, found bacilli nearly related to the Shiga bacillus, but which were not

(1) Beitrage zur differential Diagnose zwischen Verschluss der Pfortader und der unteren Hohlader.
(2) Vaillard and Dopter. Annales de l'Institut Pasteur, July, 1903.
(3) Strong and Musgrave. Report of the Surgeon General U. S. Army, 1900.
(4) Kruse. Centralb. f. Allge. Gesundheitspflege, 1900; Deutsche med. *Wochens., 1900; Deutsche* med. Wochens., 1903.

agglutinated by the serum of an animal immunized by Shiga bacillus, though they were agglutinated by the blood of the patients. Kruse thought there were many—possibly ten species of pseudo-dysenteric bacilli. The work of Vedder and Duval[1] developed no facts sustaining the conclusions of Kruse. Vaillard and Dopter say that the statement should be that there are dysenteries, not that there is dysentery.

Vaillard and Dopter were able to produce dysentery in animals by injecting either the bacillus, the cultures in which the bacilli have been killed, or a filtered culture. According to these writers, experimental dysentery was much more like human dysentery pathologically than symptomatically. The intestinal lesions were identical with those in the human disease. The bacilli were very abundant in the mucosa, especially in the region of the necrotic zones. Vaillard and Dopter found bacilli in the neighboring mesenteric glands in about half their examinations; they found them twice in the spleen and four times in the liver in 25 examinations; they did not find them in the heart's blood.

Summer Diarrheas of Infants. The question of the *etiology of the summer diarrheas in children* has been very materially cleared by the work of Duval and Bassett.[2]

In 42 cases of summer diarrhea of infants, these investigators were able to isolate the Shiga bacillus. This bacillus was found in the stools, in the mucous membrane of the fatal cases, and also in one case in the mesenteric glands and in the liver. The number of Shiga bacilli occupied some relation to the severity of the symptoms. The bacillus was identical with that of Shiga, of Flexner, Strong, Kruse and Vedder and Duval.

They got agglutination where their bacillus was tested with the blood serum of the patients, with the serum of

(1) Vedder and Duval. Centralb. f. Bakt., 1902, 1903.
(2) Centralblatt für Bact., Parasit., etc., Bd. 33, No. 1.

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Dupray. Jour. of Trop. Med. 1902. Flexner, Centralb. f. Bakt., 1902. Shiga. Deutsche med. Wochens., 1901. Gay. Bulletin Univ. of Penn., 1902.

other cases of summer diarrhea, with the serum of adults suffering from acute dysentery, and with antidysenteric immune serum. They did not find the Shiga bacillus in healthy children, in simple diarrheas, nor in the malnutritions nor in the marasmus cases. They conclude that the summer diarrhea of children is a specific disease due to a specific bacillus—the Shiga bacillus.

Woolstein[1] has studied 114 cases of infantile diarrhea with a view to determining the truth of the findings of Duval and Bassett. The stools of ten healthy infants and ten new born babes were examined. The Shiga bacillus was never found in these normal cases.

Of the 114 cases, mostly diarrheas, Shiga bacillus was found in 39. It was found in every case in which blood was found in the stools. It was found in 19 cases in which blood was not present. All of the bacilli reacted with Flexner's Manila serum (Harris) in dilutions from 1 to 50 to 1 to 3000, and with the Shiga serum up to 1 to 200. She never found the bacillus in any of the internal viscera in five autopsies. Rosenthal[2] found but one septicemia in 14 autopsies. Woolstein found the serum reaction uncertain during the first week, frequently positive after the sixth day, but may be absent for two weeks. She does not think that the serum reaction is of value as an early diagnostic procedure. The dysentery bacillus infections have a marked tendency to give a dysentery with mucus and especially bloody stools. The bacillus that she found was almost invariably of the Manila type.

Dysentery Bacilli in the Water. Bergey,[3] as the result of a comparative study of the biologic characters of the water bacteria, concludes that none of them are true dysentery bacilli.

Anaerobic Bacteria of the Intestines. Friedman[4] has studied the anaerobic bacteria of the intestines by tying off the colon at post mortems in twelve cases. Twice he

(1) Journal Medical Research, August, 1903.
(2) Deutsche medicinische Wochenschrift, February 5, 1903.
(3) Journal Medical Research, August, 1903.
(4) *Trans.* Chicago Pathological Society, Nov. 12, 1902.

made use of appendices removed by operation. Once he used a strangulated intestine, and once a peritoneal exudate. He found B. aërogenes capsulatus present 9 times. Rodella's bacillus was present 5 times; B. muscoides of Liborius and B. cadaveris sporogenes, and three undescribed forms were also found. He thinks that the popular conception of the frequency of colon bacillus is due to the fact that cultures from the feces are usually made aerobically. When the contents of an intestinal segment are isolated as by constipation, or by artificial means, or by hernias, the facultative anaerobes are the first to multiply. After a few days, multiplication of the true anaerobes comes into the ascendancy.

Tubercular Appendicitis. Crowder[1] reports a case of hyperplastic inflammation that was histologically tubercular, and was limited to the appendix. He says that the literature now contains reference to 100 cases of hyperplastic tuberculosis of the intestine, but he has been able to find no case in which the appendix alone was involved.

Hyaloserositis. Nichols[2] in his very able study of this condition, arrives at the same conclusions as did Kelly. In his opinion it is a mild inflammatory hyperplasia, due to an infection with some mild bacterium. In this connection the work of Adami and his school, on special infections with B. coli communis, is to be recalled. In Nichols' opinion, tuberculosis sometimes gives a similar, though usually distinguishable, chronic hyperplastic process. This chronic hyaloserositis is generally unassociated with cirrhoses of the subjacent organs. Strange to say, it is more frequently right sided and it has some disposition to confine itself to the lateral half of the body. Found most frequently in the convexity of the liver, it traverses the diaphragm rather readily and appears right promptly in the right pleura and in the pericardium. Later, it may be found involving almost any serous membrane of the body.

(1) American Journal Medical Sciences, August, 1902.
(2) Studies from the Royal Victoria Hospital, Montreal, No. 3. 1903.

Kelly[1] has given a most logically analytical résumé of the condition which Pick had in mind when he wrote of pericarditic pseudo-cirrhosis of the liver. He agrees with Nichols and other recent writers that the usual primary and essential lesion is a mild hyperplastic peritonitis, which extends either directly through the diaphragm or indirectly by way of the central tendon, the mediastinum, and sometimes the general circulation. Later, there is the same character of inflammation in the pericardium and the pleura. In either of the serous membranes there may be, and usually is, a varying amount of exudate. In the pericardium, adhesions are the rule. This is true in a lesser degree in the pleura and still less frequently in the peritoneum. The pericardium is quite prone to calcic deposit. Kelly reports such a case. [In a case seen by the Editor, in the practice of Dr. R. H. Babcock, the calcic deposits occupied the entire pericardium, except along certain hinge lines, which allowed contraction of the heart. In this case there were also calcareous deposits in the substance of the lung.] The great question is as to the etiology of this serositis. Kelly agrees with Nichols that it is due to a mild infection—the sub-infection of Adami. In some cases the infecting bacterium is the tubercle bacillus. In many it is some other bacterium. These produce a hyperplastic inflammatory process beneath the endothelial layer. Considerable effusion is the rule. Adhesions in the peritoneum are the exception, though sometimes they are quite extensive. Should a certain case give a history of a primary ascites that had existed a long time, demanding repeated tapping, the supposition is that the serositis is probably peritoneal. If the history is that of a leg edema, as the earliest watery accumulation, the pericardial form is probable.

Experimental Cirrhosis of the Liver. Wells[2], stimulated by Adler's work[3] on the effect of tobacco on rabbits, *produced cirrhosis of the liver by injecting peptone*

(1) Amer. Jour. Med. Sciences, January, 1903.
(2) Trans. Chicago Pathological Society, March, 1903.
(3) Journal of Medical Research, November, 1902.

sub-cutaneously. He found that the overgrowth of fibrous tissue was not consequent upon degeneration of the liver cells, but was a primary process.

Deformity of the Gall-Bladder. Gay[1] reports a case of developmental deformity of the gall-bladder. The common duct emptied independently of the pancreatic duct. There was an accessory hepatic duct almost as large as the common duct. The gall-bladder measured 3.8 cm. x 2.4 cm., and it showed no signs of any inflammatory process. It was located in the extreme right of the transverse fissure and almost completely surrounded by liver tissue. Gay collected 19 cases of absence of the gall-bladder. It would appear that the gall-bladder is quite uniform in size, shape, and position, as Gay found few reports of such deformity in a comprehensive search of the literature.

Biliary Calculi in the Wall of Gall-Bladder. Peraire[2] records some observations on this subject reviewing the literature critically, beginning with Morgagni; he believes that concretions are formed deep in the wall of the gall-bladder. He does not accept the idea that stones found in that position have been formed in the cavity and have destroyed tissue until they come to lie in the wall.

Effect of Introduction of Foreign Bodies Into the Gall-Bladder. Carmichael[3] placed a solid, non-irritating sterile body in the gall-bladder. Some weeks later he found deposits of calcium carbonate thereon. Cholecystitis did not seem to bear any etiologic relation to the formation of the calculi. In other cases along with the foreign body he placed a culture of coli communis or typhosus. So far as the formation of the concretion was concerned, this did not matter materially. Some weeks later he would recover a good vigorous growth of the inoculated bacterium, generally in pure culture. The precipitate never contained cholesterin. The work was done to verify

(1) Trans. Chicago Path. Society, Vol. VI.
(2) Revue de Chir., July 10, 1903.
(3) Journal of Pathology and Bacteriology, May, 1903.

that of Naunyn and Lubes.[1] He arrives at an opinion contrary to theirs.

Simple Adenoma of the Pancreas Growing From an Island of Langerhans. Nicholls[2] describes a benign tumor, adenomatous in character, growing in the body of the pancreas. It was about the size of a bean. It was yellowish white in color and encapsulated. He was able to find seven cases in the literature. Those of Thierfelder,[3] Neve,[4] and Rugge[5] were probably malignant. Those of Caeseris Demil,[6] Biondi,[7] Baudach[8] and Martin,[9] were probably adenomata. They did not throw very much light on the question of the histologic origin of the tumor. Nicholls thinks that his specimen sprang from the islands for the following reasons: He saw no lumina and no glandular arrangement. With such stains as Van Gieson's, thionin and Mallory's connective tissue, the staining was characteristic of the islands of Langerhans, as distinguished from the other structures of the pancreas. He believes that many of the pancreatic cysts are true cyst adenomata.

Expcrimental Fat Necrosis. Wells[10] produced fat necrosis by injecting into the peritoneal cavity of cats and dogs, weak alkaline, weak acid and plain watery extracts of hog pancreas. Commercial pancreatin produced the same effect. The power was lessened when the extract was heated to 55°C. and it disappeared at 70°C. It is Well's opinion that the splitting of fat was not due to trypsin. It appeared to be due to lipase, though some other ferment seemed a necessity. For example, when fatty tissue anywhere in the body is made necrotic, lipase is present, but the phenomena of fat necrosis do not appear. In the nodules of fat necrosis the first phenome

(1) Klinik der Cholelithiasis. Leipzig, 1892.
(2) Journal Medical Research, November, 1902.
(3) Thierfelder Atlas de Path. Hist. 3 Lief. Leipzig, 1874.
(4) Lancet, Volume 11, page 659, 1891.
(5) Giorrale Internazionale delle Science Med. 1890, No. 12.
(6) Archives per le Sc. Med. 1895.
(7) Biondi. Cagliani P. Valdes. 1897.
(8) Baudach. Inaug. Diss. Freiberg, 1885.
(9) Virchow's Archiv Bd. CXX.
(10) *Trans. Chicago* Pathological Society, Jan. 12, 1903.

non is a simple necrosis which is followed by the specific phenomena of fat necrosis. Soon leucocytic limitation occurs. Repair without adhesions usually takes place in about two weeks. Wells thinks that the enzymes spread along the lymph routes, though they are prone to remain rather circumscribed.

INTESTINAL PARASITES.

Certainly the most important work of the year in human parasitology was the visit of the representatives of the Public Health Service to the Southern States, made with a view of determining the degree of prevalence of uncinariasis, especially in the Southern States. The party started from Washington and traveled to Florida. Stiles[1] was able to find in the literature, by personal observation, and by conversation with physicians, 678 cases, which he thought were probably uncinariasis. While he does not agree with Harris[2] that it is the most common of the serious diseases of the Southern States, nevertheless he thinks it of the greatest importance hygienically and economically. In American Medicine, May 10, 1902, Stiles reports a new parasite known as Unciniaria-Americano. This parasite differs, especially in the arrangement of its buccal cavity, from the old world Uncinaria, known as Anchylostoma duodenalis. It would seem also that it is a less harmful parasite than is that of the old world. There is a greater need for continued infection in order that the disease should assume a grave type. The mature worm is found below the pylorus and generally speaking within the first two feet of the small intestines. Dubini has found the European form in the ilium once. It holds on to the mucous membrane by means of teeth, and it is prone to change position from time to time. Hence there is considerable oozing, and by the blotting paper test (which

(1) American Medicine, Vol. 4, Nov. 15, 1902.
(2) Bulletin No. 10, Hygienic Laboratory. Public Health and Marine Hospital Service.

we will presently describe) blood can be found in the stools in the severer cases.

The constitutional effects produced by the worm are due:

First. To the abstraction of blood.

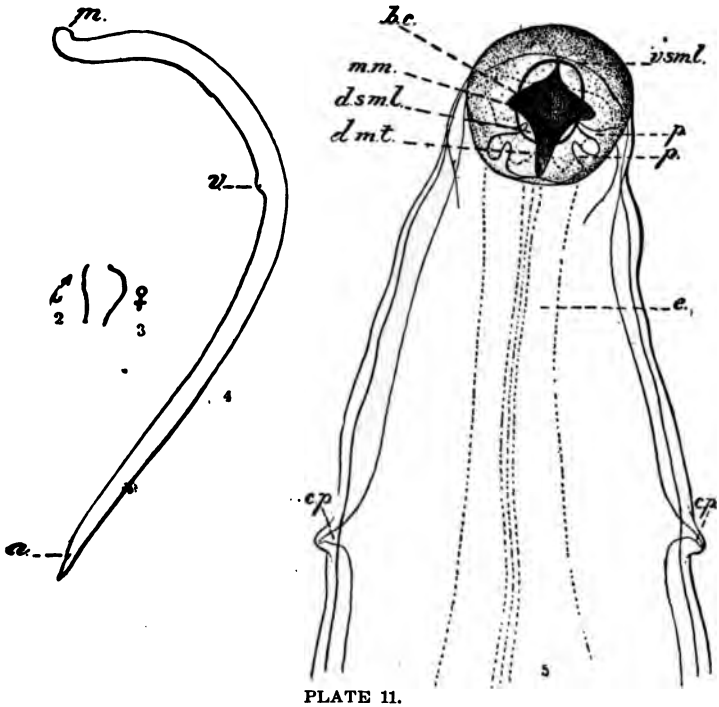

PLATE 11.

Fig. 2. New World male hookworm (Uncinaria americana). Natural size. (After Stiles, 1902b, p. 190, fig. 120.)

Fig. 3. New World female hookworm (Uncinaria americana). Natural size. (After Stiles, 1902b, p. 190, fig. 121.)

Fig. 4. The same, enlarged to show the position of the anus (a) and the vulva (v). (After Stiles, 1902b, p. 190, fig. 122.)

Fig. 5. Dorsal view of anterior end of New World hookworm (Uncinaria americana): b. c., buccal cavity; c. p., cervical papillæ; d. m. t., dorsal median tooth, projecting prominently into the buccal cavity; d. sm. l., small dorsal semilunar lip; e., esophagus; m. m., margin of mouth, the prominent oval opening seen upon high focus; p. p., papillæ; v. sm. l., large ventral semilunar lips homologous with the ventral hooks of A. duodenale. Greatly enlarged. (After Stiles, *1902b, p. 190,* fig. 123.)

Second. To the bleeding from the bites.

Third. To chronic inflammation in the intestinal walls and secondary degeneration in the intestinal glands brought about by the bite of the parasite.

Fourth. Possibly, to certain toxic substances secreted by the parasite.

The female lays eggs from time to time, but these never hatch within the host: therefore, the patient never infects himself, as is the case with certain other parasites—for instance, oxyuria. The eggs escape in the feces where they hatch within 24 hours, if the feces are kept warm, and within 48 hours at a lower temperature. The egg is about 60 mikrons in size, has a gelatinous capsule, and usually when seen is apparently segmenting. The young larvæ are about .3 mm. in length and move by an active worm motion. At the end of two days this larva sheds its skin, becoming larger and acquiring a more elaborate buccal opening. After five days it casts off its second skin. It does not stand water well during its first days of larval life, but during its secondary larval stage it lives freely in water.

This ends the free life of the parasite. It finds its way into the intestinal tract, in a way to be described presently, and there passes through three more larval stages before it becomes a mature worm. After casting off each of these skins it becomes larger and larger, and its buccal opening becomes more complex. The time required to reach maturity after being taken into the stomach, according to Stiles, is from four to six weeks. Looss[1] notes that 71 days elapse from the time that larvæ are taken into the stomach until eggs appear in the stools. The extra-corporal portion of the cycle does not consume over one week, where the conditions are just right. Certainly one of the most important of Stiles' observations was that a sandy soil is perhaps a necessity. Uncinariasis was probably never found except in those who had lived, or

(1) Centralblatt f. Bacteriologie, Parasit., etc. Vols. 33, 31, 29, 24, u. 21.

were living on a sandy soil. A freezing temperature destroyed both eggs and larvæ. Neither stood drying well. Too much water especially in the stools destroyed the eggs, but did not harm the larvæ, especially in its second stage. Our illustrations taken from Stiles' bulletin show

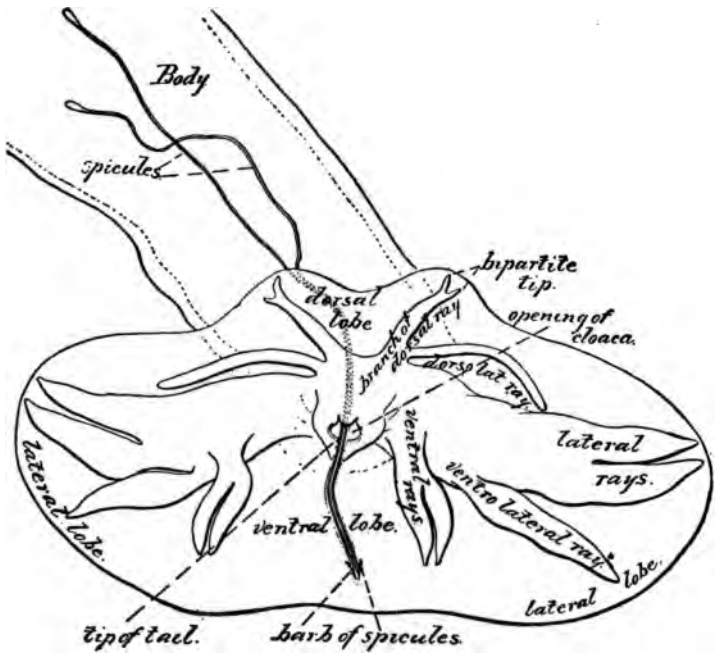

Fig. 12. The caudal end of the New World male hookworm (Uncinaria americana). The bursa is spread out to show the arrangement of the rays. Note the short dorsal lobe which is subdivided, forming two lobes; note also the indistinct ventral lobe connecting the two lateral lobes. The dorsal lobe is thrown back over the body. Greatly enlarged. (After Stiles, 1902b, p. 191, fig. 126.)

the eggs of different parasites which are liable to inhabit the human intestine in the United States, the larva form of uncinaria and the head of the mature form.

In symptomatology Stiles, after detailing the known conditions of the so-called dirt eater, adds a new symptom. *If you will have* the subject look steadily into your eye

his pupils will dilate and he will stare like an epileptic recovering from a seizure. Stiles does not state his belief as to the mode of infection, but he devotes a good deal of space to the possibility of infection through the skin, as set forth by Looss[1] and by Bently[2]. He thinks that while "ground itch" or "dew poison" may be manifestations of infection by a skin route, it is probable that the usual route is by larvæ taken by way of the mouth. A very important question is that of continuance of infection, for uncinariasis in this country is probably milder than it is in Egypt or in Europe. As the eggs never hatch within the intestines either new larvæ must be taken in

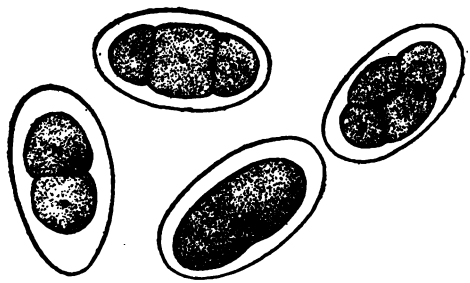

Fig. 13. Four eggs of the New World hookworm, Uncinaria americana, in the 1, 2, and 4 cell stages. The egg showing 3 cells is a lateral view of a 4-cell stage. These eggs are found in the feces of patients and give a positive diagnosis of infection. Greatly enlarged. (After Stiles, 1902b, p. 192, fig. 127.)

from time to time or the disease will be limited to the life period of the adult parasite. It is probable that most of the cases to be found amongst the "crackers" are cases of continued infections. Stiles thinks that in certain of his cases the adult parasites had lived in the intestines for six to seven years. This is especialy important where the infected patient has moved to a non-infected locality. Such a case may be a source of infection to a new district for years after his arrival.

(1) Loc. Cit.
(2) British Med. Jour., Jan. 25, 1902.

His method of examining the stools is as follows:

First. Blotting paper. An ounce or two of fresh feces is wrapped in blotting paper. Let it stand 20 to 60 minutes; remove the feces: if uncinaria are present small blood stains will be found on the blotting paper.

Second. Direct microscopic. Take a small bit of

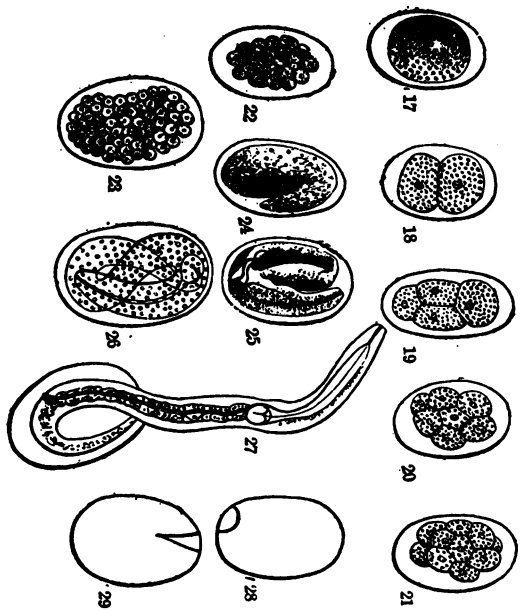

PLATE 14.

Figs. 17-29. Embryology of the old World bookworm (Anchylostoma duodenale) of man; 17-23, segmentation of the egg, 24-26, the embryo; 27, a rhabditiform embryo escaping from its eggshell; 28-29, empty eggshells. Greatly enlarged. (After Perroncito, 1882, p. 342, fig. 142.)

feces about the size of the head of a pin, add a drop of water, spread very thin—now add a cover glass. Examine for eggs with a magnification of about 60 diameters—a low power lens.

Third. Sedimenting method. Put an ounce of feces in

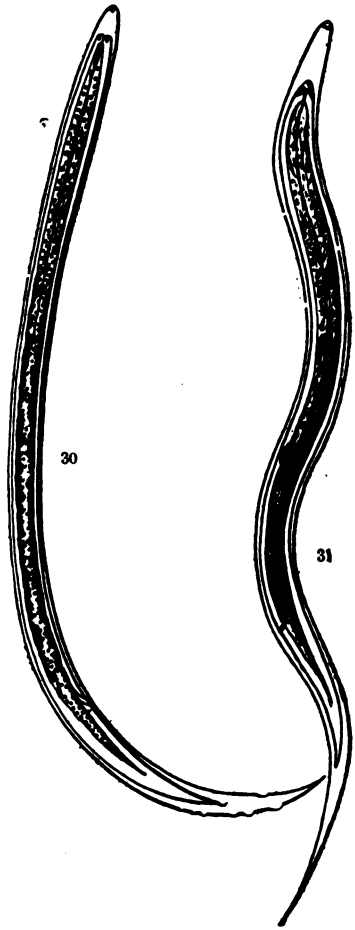

PLATE 15.

Figs. 30-31. Two larvæ of the Old World hookworm at the end of the second stage ("encysted larvæ"), representing the young worms retracted from their skin. (After Perroncito, 1882, p. 350, figs. 145 a-b.)

PLATE 16.

Fig. 43. Egg of the common ascaris (Ascaris lumbricoides) of man, as found in feces. Seen with superficial focus. Greatly enlarged. (After Stiles, 1902b, p. 202. fig. 158.)

Fig. 44. The same, as seen with median focus. Greatly enlarged. (After Stiles, 1902b, p. 202, fig. 159.)

Figs. 45-54. Embryology of the common ascaris (Ascaris lumbricoides) of man, showing the changes undergone by the egg after being discharged in the feces. (After Leuckart, 1867, p. 213, fig. 154.)

Fig. 55. Embryo of the common ascaris (Ascaris lumbricoides) of man, in the eggshell. (After Leuckart, 1867, p. 215, fig. 156.)

Fig. 56. Free embryo of the common ascaris (Ascaris lumbricoides) of man, casting its skin. (After Leuckart, 1867, p. 214, fig. 155.)

Figs. 57-64. Embryology of the common pinworm (Oxyuris vermicularis) of man, showing the changes undergone by the egg while in the female worm. (After Leuckart, 1868, p. 322, fig. 191.)

Fig. 65. Embryo of the common pinworm (Oxyuris vermicularis) of man, in the eggshell, as found in fresh feces. (After Leuckart, 1868, p. 328, fig. 196.)

Fig. 66. Full-grown embryo of the common pinworm (Oxyuris vermicularis) of man, after it has escaped from the eggshell. (After Leuckart, 1868, p. 328, fig. 195.)

Figs. 67-70. Egg of the common whipworm (Trichuris trichiura) of man, showing changes undergone while still in the female worm; fig. 70 is the stage found in fresh feces. (After Leuckart, 1868, p. 491, fig. 275.)

Figs. 71-73. Later stages of development of an allied whipworm (Trichuris affinis) of sheep and cattle, showing changes after the egg escapes in the feces. (After Leuckart, 1868, p. 494, fig. 276.)

Fig. 74. Isolated embryo of Trichuris affinis. (After Leuckart, 1868, p. 495, fig. 277.)

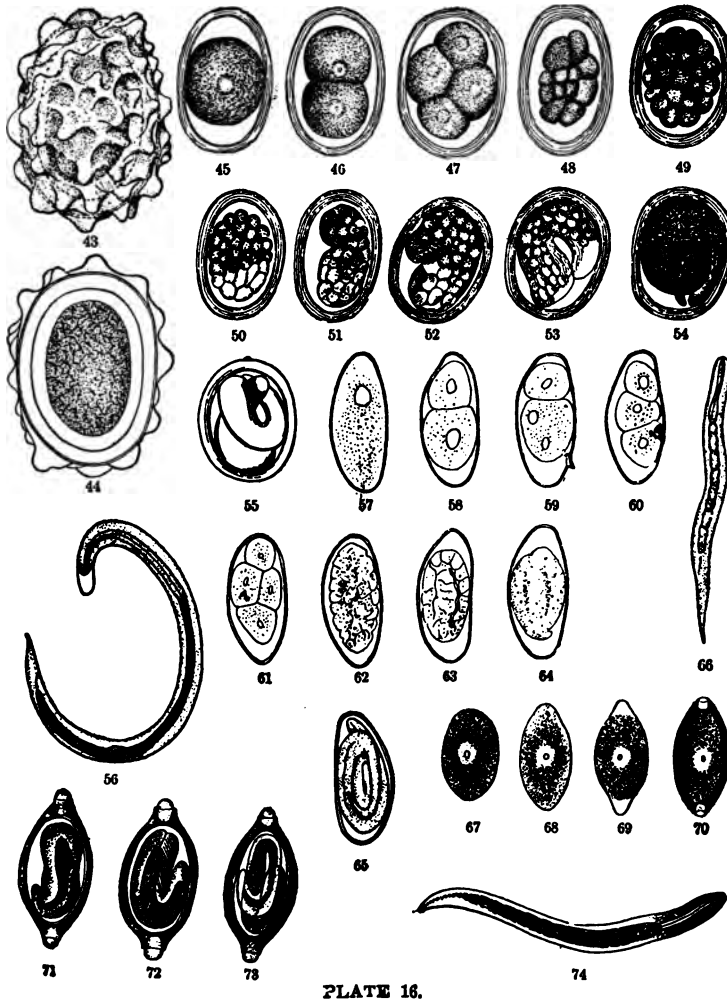
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PLATE 16.

PLATE 17.

Fig. 78. Egg of the common liver fluke (Fasciola hepatica) examined shortly after it was taken from the liver of a sheep; this is the same stage that is found in human feces; at one end is seen the lid or operculum, o; near it is the segmenting ovum; the rest of the space is occupied by yolk cells which serve as food; all are granular, but only three are thus drawn. X 680. (After Thomas, 1883, p. 281, fig. 1.)

Fig. 79. Egg of the common liver fluke containing a ciliated embryo (miracidium) ready to hatch out; d, remains of food; e, cushion of jelly-like substance; f, boring papilla; h, eye-spots; k, germinal cells. X 680. (After Thomas, 1883, p. 283, fig. 2.)

Fig. 80. Embryo of the common liver fluke (Fasciola hepatica) boring into a snail. X 370. (After Thomas, 1883, p. 285. fig. 4.)

Fig. 81. Egg of lancet fluke (Dicrocœlium lanceatum) with contained embryo. X 700. (After Leuckart, 1889, p. 379, fig. 171.)

Fig. 82. Egg of human blood fluke (Schistosoma hæmatobium) with contained embryo, passed in the urine or in the feces. X 285. (After Looss, 1896, pl. 11, fig. 112.)

Fig. 83. Egg of beef-measle tapeworm (Tænia saginata) with thick egg-shell (embryophore), containing the six-hooked embryo (oncosphere) enlarged. (After Leuckart.)

Fig. 84. Eggs of pork-measle tapeworm (Tænia solium): a, with primitive vitelline membrane; b, without primitive vitelline membrane, but with striated embryophore. X 450. (After Leuckart, 1880, p. 667, fig. 297.)

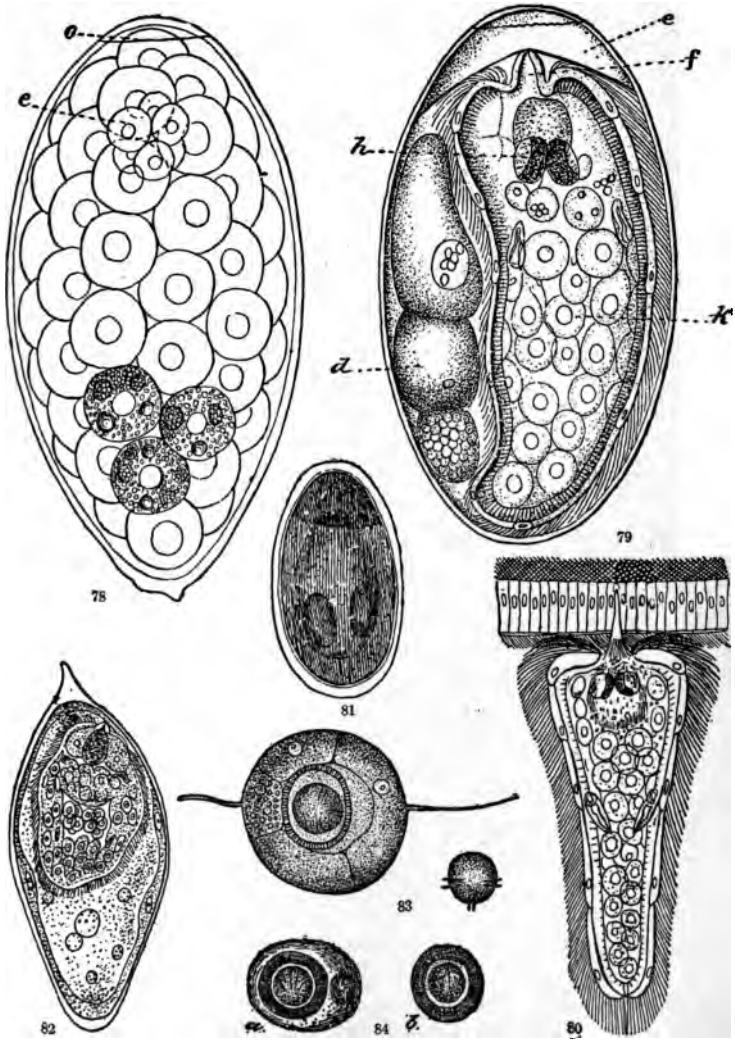

PLATE 17.

a jar. Leave for 24 hours, add a quart of water and mix well. Let it settle for a while and then pour off all that floats. Repeat this several times. Examine the fine sediment at the bottom. Eggs and larvæ will be found in this sediment.

Fourth. To search for mature worm. Give a small dose of thymol (10 grains) and follow by a dose of salts. Wash all feces several times in a bucket of water. Examine sediment for worms about half an inch in length, and about the size of a hat pin.

Capps[1] reports the following case: During the fall of 1902 a man came into the wards of Cook County Hospital after leaving Central America some time before because of a progressing anemia. He had been in several hospitals in the South where his disease was not recognized. The anemia was found to be of a secondary type. Eosinophilia was present. The abdomen was greatly distended. This combination of symptoms, together with the absence of an adequate cause for the anemia, directed attention to the stools. Eggs and larvæ were found, and subsequent to the administration of thymol, mature worms appeared. At the death of the man a post mortem was performed by Harris: uncinariæ were found in considerable numbers in the jejunum, and some were found in the ilium. Capps found that the eggs did not hatch nor the larvæ develop if the air was excluded from the feces, but if the feces were kept in the incubator at 27°C., tightly corked and tightly stopped, whereas there would be no growth when the cork was in place, there would be a growth after it had been removed. Very little pathology was found except the small hemorrhagic spots in the small intestines. Sippy found eggs of strongyloides in this case also.

That uncinariasis in the U. S. is not a very serious disease is proven by a statistical study of the intestinal parasites in 500 white male patients at the U. S. Hospital for the Insane by Garrison, Ransom and Stevenson;[2] un-

(1) Jour. American Medical Association, Jan. 3, 1903.
(2) *Bull. No. 13* Hyg. Lab., Pub. Health and Mar. Hosp. Serv.

cinariæ were found in 15 cases or 3 per cent of the total number. Brown[1] reports the case of a Porto Rican who had uncinaria eggs, strongyloides larvæ, and trichiuris eggs. This patient had a moderate anemia, 75 per cent hemoglobin, and he had an eosinophilia of 10 per cent. His other symptoms were some diarrhea, and some indefinite abdominal tenderness and pain. He reported a second case in a Porto Rican, who had no symptoms except certain vague abdominal pains, and an eosinophilia of 5 per cent. His stools showed larvæ of strongyloides, and eggs of trichiurias and, probably, uncinariæ. [Within the last two weeks the writer has seen a case of mild infection with uncinaria. The patient, a lady recently from

Fig. 18. Egg of the dwarf tapeworm (Hymenolepis nana) of man. Greatly magnified. (After B. H. Ransom, unpublished.)

Mississippi, had a hemoglobin of 45 per cent, and red cells 3,400,000; there was no eosinophilia. There were no intestinal symptoms. Eggs and larvæ in small numbers were found in the stools.

In a statistical study of 500 white male patients at the United States Hospital for the Insane, Garrison, Ransom and Stevenson[2] found that 13.2 per cent of the patients were infected with intestinal parasites. The percentage of those infected was found to be largest amongst the people who had recently returned from the new island possessions of the United States Government. Uncinaria was found in 15 cases. Trichiuris in 54, oxyurias in 4, strongyloides in 3, and ascarides lumbricoides in 2. It

(1) Boston Medical and Surgical Jour., May 28, 1903.
(2) Loc. Cit.

appears, as Stiles[1] thinks, that ascaris lumbricoides is becoming less frequent in this country. Brown[2] has reported three cases of strongyloides infection. Two of them have already been referred to. The third was a native of Tennessee, who had lived in California for 30 years, and had never been in any foreign land. His blood showed hemoglobin 65 per cent, and red cells 76 per cent. He had an eosinophilia of 6.33 1-3 per cent. The patient having died of inter-current pneumonia, necropsy was held. This showed the worms from just below the pylorus to the ascending colon. The parasite was attached to the mucous membrane as deep as the muscularis mucosa, but not into it.

The first case of strongyloides infection resulting in Cochin China diarrhea found in this country was that of Strong.[3] Thayer[4] reports two strongyloides cases, in both of which the infection had probably occurred within the United States.

After citing the statements of Grassi, Perroncito, Sahli, and Lutz, that strongyloides and uncinaria were very frequently found associated, he makes the statement that the discovery of Strongyloides intestinalis should emphasize the possibility that uncinariasis may also occur in this country.

GENITAL ORGANS.

Chronic Cystic Mastitis. Greenough and Hartwell[5] have made almost serial sections of thirty mammary glands, of which twenty-seven were not malignant. The question to which they have addressed themselves especially is the condition of fibrous tissue overgrowth, usually circumscribed and cystic that occurs in women about the menopause. In their opinion, cystic mastitis is the most appropriate term to use. They do not believe this inflam-

(1) Loc. Cit.
(2) Loc. Cit.
(3) Thayer, Journal Exp. Med., Vol. 6, No. 1.
(4) Johns Hopkins Hospital Reports, 1901, Vol. X.
(5) *Journal* of Medical Research, June, 1903.

mation to be bacterial. In this they disagree with Binard and Braquehaye.[1] They believe that neither clinically nor histologically can these tumors be differentiated absolutely from true carcinoma, into which possibly they very frequently run. In the most benign of them, they found not only new glands, but adenoma formation and papillary cyst growth in some areas. Not only did they find this range of epithelial arrangement grading from simple glandular through cystic forms to papillary masses, but they found epithelium of all kinds from low cubical to tall columnar. They do not agree with Paul[2] that a tall columnar cell is not a reproducing cell, and is therefore a thoroughly benign cell.

As to the vexed question of the relation of the epithelial overgrowth to that of connective tissue, their views are akin to those of Ribbert. They consider it a reactionary hyperplasia of epithelium placed under abnormal conditions. Such masses are very prone to eventuate in glandular carcinoma; they do not think there is any disposition toward any other form of carcinoma. Fortunately glandular carcinomas of the breast are only mildly malignant.

Carcinoma of the Uterus. Ries[3] is of the opinion that carcinoma of the uterus is very prone to extend to the pelvic lymph glands. In invading such a gland the primary growth is at the hilum, and along the connective tissue structures. Invasion of the lymph tissues proper is a later phenomenon. On four occasions Ries has noticed columnar or cubical celled epithelial ducts in carcinomatous lymph glands. At least one of these was in a case of squamous celled carcinoma. He is of the opinion that they are due to Wolffian body rests. Ries has observed hemolymph glands in the pelvis. Warthin[4] has verified this observation. Ries is of the opinion that carcinoma metastases can be spread in this way to the blood current.

(1) Dentu et Delbet, Traite. de Chirurgie, Vol. vii., Paris.
(2) British Medical Journal, Dec., 1900, and Trans. London. Path. Soc., Vol. lii., p. 30.
(3) Trans. Chi. Path. Society, Vol. V., No. II.
(4) Trans. Chi. Path. Society, Vol. V., No. II.

Sampson[1] thinks that the pelvic glands are very prone to become infected in carcinoma of the cervix. One hundred and forty-three cases operated on at Johns Hopkins gave a primary mortality of 14.6 per cent. Three or more years after the operation they had learned of recurrence in 79.7 per cent of the cases that they were able to follow. In those cases kept under observation for five or more years recurrence had taken place in 87.7 per cent. In 30 of Wertheim's[2] 90 cases the glands were proven by serial section to have been involved. Doederlein[3] reported the same result in 18 out of 26 cases studied. Clark quoted by Sampson does not think that the glands are often involved. When they are he thinks it of prognostic rather than diagnostic importance, as he thinks that general metastasis is sure to occur. With this Wertheim does not agree. Sampson thinks that the pelvic lymph glands are involved in one-third of the operable cases.

Cysts Near the Fallopian Tube. Dickson[4] has studied those small subperitoneal cysts lying in the broad ligament structures around the tubes, and not developing from the Wolffian bodies, the ducts of Mueller, Gaertner's ducts, or any of their branches. These cysts bear some resemblance, so far as their naked eye appearance goes, to the cysts from Rosenmüller's tubes, but they are seldom of any size, or have any clinical interest for that matter. The attention they attract is simply in the matter of differentiating them from other bodies. They are generally multiple. Dickson believes that they are due to dilatation of the lymphatics.

Adrenal Tumors. Wooley[5] has critically studied the tumors of the adrenal gland. Amongst the benign tumors, he thinks that we probably have fibroma and lipoma. He inclines to the idea that the cases reported by Virchow and Marchand as gliomata and those by Weichselbaum, Armanni and Formad, as neuromata, were possibly mis-

(1) Johns Hopkins Hospital Bulletin, Dec., 1902.
(2) Archives of Gyn., 1900 and 1901.
(3) Centralblatt f. Gyn., 1902.
(4) American Journal of Obstetrics, July, 1903.
(5) American Journal Medical Sciences, Jan., 1903.

takes in diagnosis. The more complex tumors were cysts, angiomata and adenomata. Wooley thinks it probable that many of the cysts are adenomata, in which hemorrhage has taken place.

The primary malignant tumors of the adrenal, Wooley thinks are carcinomas and sarcomas. By reason of the mesothelial origin of the adrenal, a morphologic basis for such tumors is very unsatisfactory. They stand midway between epithelium and connective tissues and are liable to partake of the character of either. The usual metastasis was *via* the blood to the liver and lung. Those called carcinomas are very much like sarcomas in their cells and in their arrangement.

The author has found twenty-two cases in the literature. His bibliography is very complete and well digested.

TERATOMATA.

Testicular Teratomata. Carey[1] reports *two cases of teratoma* to which in his report he adds summaries of the conditions in twenty-four other cases gathered from the literature. These tumors usually develop in adult life. Sometimes there is a history of trauma, though there is probability that its relation to the growth of the tumor is fortuitous.

The tumors generally spring from the rete. They form metastases, but these are by continuous growth. In the opinion of Carey, in teratomata, single cells cannot form metastases by discontinuous growth. Such tumors are usually cystic. The consistency varies according to the contents. Bony or cartilaginous areas can usually be made out. The cysts sometimes contain hair, teeth and fatty debris. Microscopically, every kind of tissue has been found in a heterogeneous mass. Muscle and nerve tissue are jumbled up with bone and internal glands. It is the rule to find evidence of the presence of each of the genetic

(1) Johns Hopkins Hospital Bulletin, November, 1902.

layers. Placental tissue has sometimes been found. Carey discusses five theories, as to the origin of these tumors. He refers to the theory of metaplasia as having been abandoned at the present time. The second theory is that of Meckel, that two ova are impregnated, one preying upon the vascular supply, as Thoma contends, of the other and overshadowing it. While Carey does not discuss this theory as a whole, he thinks that Klebs' sub-division into (1) preallantoid, in which one fetus develops within the other, and therefore has no placenta, and (2) allantoid, in which the inclusion is not so complete and there is no placenta, would not fit the facts in one of his cases. The third theory is that of cell inclusion, as advocated by Bonnet. The idea of Bonnet is that a fertilized polar body, or a few segmenting cells become separated from their proper habitat and are caught in the testis antecedents. He quotes Roux and others as proving that a single cell up to the sixteen cell period of segmentation can develop a complete fetus. Carey thinks this the most plausible explanation. The fourth theory is that the cells develop from the proper tissue cells of the testis. The fifth theory assumes the presence of an ovum in the testis, which later becomes impregnated and develops into a fetus.

As to the malignancy of these tumors, Carey thinks that we must accept the histologic findings with a great deal of caution, since ordinary indications of malignant tendencies are more or less physiologic in such embryologic structures. He thinks that they are sometimes malignant. The metastasis may be of any one of three genetic layer structures. Perhaps at times more than one kind of cell can undergo metastases. In his first case, the patient died of a tumor in the vicinity of the pancreas. This tumor was probably a sarcoma.

Jung[1] reports two teratomas of the ovary; one of them was very largely cystic and the other was cellular. Each of them contained cells from every genetic layer. In one brain tissue, with a central canal lined by ciliated epithe-

(1) *Centralblatt* f. Gyn., 1902, No. 15.

lium was demonstrated. He does not think that these tumors are malignant. The ordinary histologic picture of a malignant tumor is unreliable in any rapidly growing embryo. Further than this, however, it is his opinion that metastasis does not constitute evidence of malignant character. He quotes the observation of Pick of a metastasis of a hydatid mole and Minkowski of metastasis of a uterine myoma. In one of his cases, certain small white peritoneal nodules that might have been regarded on superficial observation as metastases were found by microscopic examination to be obliterated blood vessels. He collects twenty cases from the literature, twelve of which were regarded as sarcomas.

THE NERVOUS SYSTEM.

Changes in the Spinal Cord and Medulla in Pernicious Anemia. Billings[1] reports 41 cases of pernicious anemia. In his analysis of the symptoms stress is put on those referable to the nervous system. In three of the cases autopsy was made and the cord was carefully studied.

In the first case there were no nervous symptoms, except numbness and tingling. In the second, ataxic phenomena were much more prominent. In the third, disturbance of the senses and of motion was pronounced.

The lesions found were scleroses involving the posterior and lateral columns. These areas were very irregularly distributed. The sclerosis was sometimes diffuse and sometimes focal. Lesions of these two kinds were sometimes found coexisting in the same cord. Generally, the process tended to diminish toward the crura. The entire neuron was not involved. Sometimes degeneration in the axis cylinder was seen alone; more frequently there was glia increase and connective tissue overgrowth. Perhaps the most striking fact noticed was the irregularity of the lesions. The changes in the gray matter were minor.

(1) Chicago Medical Recorder, Jan., 1903.

Billings concludes:

1. There is a well established relation of diffuse cord degeneration with pernicious anemia.

2. It seems probable that the hemolysis and the cord changes are due to the same toxin.

3. While the source of the toxin is unknown, the fact that gastrointestinal disturbance is so common in the disease would lead one to suppose that it is of intestinal origin.

4. The diffuse degenerations of the spinal cord which occur in conditions without pernicious anemia do not appear to differ essentially from those of pernicious anemia.

5. It is possible that a common blood circulating poison exists which may expend its force upon the blood in one individual, upon the nervous apparatus in another, and coincidently upon the blood and spinal cord in others.

Secondary Carcinoma of Brain, Cerebellum and Spinal Cord. Gallvardin and Varny[1] collect 1,497 cerebral tumors from the literature. Eighty-five of these were carcinomas. They cite Buchholz[2] as having established more than a probability that all cerebral carcinomas are secondary; with this they agree. In the 64 cases in which they were able to determine with certainty the primitive site of the carcinoma, they found 24 in the breast and 12 in the lungs. No other site furnished a preponderating number. In studying the location of the secondary carcinoma in 68 cases they find the cerebrum affected 65 times, 49 times alone. The cerebellum was the site of 13 cases, in 2 of which it was the sole encephalon seat. In three cases the mesencephalon was affected; in two of these it was the only part of the brain affected. Four times the secondary carcinoma was located in the spinal cord.

Multiple Sarcomatosis of Central Nervous System. Intra-Medullary Primary Sarcoma of Spinal Cord. Spiller and Hendrickson[3] report three cases. In one there was

(1) Revue de Medicin, Paris, July, 1903.
(2) *Monat*schrift für Psych. und Neurol., 1898. Bd. iii., iv.
(3) *American* Journal Medical Sciences, July, 1903.

a large sarcoma of the cerebellum and two in the thoracic cord; the pia was involved in several places.

In the second, there were multiple tumors in the cerebellum and at the base of the cerebrum.

In the third case a primary sarcoma was found in the thoracic cord.

Schlesinger,[1] quoted by Spiller and Hendrickson, was able to collect twenty cases of primary sarcoma of the central nervous system in 1898. Involvement of the cerebellum occurred nine times; involvement of the pia of the cord, especially rather low down, was frequent. Schlesinger was able to collect thirteen cases of primary sarcoma of the spinal cord; Spiller's makes fourteen.

Myositis Ossificans.

Taylor[2] reports two cases of ossifying myositis. One was consequent upon a considerable trauma. The second began when the patient was nine years old. It progressed slowly for several years and at the time of observation had existed for 31 years.

The reports on the specimens made by Coplin and Spiller showed the usual findings. There does not seem to be any necessary relation between this condition and trauma,—in fact, no etiology is known. [The Editor has seen one case in which the ossifying process took place in the wound of an operation for radical cure of hernia. The wound had been carefully protected against trauma except of course the trauma of operative procedure. The bone had developed within three weeks of the time of operation.]

(1) Beitrage zur Klinik der Ruckenmark und Wirbeltumoren.
(2) Annals of Surgery, June, 1903.

SECTION III.

BACTERIOLOGY.

GENERAL.

One of the most noteworthy contributions to bacteriology during the year is the Huxley lecture on general immunity, by Prof. William Welch.[1] This is of special interest to medical men, because the present trend of thought and study is so clearly outlined. The lecturer reviews most of the now well known phenomena of immunity, and then brings forward several views regarding which there has been a vague conception, but no outspoken statement. From a purely chemical standpoint, some bacterial toxins have been separated into constituent poisons, but a further complexity is now apparent; this must be worked out along physiologic lines. The intermediary bodies, or atom groups, that link the complements to the cell structure, must be separated if possible, and subjected to individual experiments. Further, the variations in the action of bacterial poisons may depend greatly upon the nature of the atom group with which it is brought in contact when applied to the experiment animal; this will undoubtedly lead to the discovery of substances in a measure toxic, in bacterial cultures now considered entirely devoid of such properties.

The hemolytic powers of bacterial cultures deserve more study, especially as regards this action within the body. The author suggests a causal relation between such action and the secondary anemias that appear during and after many bacterial infections.

(1) *British* Medical Journal, Oct. 11, 1902.

The practical value that knowledge as to the antibodies in blood serum will have to physicians and surgeons is considered. The development of methods for determining the total germicidal power of the blood in the human subject in health and disease is urged. Further, there is great need of an exact determination as to the number and kinds of combining forces in human serum. The essayist draws attention to the variability in the capacity of the body to assimilate the factors that give immunity, speaking particularly of this in its bearing on the disease resistance of young children. The babe appropriates the mother's immune bodies in the milk, and should not be deprived of them through a milk that has been artificially treated.

The door has also been opened that may lead to an explanation of the normal activity which controls cell multiplication. The production artificially of auto-cyto-toxin has shown that in the processes involved in the destruction of cells within the body, this property of cell destruction is developed. In this direction, the self-digestion or autolysis of inflammatory exudates can be taken up with renewed interest. How is the production of these agencies or ferments controlled, and can it be unbalanced during pathologic states and the self-formed poison lead to anemia, interstitial inflammations, uremia, etc.? Methods of bacterial study, especially as regards pathogenesis, must now be conducted on these newer lines; the minute and the distant effect of the parasite on the host are the open possibilities that can explain the phenomena of our clinical observations.

PATHOGENIC BACTERIA.

Cholera. Bacteriologic Diagnosis. The past year shows very little as regards special study of the Vibrio choleræ. Recently the results of an extended investigation of diagnostic methods and the various organisms isolated during

the cholera epidemic in Egypt in 1902 are reported by Kolle and Gotschlech.[1] About 1,000 individual observations make up the material for the deductions. It was found that cholera-like organisms could be isolated from the stools much more frequently than is ordinarily supposed. The method of peptone salt solution was used to start the cultures as has been the prescribed procedure for many years. The finding of these irregular forms may make one accept such a culture as V. choleræ and diagnose the case as cholera, or it may be isolated in place of the V. choleræ which is also present and is the real cause of the sickness. The writers found that it was very difficult to distinguish strains of vibrios by cultural methods, but that the serum reaction was infallibly specific in its distinctions. Sixteen irregular forms were studied and it was found that the V. choleræ was not agglutinated in higher dilutions by any of the sixteen immune sera prepared from these cultures, while the reverse was also true in that a specific cholera immune serum did not agglutinate any of these irregular strains. The serum reaction is therefore lauded as a specific means of differentiation, and on account of the results the absolute etiologic relation of the Vibrio cholera asiaticæ to cholera is established. The same results were obtained with each of 30 cultures of cholera spirilla isolated at this time. The serum reaction is a most important adjunct to the practical bacteriologic diagnosis. As a procedure for diagnosis the following is recommended: Specimens from the stools are cultivated in the incubator on peptone salt solution for eight hours; agar plates are now made, and these should show colonies after a further eight-hour incubation. The bacteria in the resulting colonies are now tested by the serum reaction. It is absolutely necessary at this point to have an immune serum of high value and entirely specific. The diagnosis in this way can be completed in 16 hours.

Diphtheria. *Cultivation of B. Diphtheriæ.* Bosse[2] re-

(1) Deutsche med. Wochens., No. 30, 1903.
(2) Centralb. f. Bakt. I Abt. Orig. Bd. xxxiii, No. 6.

ports certain comparative tests that he made with Loeffler's blood serum mixture and the Deycke pepsin-trypsin agar medium; this medium is prepared as follows:

One hundred and twenty-five grams of fresh and finely ground horse heart, 3 grams pepsin and 400 c.c. distilled water are digested in the incubator for two days; the mixture is then filtered, neutralized with Na_2CO_3 and sterilized. Pancreatin is now added, and a further digestion of six hours in the incubator allowed. It is now neutralized with NaCl and 6 grams salt, 39 grams agar and 1,950 grams water are added. Solution is accomplished by boiling and the medium is filtered through cotton. It is filled into retainers for use. Ordinary tube, streak or plate cultures may be made.

Other bacteria are somewhat restrained on this medium, while the diphtheria bacillus shows active growth. In a number of instances, the writer reports that no growth in 24 hours was apparent in the absence of diphtheria. Colonies of the diphtheria bacillus appear as yellow brown dense areas, coarsely granular, with somewhat irregular margins; the older colonies are darker in the center. When colonies are confluent, they take on polygonal forms, thus causing a peculiar checkerboard appearance. The observations are drawn from 200 diagnosis cultures.

Morphology. Some interesting observations on the morphology of B. Diphtheriæ and the allied members of this group of bacilli are reported by Denny.[1] He finds that when cultures are examined systematically every few hours during the period of growth on media, that the appearance of the individuals changes as the period of growth progresses. In diphtheria the young cultures show mostly individuals staining evenly, 12 hours, while later the polar and granule staining becomes apparent. Still later the irregular and fully developed forms appear. These appearances must be taken into account in distinguishing the diphtheria bacillus in early cultures from the pseudo forms. The strongly barred appearance of the stained

(1) Amer. Pub. Health Report, Vol. XXVIII, 1902.

Xerosis bacillus is especially to be distinguished in the older cultures. Speaking of pseudo-diphtheria bacilli, the writer states that in very early cultures they cannot be distinguished from true diphtheria, but that longer incubation will always settle the question.

From time to time, irregular forms of diphtheria organisms are described; the branching forms are noted with some frequency. Spirig[1] carries the point a step further, in that he describes appearances closely resembling hyphæ or aerial mycelia in certain cultures that he studied. These cultures as they became older formed a fine granular and elevated area in the center of some of the colonies; the resulting appearance was much like actinomycosis cultures. Closer study of these colonies showed the presence of many cells with branches that were evidently projecting over the substratum. The writer also describes a mycelium form of organism apparently intermediate between diphtheria and actinomycosis, that gave degeneration or fragmentation forms, at times resembling typical diphtheria bacilli, and again cocci. These organisms did not produce pathogenic changes in animals. Complete descriptions of the several organisms are presented.

Dysentery. Gay[2] reports his experiments on vaccination and serum therapy in dysentery. The experiments are based upon guinea-pig experiments, and certain injection experiments on horses. The strains of dysentery bacilli of Flexner, Shiga, Kraus and Duval were used. The minimal lethal dose was determined by animal passage, and was found to be about one loopful from a slant agar culture. The animals die either with acute peritonitis (peritoneal inoculations) and septicemia, or more slowly with symptoms of toxemia. Subcutaneous inoculations require larger doses in order to cause death. A vaccine was prepared from such culture, by adding 0.5 per cent tricresol; this will kill the cultures in about two hours. The injurious effect of heating such cultures is

(1) Zeits. f. Hygiene, Bd. 43, No. 3.
(2) Univ. of Penn. Medical Bulletin, November, 1902.

avoided. From 4 to 8 c.c. of the vaccine suspensions was found to be the minimal lethal dose for guinea-pigs. The protocol of experiments is given. Guinea-pigs were injected several times with the vaccines as described, and then inoculated with virulent cultures.

Guinea-pigs which have received one or more subcutaneous injections of subminimal lethal doses of dysentery vaccines show a marked protection against multiple intraperitoneal lethal doses of the living organism. The amount of vaccine used at each inoculation varied from 1.5 to 6 c.c. according to the toxicity of the suspension used. Successive inoculations followed in periods of from seven to fifteen days, or, in other words, when a return to the normal weight was nearly or wholly reached. In the following collected series of experiments, the animals were vaccinated several times and then subjected with controls to exactly similar conditions of infection with varying multiples of M. L. D. of living cultures freshly isolated. Several entire series of experiments have been grouped for convenience. From these experiments with the Shiga vaccine we see that animals inoculated two or more times are protected for intervals of from five to twelve days against multiple (four to ten) M. L. D. of the Shiga strain of B. dysenteriæ.

In studying the value of the immune serum of the horse, it was found that the bacillus of dysentery resists bacteriolysis to a greater extent than some other members of the colon typhoid group of bacilli. The protective value of the immune serum is clearly established, but the amounts (6 c.c. for guinea-pigs) indicate that it is a low-grade serum.

In conclusion the writer says: "A useful serum therapy of bacillary dysentery in summer diarrhea of infants is rendered highly promising."

Shiga Bacillus. Hiss and Russel[1] describe a bacillus from a fatal case of diarrhea in a child. The following method of isolation was used:

(1) The Medical News, Feb. 14, 1903.

An emulsion of material scraped from the intestinal wall was made in broth. This was grown in Hiss's medium. No threads are formed by the Shiga bacillus in this medium, and the colonies can be distinguished from colon colonies in that they are smaller and lighter in color. When such colonies appear, they are transferred for further study. As the characters of these organisms are so important to practical workers, the description is here given in extenso:

"*Morphology.* The organism is a short, fairly stout bacillus, with rounded ends, in some cultures almost coccoid in form. It occurs singly, but often in pairs, and rarely short threads may be seen. There is a tendency in some preparations to appear in clumps. The bacillus stains with the anilin dyes, but, as is the case with Shiga's bacillus, the staining of all the bacilli in a preparation is not equally intense. Near well stained individuals it is the rule to find many which have taken the dye very poorly. The staining of the protoplasm of individual bacilli is also at times irregular. The organism is decolorized by Gram's method. Spore formation has not been observed. The bacillus is not motile. Flagella stainings have not, so far, been made. No capsules have been noted.

"In general morphology and staining reactions the organism answers most closely to that of the dysentery bacillus as represented by Kruse's culture. Flexner's bacillus is not so stout and is longer than ours, thus corresponding morphologically more closely to the bacillus of typhoid fever.

"*Cultural Characters:* The bacillus grows well on the usual culture media, and corresponds on these in all essential respects to the Shiga bacillus. Gelatin is not fluidified; nor is indol, according to our tests, produced. On the usual nutrient broth, which is clouded uniformly by the growth, a small pellicle may be formed after ten or twelve days at 37° C. When a fermentable sugar is present in the broth, no pellicle is formed. The same

observation holds good for typhoid and colon bacilli. This absence of pellicle is probably accounted for by the ease of obtaining oxygen from the sugar and subsequent inhibition of growth by acid production. A failure to recognize this fact has undoubtedly led to discrepancies in descriptions of organisms. Some facultative organisms, usually not forming pellicles, will do so in sugar-free media, or at all events grow more abundantly in the upper portions where the necessary oxygen is present. None of our cultures of true Bacillus dysenteriæ have, however, formed pellicles.

"*Fermentation Tests:* A study of the organism in dextrose, lactose and saccharose bouillon in fermentation tubes shows a complete correspondence with the bacillus of Shiga, as represented by the cultures 'Kruse,' 'New Haven' and 'Flexner.' All of these organisms ferment dextrose with the formation of acid and a clouding of the open and closed branch of the fermentation tube. Neither lactose nor saccharose are fermented by any of these organisms, hence are not available for anaerobic growth, and the closed branch of the tube remains clear. These bacilli are not able to grow anaerobically in broth not containing some available carbohydrate or allied source of oxygen supply.

"*Pathogenicity.* Our bacillus is pathogenic for both rabbits and guinea-pigs, but not markedly so. No characteristic lesions have so far been noted. Some hyperemia of the small intestine is apparent. Rabbits may be readily accustomed to large doses of living cultures without showing a marked loss in weight.

"*The etiologic* bearing of this group of organisms is becoming more apparent, and as they often show pathogenic properties more acutely active than either colon or typhoid bacilli, they must receive just attention as being dangerous parasites. In this case, as in others, the pathogenesis of the cultures for laboratory animals was a marked feature of their activity."

Distribution. Observations as to the distribution of

pathogenic bacteria are full of interest. Bergey[1] has examined a number of cultures obtained from water with the view of determining their relations to dysentery bacilli. The relationship was tested by studying the serum reactions of such cultures with specific immune sera of such cases or experiment animals. The conclusions reached warrant the following: The agglutination reaction with dysentery immune serum cannot be relied upon to differentiate the members of the dysentery group unless the limits of the reaction are known. The normal sera of horses, rabbits and dogs contain agglutinins in small amounts for several animals. The immunization of an animal against a particular organism increases the agglutinin for that species, but also for other especially allied species. Absorption experiments show that the agglutinins for the specific organism are not alone absorbed, but also those for other species to some extent. The organisms from water studied in this series are shown not to be true dysentery bacilli, but probably related organisms, pseudo-dysentery (Lentz, Kruse). As yet no evidence shows that B. dysenteriæ is ordinarily present in waters.

Additional reports are being presented which add to the evidence of Duval and Bassett relative to specific organisms as a cause for infantile diarrhea. Martha Wollstein[2] presents the results of examination of such cases as were presented to the New York Foundling Hospital. In all, 114 cases were studied during a winter and a summer period. The method was that recommended by Flexner. The material was suspended in broth, and acid agar plates were prepared from the suspension. These remained in the incubator 24 hours. Glucose agar tubes were now inoculated from the colonies, and in another 24 hours the gas-producing cultures were rejected. The selected cultures were now tested as to reaction with serum from a horse that was immune to the Flexner Manila culture.

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(1) Jour. of Med. Research, August, 1903.
(2) Jour. of Med. Research, August, 1903.

In no instance was the dysentery bacillus isolated from the stools of healthy infants; ten of these being examined at periods of from 12 hours to 8 days. On the other hand, every case in which blood and mucus was present showed the dysentery bacillus. In many negative cases there were undigested food and mucus, but no blood. It seems then that this infection leads to the classical clinical picture of dysentery. Thirty-nine cases showed the dysentery bacillus. The ages of these children varied from 2 months up to 3 years. Twenty-seven were under 1 year old. Of the positive cases 29 died. The bacillus is a short non-motile rod with round ends, almost coccoid and either single or in pairs. It is negative to Gram. It does not liquefy gelatin or coagulate milk. Fermentation of sugars is absent. The colonies on plate are markedly pearly white. They are finely granular and the edges become irregular after 24 hours. Broth is evenly clouded, and in two of the cultures a thin pellicle formed on the surface; in all the others this was absent. Indol is formed slowly only after 3 to 7 days. Acid is formed in the presence of mannite and maltose. The serum reactions were positive for the identification of this organism.

As regards serum reactions of the blood of the little patients it was found that this was very irregular and sometimes absent. This has been noted by other observers. The bacilli were most numerous in the stools that contained blood and mucus. Five cases came to autopsy, and in none of these was bacilli found in the blood or in any viscus. Several cases are described in which the infection developed in the hospital, but no deductions as to the manner of transfer are given. Cases of dysentery were present in the wards at the same time.

Influenza. Wynekoop[1] describes the usual characters known for this bacillus, and calls special attention to the cultivation from the throat on blood serum. In conclusion he states that at no time since the winter of 1898-1899, when examinations were undertaken by the Health De-

(1) Chicago Medical Recorder, May, 1903.

partment, has Chicago been free from this organism. The throat manifestations of 173 of the 2,460 cases from which cultures were examined since this time, were so severe that a probable diagnosis of diphtheria was made, yet the diphtheria bacillus was not found, while influenza bacilli were present in nearly pure culture.

Serum therapy in influenza has been the hope of the laboratory worker as well as the clinician, but, after reviewing all the effort that has been put on this organism, it is seen that little has been accomplished. Quite recently Cantani, Jr.,[1] reports a long series of observations in experimental influenza immunity. In general the results of experiments reported show that animals did not develop increased resistance through influenza injections or inoculations. As a result of the present study, the author reaches the conclusion that artificial immunity against influenza can be established.

Observations as to the pathogenesis of the influenza bacillus have shown great irregularity, and, from the hands of some observers, negative results. As a general proposition, there is very little growth of the bacteria in the bodies of animals, and the lethal effects are almost entirely toxic in nature. When inoculated into the brains of animals, multiplication of the bacilli takes place and the virulence is increased. Intra-peritoneal inoculations in young guinea-pigs cause death, and in some of these the autopsy shows a peritonitis with hemorrhagic exudate. Among the many cultures studied by the author, only one was found that caused death when subcutaneously inoculated. As regards resistance, it was found that guinea-pigs withstood subcutaneous injection, while one-tenth loop from an agar culture would cause death when implanted intraperitoneally. Dogs resist influenza infection when applied subcutaneously, or to the peritoneum; in the brain, however, it was found that growth took place.

The most suitable culture medium for the influenza bacillus was found to be the blood-agar mixture of Voges.[2]

(1) Zeits. f. Hygiene, Bd. 42, Hft. 3, 1903.
(2) Deutsch. med. Wochens., 1894.

In order to maintain virulence, the cultures must be passed rapidly through animals. The animals used for the immunization experiments were prepared by injecting them subcutaneously with cultures that had been heated to 56° C. for one-half hour. No injections into the peritoneum were made because this was reserved for the application of the virulent culture used to test the degree of immunity that had been produced. The repeated subcutaneous injections cause great depression in the animals, and many die after a time greatly emaciated, but without evidence of active pathologic changes. The injection of a single subcutaneous dose of dead culture causes resistance to the intra-peritoneal test culture to the extent that the animals lived 48 hours in the former case, while the control animals died in 20 hours. Better results were obtained when the injections were repeated. Several subcutaneous injections were made, and then the test with the virulent culture. Of 24 experiments 11 were satisfactory in that the animals were not killed by the supposedly fatal dose. Sterilized peritoneal exudate and influenza emulsion in brain extract were tried with similar results.

In attempting to demonstrate the duration of such artificial immunity, it was found that it was of very short duration. This would seem to correspond with observed facts in the human subject.

The testing of the serum of these animals was done by mixing the serum in question (I, from healthy animals; II, from those having had one subcutaneous dose of influenza, and III, highly immune animals) with what would be a probably minimal fatal dose of culture, and then injection of the mixture intra-peritoneally into young guinea-pigs. The immunized guinea-pigs were also capable of destroying the injected bacilli, thus showing a Pfeiffer's phenomenon. As regards serum reaction and the protective value of immune serum in influenza, the results show that they are to be taken with caution. At times normal blood has considerable agglutinating power, while

the positive agglutination of a culture with specific serum can never be carried to great dilution.

It cannot be said that any considerable artificial immunity was produced in these experiments. The method of Cantani is worthy of further application in experiments along these lines.

Leptothrix Infections in Animals. Pearce[1] considers the subject of leptothrix infections in animals. He was led to the investigation through the spontaneous infection and death of six rabbits with a leptothricial organism. The lesions in the six animals were much diversified, but showed the presence of filamentous organisms, which from the microscopic study were probably identical. The only cultivations that were successful were made on portions of sterile organs of rabbits. The organs for this purpose were removed under aseptic precautions and placed in sterile Petrie dishes, then were directly inoculated from the diseased tissue. The growths were always slight and often entirely unsuccessful. Inoculation experiments in rabbits were satisfactory, and this was the means used to keep the cultures alive. Guinea-pigs were immune. Intra-pleural inoculations were made. The study of the morphology of the organism showed that it did not differ from the descriptions of Schmorl, Flexner and Loeffler, but the author could not find in his specimens any evidence of branching cells. This organism is described as a streptothrix. The descriptions of organisms similar in origin, would seem to show that they are identical. The chief difficulty is in the uncertain experiments, in that the different observers report no uniform results. As regards the name of the organism, it is imperative to drop the name streptothrix because of the inability to find branching cells. Chester classifies it in this group. Owing to priority, the name *Leptothrix necrophorus* becomes imperative.

Pneumonia. Rosenow[2] made cultures in 83 cases of lobar pneumonia, with positive results in 77 and cultures

(1) Univ. of Penn. Medical Bulletin, November, 1902.
(2) Medicine, June, 1903.

from 74. In most cases, only one blood culture was made. The full conclusions as regards pneumonia are presented:

With improved technic, using for inoculation large quantities of blood, the pneumococcus can be recovered in all cases of pneumonia, and in obscure cases of pneumococcus infection blood cultures may be a diagnostic method of positive value.

Pneucoccemia in pneumonia does not mean a specially unfavorable prognosis.

On account of the difficulties involved, agglutination of the pneumococcus is, at present, of scientific rather than of practical value.

Normal and pneumonic blood and serum appear to have no bactericidal influence on the pneumococcus.

The interesting question whether lobar pneumonia is the primary result of a direct local infection of the lung or of a secondary localization in the lung of pneumococci in the blood, is as yet hardly ripe for final discussion.

Scarlet Fever. Class[1] in his paper writes as follows:

"Before closing I wish to state that I am more firmly convinced than ever that this germ is the causative factor of scarlet fever, even though the results obtained by certain investigators have apparently been negative. One class of critics has stated that there is no such germ to be found in scarlet fever as the one I have described, while others, equally competent, have stated that this germ is found not only in scarlet fever patients, but that it has a very wide distribution, being present in all healthy throats and in numerous other places. It is therefore clear that one or the other set of critics is mistaken, and, possibly, both."

This is probably the true status of the question. More evidence is required to make the causal relation positive, while nothing short of a demonstration of the real cause will clear the view.

Weaver[2] describes his methods, which consisted in mak-

(1) Medicine, July, 1903.
(2) American Medicine, April 18, 1903.

ing cultures by the plate method on glucose-agar, glycerine-agar and earth-agar, from the surface of the tonsils and from the skin of the patient before and during desquamation. The resulting colonies were transferred and studied as pure cultures. Streptococci were found abundantly present on the tonsils, and Staphylococcus pyogenes albus fairly abundant on the skin. In closing the paper Weaver says:

"Observers who have made extensive studies of the bacteria found in the mouth have demonstrated a large number of cocci, including the pyogenic cocci in this location even in health, and the results here related differ in no way from those previously obtained. In the throat large numbers of bacteria may easily gain lodgment from the air and food and probably most of those observed have such an origin. In a few instances cultures from the skin could be recognized as identical with those of the throat.

"These observations have led to the following conclusions:

"The bacteria obtained from cultures from the skin, epidermic scales and the surface of the tonsil in cases of scarlatina are the same as those found in the same locations in health, and no one of them is constantly present except the streptococcus in the throat.

"Because of the numerous cocci which grow in such cultures, and which appear in groups of two and four or bunches of the same under the microscope, it is impossible to identify them, except by a complete study in pure culture.

"Cultures made by inexperienced persons or by those who do not fully appreciate the importance of avoiding the tongue, are especially apt to contain large diplococci or sarcinæ.

"The streptococcus is present upon the tonsil of scarlatinal patients in enormous numbers in almost all cases."

Syphilis. This disease continues to present an interesting field for research as to its etiology. Several articles have appeared on the subject. The blood of syphilitics has

been studied while the seminal fluid has been investigated with the same view. Joseph and Piorkowski[1] report their finds in examinations of the semen in cases of syphilis during the infective period. The writers were led to this line of study because of the well-known fact that a syphilitic man may infect the fetus without infecting the mother when there are no direct evidences of active syphilis. The first line of procedure was to obtain sterile placentæ. This was done by catching the placenta as passed in a large sterile petri dish. From the maternal side small pieces were cut and transferred to ordinary petri dishes. These were now smeared with freshly discharged semen and placed in the incubator. A control was established by making careful microscopic and cultural studies of the placenta and the semen. In 24 or 48 hours there appeared on the prepared pieces of placenta small colonies in those cases where syphilitic semen had been used. Two bacteria were found in these. Staphylococci were present in a small number of colonies, while in others which had the appearance of dewdrops with a greenish cast a small bacillus was found. They were about the size of subtilis and stained fairly well by ordinary methods and were positive to Gram. It was possible to transfer the organism to blood agar or urine agar and the resulting growth appeared as a thin film which could be pulled from the entire surface of the medium in one piece. Transfers to other media were negative and the organism quickly lost vitality. When such cultures were again transferred to blood agar a good growth reappeared. The bacillus was found in 22 instances. The examination of the normal semen, as also that from old cases of syphilis, were entirely negative as to the presence of this bacillus. It is only present in the semen during the active infectious period.

The writers state that they were able to stain the bacillus in the semen of syphilitics, and present several pictures to prove this point. The staining method consists in the usual spreading and fixing of the material. It is then

(1) Deutsch med. Wochens., Nos. 50-51, 1902.

stained with very dilute carbol-fuchsin stain, 20 drops of the stain to 30 c. c. of distilled water. The stain remains upon the specimen for 10-15 minutes. Tissue sections can be stained in the same way.

Streptococcus Differentiation. Schottmüller[1] reports the results of his observations on streptococci and the value of culture media as a means of differentiation. The difference in culture reaction in fluid media, especially bouillon, has long been noted. The writer uses blood agar as a solid medium and preferably that containing human blood. He would divide streptococci into two groups of which the representatives are Streptococcus longus (erysipelas) and Streptococcus mitior (brevis).

The second group is found in mild or secondary infections such as rhinitis, bronchitis, lung abscess, empyema and in association with other bacteria in various infections within the abdominal cavity. The results of growth on human blood agar show that the Streptococcus mitior (brevis) produces colonies that have a green color, while those of the Streptococcus longus never show this peculiarity.

The author's experiments as to pathogenesis would also seem to show a difference, as the virulent streptococci do not form pigment.

Aronson[2] reports further studies on streptococci and especially as regards streptococcus serum in scarlet fever. He previously reported that the serum from a horse immunized with the streptococci from cases of scarlet fever strains, namely, from erysipelas, diphtheria, sepsis, angina was active against infection with other streptococci and rheumatism. In like manner, the serum of a horse immunized with streptococci from a case of sepsis, was protective against the other strains. Moser[3] comes to the conclusion that scarlet fever streptococci are different from the strains cultivated from erysipelas and abscesses, because of certain agglutination phenomena that he was able

(1) Muench. med. Wochens., No. 20, 1903.
(2) Deutsche med. Wochens., No. 25, 1903.
(3) Berlin Karger, 1903. S. 16.

to demonstrate. Others have reported similar results, which would seem to show from the agglutination test, that strains can be differentiated. As material for the present experiments 27 strains of streptococci were used, 11 from scarlet fever, 3 from the throats of cases of rheumatism, 4 from the blood of puerperal sepsis, 2 from pus and 3 from cases of local infection, and, lastly, 4 from cases of strangles in horses. These cultures were carried on by transfers into tubes of 1 per cent glucose bouillon. Most of the strains cause no diffuse clouding, but a flocculent precipitation. In those that were cloudy the chains were found to be shorter and often diplococci were present. From even a close study of the general characters of the strains of streptococci under study no evident difference as regards species could be determined.

Horses were immunized as follows:

1. Seven varieties of scarlet fever.
2. Scarlet fever strain No. 8.
3. Puerperal sepsis, No. 1.
4. Puerperal sepsis, No. 2.
5. Articular rheumatism, No. 1.
6. Articular rheumatism, No. 2.
7. Otitis streptococcus.
8. Druse strain.
9. Scarlet fever—mouse, virulent culture.

The blood of these horses was used for immunization. From the records of the individual tests made on the cultures and with these various sera, it appears certain that there is no group differentiation, although the serum always reacted best with the specific strain used in the immunization. As Aronson had a number of specific antistreptococci sera, these tests should be conclusive. He found in one instance that an antistreptococcus serum prepared with a culture from sepsis showed agglutinating power for a scarlet fever streptococcus, much beyond that for the sepsis species.

The protective value of these various sera was also tried and it was found that no definite results as to the differentiation of species could be determined. Reviewing his study the writer says that, as it is impossible to identify strains of streptococci by either culture methods, microscopic study, animal experiments, agglutination results or serum protection, it would further appear that a particular culture cannot be held accountable for any particular kind of streptococcus infection nor does it seem at all probable that such a definitely characterized infectious disease as scarlet fever can be due to streptococci. It would also appear that there can be no specific streptococcus scarlet fever serum. As a given serum will not act upon different strains to the same degree, the writer raises the question as to immunizing horses with as many strains as possible, or the mixing of sera from different horses.

Piorkowski[1] reaches a similar conclusion from his observations. Although not extensive, they are worthy of notice. The author has prepared an antistreptococcus serum against a streptococcus infection of the air passages of horses. He finds that the serum agglutinates the cultures obtained from the cases, but not other varieties. He thinks that success in this direction must be a very specific serum for the species in the case, and does not think that all streptococci are of the same variety.

The Protective Value of Streptococcus Sera. A series of experiments is reported by Summerfield[2] in which he shows that the antistreptococcus serum of Aronson has great protective power for mice inoculated with streptococci, as compared with the sera from Institute Pasteur, Roux, Moser, Paltauf and Tavel. The method used was to inject intra-peritoneally a varying amount of the serum, thus making a series of prepared animals. Into all of these the same amount of a 24-hour virulent bouillon culture of streptococci was injected. This occurred twenty-four hours later and was also intra-peritoneal. The re-

(1) Berliner klin. Wochens., No. 48, 1902, p. 1125.
(2) Centralb. f. Bakt. I. abt. Orig. Bd. xxviii, No. 9.

sults showed that the Aronson serum regularly saved the lives of the animals when applied in amounts as small as .0002 c. c. against a culture of streptococci from bone marrow of a case of scarlet fever, the dose being one-millionth cubic centimeter, or ten minimal fatal doses. Cultures of streptococci from other sources were used and failed to kill the animals, although they were not so markedly specific as the scarlet fever cultures.

The serum of Moser-Paltauf was shown to possess a protective value to a much less degree; that of Roux and Tavel, however, was shown to be worthless in these experiments—the serum-injected animals died as soon as the control mice. It would seem that there must be some wide difference between the serum of Aronson and that of other producers.

Streptococci Agglutination. Meyer[1] experimented with various antistreptococci sera—Marmorek, Tavel, Aronson, Meyer—to determine the agglutination properties as regards various strains of streptococci. He found that the immune serum would promptly agglutinate the organism that had been used in the treatment of the animal, but not other strains unless passed through the same species. It was further determined that a strain of streptococci that was not agglutinated by a particular serum, would be promptly acted upon if it were passed through this animal. It would then seem that the passage of an organism through animals produces receptors that occasion the agglutination.

On this basis the author attempts to explain therapeutic failures, and proposes that for human therapy, only cultures from a human source should be used.

Meyer[2] reviews the work of others and from experiments concludes that human streptococcus cultures must be differentiated from those from lower animals.

Immunizing experiments. Growth in media in which other streptococci are grown and the pathogenesis are the

(1) Deutsche med. Wochens., No. 42, 1903, p. 751.
(2) Berliner klin. Wochens., No. 4, 1902.

differential points. Marmorek has taken his position because he was able to get different strains of culture equally virulent by cultivation and passage through animals. This view is strengthened by the fact that cultures of streptococci will not grow in media in which other streptococci have previously been cultivated. The writer comes to the conclusion that streptococci of the pus producing variety are to be differentiated from those attained from the throats in anginas and those from cases of scarlet fever.

Tetanus Occurrence. A case of tetanus following injury to the nose was presented to Holbeck.[1] About five hours before the death of the patient a quantity of blood was withdrawn from the veins of the forearm. All usual microscopic examination was negative. Cultivation in bouillon showed, after some days, a slight growth which upon examination was found to be a mixture—a bacillus resembling B. tetani and a few cocci. Further cultivation was accomplished under careful anaerobic methods. Inoculation of animals caused death, but convulsions were not distinct. In the blood of these dead animals it was conclusively proved that the bacillus was not present. This is one of the few cases where this bacillus has been demonstrated as invading the blood and shows the possibility of finding tetanus in internal organs where it has been carried by the blood.

Tuberculosis, Strains and Virulence. De Schweinitz, Dorset and Schroeder[2] review their investigations on tuberculosis from which they conclude that there is evidence to show that tubercle bacilli from man can cause tuberculosis in cattle and that the surroundings influence their virulence and morphology. The inoculation of bacilli from birds, cows, dogs, carp and from man into glycerin bouillon showed at the end of a few weeks to three months marked morphologic differences. It was also noted that the more virulent cultures showed less change in appearance than the others. The chemistry of the bacilli from

(1) Deutsche med. Wochens., No. 10, 1903.
(2) Tuberculosis Congress, Berlin, 1902.

human and bovine source indicates a close relation between them. Tuberculin made from different cultures shows that it has the same effect in producing reaction in man and in animals. The inoculation of monkeys shows that the bovine bacillus is more virulent, but in both human and bovine inoculations into monkeys, the lesions were widely disseminated. From these monkeys, young heifers were inoculated and it was seen that the bacilli had little virulence for the animals.

The authors go on to report a number of experimental inoculations of cattle with bacilli from the human subject. Cultures are now at hand which are pathogenic for calves.

"That tubercle bacilli obtained from cases of human tuberculosis can produce disease in cattle is to my mind (de Schweinitz), by our own work, not to mention that of many investigators, like Nocard, Ravenel and others, *absolutely proved.*"

Virulence of Bacillus from Different Localities. Kronpecker and Timmerman[1] present an interesting article on the virulence of the tubercle bacillus as isolated from various tubercular lesions in the human subject. A special point is made that in many previous experiments animal inoculation was used in the isolation of the bacilli from the cases. This no doubt adds an element of uncertainty in that the influence of the experiment animal may modify the virulence of the original human bacilli. Material should, therefore, be obtained directly from the cases. For this purpose the authors used the method of glycerin potato culture from cases where there was no mixed infection. Mixed infection always caused overgrowth and failure in the result. As soon as an apparent growth occurred rabbits were inoculated directly from the glycerin potato cultures. The results of their inoculation were recorded, as regards the time of appearance of manifestations, the extent of the disease and the possible individual disposition of the animals. Out of 30

(1) Centralb. f. Bakt., I Abt. Orig. Bd. xxxiii, No. 8.

cases studied, pure cultures were obtained in 26. It was shown that the manner and extent of growth from the original source on the potatoes did not indicate in any way the virulence of the particular bacilli. The individual predisposition of the rabbits constantly caused discrepancies in the experiments. Everything else being equal, this fact frequently caused differences. Cases of surgical tuberculosis contained tubercle bacilli which induced general tuberculosis when injected intravenously in periods ranging from 30 to 40 days. The inoculation experiments with the cultures from cases of surgical tuberculosis would show that these bacilli are equally virulent. Omitting the few instances of individual susceptibility upon the part of the animals, the authors believe that there are special reasons of resistance or predilection that cause the rather uniform disposition of tubercles in cases of miliary tuberculosis. The skin is very seldom involved. The brain rarely. In rabbits we see in general infection the liver rarely involved, while the lungs and kidneys seem specially predisposed to the infection. The presence of early appearing fever is a factor in determining relative virulence.

Points of Entrance. As we have seen the points of entrance of tubercle bacilli into the body has been a much-discussed question, it has repeatedly been argued that the lymph channels are a frequent route by which they reach deeper structures or become generalized. MacFadyen and MacConkey[1] have made a special investigation as to the presence of virulent tubercle bacilli in the mesenteric glands, tonsils and adenoids. It was primarily undertaken to show the presence or absence of such bacilli in young children by an examination of suitable post-mortem material.

The method used was somewhat different from the usual procedure. Inoculations into guinea-pigs were made. The glands upon removal were placed in a mixture of glycerin and normal salt solution and taken to the laboratory. Here each gland was disintegrated separately by means of a

(1) British Med. Jour., July 18, 1903.

special device that reduced the tissues to a pulp so that it could be injected by a hypodermic needle. By this means any bacilli embedded in remote parts of the gland would be exposed and the test approached more nearly an absolute test. One-half of the emulsion was injected subcutaneously, the other half intraperitoneally into guinea-pigs. If the animals did not die they were killed in 6 or 8 weeks. The basis for the result was always made upon microscopic examination of the lesions present in the animals. Twenty-eight cases were examined in this way. Of these 8 showed tuberculosis. All were children under 5 years of age except 2, one of them being 6 and the other 8 years old. There were no intestinal lesions present in any of the cases. The inoculation experiments showed that virulent tubercle bacilli were present in the mesenteric glands in 10 of the 28 cases. Among the 8 cases of tuberculosis 5 gave positive and 3 a negative result. The positive results based on the entire number of cases gives a percentage of nearly 36 per cent. Of the non-tubercular cases 5 showed the bacilli, giving a percentage of 25 for this group.

It would appear, then, from these experiments that tubercle bacilli are present in the mesenteric glands more frequently than ordinary post-mortem examinations would lead one to suppose.

The tonsils from 34 cases and the adenoids from 44 cases were examined after their removal by operation. The results of these experiments were entirely negative. The only virulent organisms found in them belong to the group of the micrococci. In this connection the report of Morf[1] on *primary tuberculosis of the abdominal lymph glands* is interesting. Tubercular glands were sought among 232 consecutive autopsies, the glands about the cecum, root of the mesentery and about the abdominal aorta being specially examined. Eighty-six bodies or 37 per cent were tubercular. Of the 86 tubercular bodies 9 showed involvement of the abdominal lymphatics. Of the non-tubercular cases (other than abdominal), only 2

(1) Trans. Chicago Path. Soc., March 9, 1903.

showed macroscopic evidence of tuberculosis in these structures. In 26 cases microscopic examinations were made and in no instance were bacilli found where tuberculosis was not evident to the naked eye.

Serum diagnosis. The work of Arloing, Courmont, Koch and others have given rather irregular results in serum diagnosis of tuberculosis. Loeb[1] briefly reviews these experiments and reports his own experiments. The method of diagnosis if exact would be of the greatest value. The requirements proposed are a homogeneous culture and the serum, the test being conducted like that for typhoid fever. The cultures are prepared by growing the bacilli on potato moistened with 6 per cent glycerin water. After 3 or 4 weeks' growth the colonies are broken up with a glass rod and transferred to veal broth having 1 per cent peptone and 6 per cent glycerin. These tubes are frequently shaken and are turbid after 8 to 12 days. They are now ready for use. The reaction is conducted by adding the clear serum from the suspected case to such tubes of tubercle bacilli in proportions of 1 to 5, 10, etc. After 4 to 6 hours the reaction should be distinct. If it is positive the mixture will have become clear and a flocculent precipitate will be in the bottom of the tube. The author's experience included 52 cases. The following data were obtained:

Cases in which blood sera were used; *a,* non-tuberculous 2+(15 per cent), 12—(85 per cent); *b,* tuberculous 2+ (100 per cent) 0—

Cases in which serous effusions were used; *a,* non-tuberculous 6+(30 per cent) 14—(17 per cent); *b,* tuberculous 12+(73 per cent) and 4—(27 per cent).

(+ = positive results, — = negative.)

It has been stated by Arloing that the reaction is due to the formation of antibodies and in irregular cases it is not formed in sufficient amount. The work of others rather disproves the formation of any body resembling antitoxin in tuberculosis. The writer concludes that under

(1) Trans. Chicago Path. Soc., Oct. 13, 1902.

various conditions animal sera will agglutinate cultures of human tubercle bacilli.

Such sera may be obtained from the human or animal body even when it is free from tuberculosis. It is doubtful whether the agglutinative powers are ever due to the specific action of B. tuberculosis. The presence or absence in adult human blood of the agglutinating property is not evidence as regards tuberculosis.

Typhoid Occurrence. Kerr and Harris[1] examined 56 cases of typhoid by *blood cultures,* and found the bacillus present in 31 cases and absent in 25—or positive in 57 per cent. As to the time in the disease when the bacilli were found, 9 in first week, 7 positive and 2 negative; 20 in second week, 10 positive, 10 negative; 16 in third week, 11 were positive, 5 negative; 8 in fourth week, 2 cases were positive and after this only one case, a relapse, showed bacilli present. As to the numbers of bacilli present, the authors say:

"The number of bacilli in the blood is probably not large; only 8 out of 31 positive cases showed the bacillus in all of the flasks inoculated."

The usual method for blood cultures was used and the distribution to the flasks was made in amounts of 1 c. c. The conclusions reached are, that the bacilli are present in the blood early in the sickness, where they can be found if a large amount of blood is examined and that it is an important part in the diagnosis of typhoid fever.

Gehrmann[2] cultivated the *typhoid bacillus from the urine* of 15 cases out of 71 cases examined. Only one specimen was taken from each case. In 12 specimens B. coli was present; in 6, pus cocci; in 31, saprophytes, and 20 were sterile. Colon and typhoid bacilli were found mixed in 3 cases. As regards clouding of the urine and the presence of typhoid, it was found here that 9 cases were cloudy of the 15 that were positive for typhoid.

Typhoid and Paratyphoid. In the course of a bacterio-

(1) Chicago Medical Recorder, Oct., 1902.
Chicago Medical Recorder, Oct., 1902.

logic study of the blood in thirty cases of clinical typhoid, Ruediger[1] found two cases which proved to be paratyphoid. Thirty patients were examined and in twenty positive blood cultures, the bacillus was found nineteen times in the first culture that was made. In this first negative case a positive culture was obtained in the second examination which was made seventeen days after the first.

The following is a statement of the results of growths of the cultures obtained from the two cases of paracolon infection, together with the serum reactions as obtained in these cases. As no difference was noted between the organisms isolated from these cases (10 and 15) one description will suffice for both.

The organism resembles the typhoid bacillus; produces no spores; stains like the typhoid bacillus, decolorizing by Gram's method. The organism is markedly motile in bouillon cultures at 37° C., and retains its motility for several days. Eight to twelve flagellæ are present in peritrichal fashion. In gelatin and agar the cultures resemble those of typhoid bacillus. It produces gas in glucose agar stab cultures. Gelatin is not liquefied. Agar plates emit an odor resembling that of the colon bacillus. Macroscopically and microscopically the colonies resemble those of the typhoid bacillus.

Bouillon—Diffuse cloudiness in twenty-four hours. There is no scum formed after seven days. The cultures emit an odor resembling cultures of B. coli.

Agar Slant—White streak along the line of inoculation resembling a growth of B. typhosus.

Glucose-Agar Stab—White growth on the surface and along the stab. Numerous gas bubbles are produced in the media.

Litmus Milk—Distinct acid reaction after twenty-four hours. After a few days is again normal, and is markedly alkaline after fourteen days.

Potato—A slight, white shining growth resembling B. typhosus.

(1) Medicine, April, 1903.

Gelatin—White growth on the surface and along the stab. No liquefaction.

Indol Reaction—Absent in sugar-free broth tested after seven days.

Fermentation of Carbohydrates—Glucose, maltose, levulose and mannite are split up with the production of acid and visible gas. Saccharose and lactose are not affected.

Serum Reaction—On August 16, 22, 28 and September 10 the serum from case 10 did not agglutinate typhoid bacilli at a dilution of 1 to 5. In case 15 typhoid bacilli were not agglutinated by the serum of the patient at a dilution of 1 to 5 on August 27, September 10 and 19.

The bacillus isolated from the blood of case 10 was not agglutinated at a dilution of 1 to 5 by immune goat's serum known to agglutinate B. typhosus at high dilution. By the serum from this patient and from case 15 it was agglutinated at a dilution of 1 to 1,000, but the limit of dilution at which serum would agglutinate was not determined.

The bacillus isolated from the blood of case 15 was not agglutinated at a dilution of 1 to 5 by immune goat's serum. The serum from the patient agglutinated this organism at a dilution of 1 to 2,000 and the serum from case 10 at a dilution of 1 to 1,000.

In comparing the characteristics of the organisms isolated from these two cases, it becomes evident that morphologically and culturally they appear to be identical with those isolated by Gwyn, Schottmüller, Cushing, Libman, Hewlett, Johnston, Longcope, Herbert W. Allen and others from cases resembling typhoid fever clinically.

Agglutination: Typhoid, Coli and Paratyphoid. Bruns and Kayser[1] report a study of the agglutinating effects of specific sera upon cultures belonging to this interesting group. Summarizing their observations, we find that a high grade immune serum will agglutinate the related bacteria besides those for which it is specific. The microscopic results are more exact, and show the reaction in

(1) Zeits. f. Hygiene, Bd. 43, Heft 3, 1903.

greater dilution than the macroscopic. The rapid occurrence of the reaction is the best means for clinical differentiation. The variation of typhoid and paratyphoid agglutination are separated by a maximum dilution of about 20 times. It is possible that both are present, and in that case the patient's blood shows a dual reaction. Positive agglutinations are more marked for the organisms standing closest in group relations. It would appear that some of the described paratyphoid cultures are the same as B. typhi abdominalis. Their agglutination is not materially different. In such a large group there may even be variations as regards agglutination. The writers prefer a medium strong serum for ordinary tests. They find that the rapidity of the occurrence of the reaction should be taken into account, and find that if in dilution 1:100, a prompt macroscopic reaction appears, it is a differentiating point. The microscopic test in greater dilution is the procedure recommended for the differentiation of various species. It is thought important that the allied species should be tried on the patient's blood in all cases of doubtful typhoid.

An Epidemic Due to Bacilli Causing Condition Resembling Typhoid. Contiadi and v. Drigalski and Juergens[1] studied an epidemic in which 90 soldiers having symptoms resembling typhoid were examined bacteriologically. In 41 cases the bacillus was isolated from the stools. Some of the cases gave positive serum reactions with typhoid only; in others, there was positive reaction with the isolated bacilli only, and in others it was positive with both. The bacillus isolated in these cases gives the following data for identification: (1) Bluish colonies showing motile bacilli, which agglutinate in immune serum; (2) fermentation and gas production in grape sugar media; (3) thick, whitish, slimy growth on slant gelatin; (4) Petruschky's whey acid production without clouding; (5) growth in milk without curd; (6) abundant growth showing an alkaline reaction on litmus-lactose agar.

(1) Zeits. f. Hygiene, Bd. 42, Hft. 1, 1903.

Serum reactions of this bacillus, when studied in comparison with the typhoid bacillus, showed that each reacted positively with its own specific serum, to a dilution of 1:4000 to 1:5000, but with the opposite serum only in dilutions of 1:500 or 1:1000. This was true for the immune serum of prepared animals, as well as for the sera from patients.

IMMUNITY AND SERUM REACTION.

Graphic Demonstration. Prudden,[1] in an article on recent researches in cytolysis and immunity, reviews the subject in a most able manner. In describing certain points, he uses a graphic method which at once clears this rather difficult subject. The poisonous element of toxin is represented thus:

"This conception may be illustrated as in Fig. 19, in which the toxophorous group *a* of the toxin molecule can be effective in damaging the cell only when united to the latter by the haptophorous group *b*."

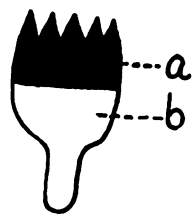

Fig. 19.

The production of antitoxin is illustrated very nicely by the increase and liberation of receptors, as shown in the diagram (Fig. 20).

"The toxic molecules *a*, uniting with the receptors *b*, lead through the injury to the cell, as well as by its deprivation of the normal use of *b*, to the production and at

(1) Medical Record, Feb. 14, 1903.

length to the overproduction of new receptors of the same kind. These superfluous receptors *c* are now cast off into the body fluids, where, as at *x*, they may freely unite with the floating toxin molecules, forming harmless compounds and preventing further access to the cell, where alone the damage can be done. Or when free, as at *y*, the receptors may be transferred in the serum, becoming effective as antitoxin in another individual."

The phenomena of cytolysis, or cell destruction, and its prevention by anticytolysins, is illustrated as follows:

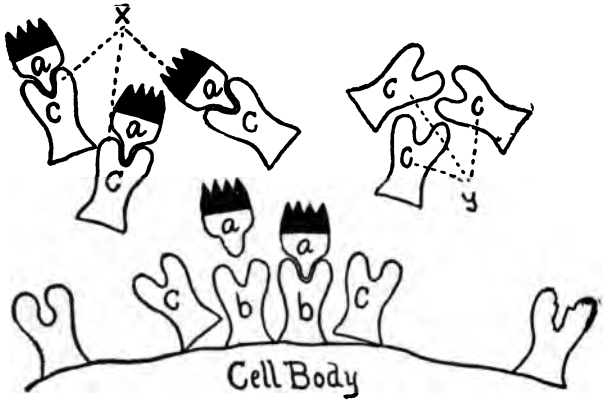

Fig. 20.

"Let *a* (Fig. 21, A) be the cell which is to be destroyed with one of its receptors indicated at *d*. Let *b* represent the immune body with one atom complex *e* capable of uniting with the cell receptor *d*, and with another *f* capable of uniting with the alexin *c* through *g*. Now the alexin which appears to be the effective agent in the destruction, cannot unite directly with the cell receptor. When, however, it becomes linked to the cell by means of the immune body *b*, its destructive capacity can come into play.

"In similar fashion one may indicate the action of anti-

cytolytic substances which may be effective through union, either with the alexin or with the immune body, as shown in Fig. 21, B and C. In B the 'antibody' *h* prevents the linking of the alexin *c* to the immune body *b* by itself uniting with the former. It then acts as an antialexin. In C the 'antibody' *i* prevents the linking of the immune body *b* to the cell receptor *d*, and hence acts as an anti-immune body."

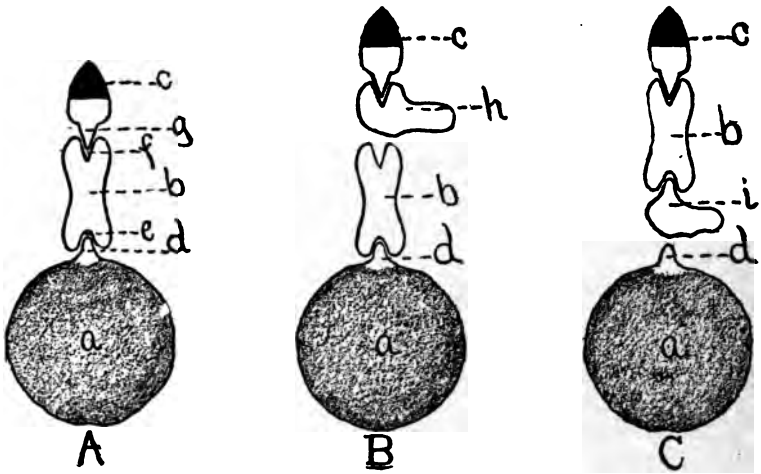

Fig. 21.

Bacterial Virulence. Walker[1] passes typhoid cultures through immune serum and finds that the virulence and activity of cultures can be greatly augmented. His method consists in making an emulsion of the culture in the serum and then plating from the emulsion; sub-cultures are started and the passage is repeated. He reports 26 passages in this manner of a typhoid culture and found that its virulence was doubled and the number of individual bacteria that resisted bacteriolysis was increased from 1,800 that grew before passage, to 19,440 in the plate for the twenty-third passage. The minimal lethal dose was

(1) British Medical Journal, Oct. 18, 1902.

found to be .2 c.c. at the time of the first passage and .1 c.c. after 27 passages as described.

Structure and Chemistry of Bacterial Cells; the Extraction of Cell Toxins. E. Levy and F. Pfersdorff.[1] Agar plate streaks are made. From these the culture is scratched and mixed with some water. An emulsion with toluol is made and placed in an air-tight receiver. This is placed in the incubator and shaken for a period of three months. The bacteria are killed, the ferments remain. A condition of bacterio-autolysis takes place.

Anthrax yielded a lab ferment, a gelatin liquefying ferment, a fat-splitting ferment and a body poisonous for mice.

Neisser and Shiga[2] by extracting B. typhosus and B. dysenteriæ with normal salt solution one hour, at 60 C., two days at 37° C. and filtering, found a fluid that contained free receptors for these bacilli. Injected into animals, it induced agglutination properties reacting in typhoid, 1:20,000 in three injections; intravenous, not poisonous to animals. It also added the agglutinating power to inactive serum. Proagglutinoids are formed.

The extract from typhoid bacilli causes in rabbits, depression and death or collapse, with congestion of intestines and evidence of diarrhea. The serum prepared from animals treated in this way had a strong bactericidal influence, killing typhoid in three hours.

Serum Reaction. Landsteiner[3] concludes that the agglutinating substance can be recovered from the agglutinated cells. Such solutions have properties specifically different from the serum from which they come. Normal serum contains agglutinins that are not specific. It is still doubtful whether the immune bodies in serum are produced from preformed bodies or are the result of combinations of bodies already existent on the side of the host and the parasite.

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(1) Deutsche med. Wochens., No. 49, 1902, p. 879.
(2) Deutsche med. Wochens., No. 4, 1903.
(3) Muench. med. Wochens., No. 46, 1902.

Diphtheria. Lipstein[1] finds by injecting dead and later living cultures of B. diphtheriæ with simultaneous subcutaneous injection of large doses of diphtheria antitoxin that a high immunity is developed. Before treatment the animals were negative as to agglutination; after 4 injections a positive agglutination in 1280 dilution was attained.

The Susceptibility of the Organs and Tissues for Bacteria and for Toxins. Kaus[2] brings forward many examples that show a resistance or failure to injure certain animals or organs by bacterial action, where toxins cause disturbance; and again others where the toxin is the disturbing element, there is a predilection for poisons for certain organs. He concludes that the entire action is dependent upon the presence of receptors for the particular poisons in question.

Differentiation of Complements. The questions as to the nature of complements in serum and as to whether there are numerous complements or only one in a given serum, have been widely discussed. Fisch[3] finds that a number of complements are normally present in rabbit blood, as shown by fractional centrifugation. He, however, says that the importance of these complements towards amboceptors is such that a dominant action exists. This may overshadow other combining forces.

Hemolytic Complements. Sweet[4] reports an excellent study of the hemolytic complement of rabbit serum for the amboceptor for bovine erythrocytes. He finds that the hemolytic complement bodies may be increased in the serum of rabbits when substances are injected that have a positive chemotactic action. Staphylococcus pyogenes aureus, oil of turpentine and sterile suspension of aleuronat were used. The hemolytic complement is not excreted by leucocytes of any type, but is present in the serous part of an exudate. In experimenting with the aqueous humor, it was found that the complement was not present in the

(1) Deutsche med. Wochens., No. 46, 1902.
(2) Centralb. f. die gesammte Therapie, Jan., 1903.
(3) St. Louis Courier of Medicine, June, 1903.
(4) Univ. of Penn. Medical Bulletin, Dec., 1902.

normal aqueous. It was found present, however, in the newly formed aqueous after the increased porosity due to emptying the anterior chamber. This would, therefore, indicate that it must be present in a free state in the circulating blood. No leucocytes are present in this freely formed aqueous, and they are, therefore, excluded in the experiment.

The hemolytic complement in the newly formed aqueous disappears after a short time. This is explained by the return to normal after readjustment of the circulation.

The author's experiments with plasma indicate that the complement is present in a free state in the circulating blood plasma, and is not set free from the leucocytes by coagulation.

As regards artificially increased natural resistance induced by intraperitoneal injections, this is to be explained by the alteration of natural osmosis between the cavity and the membranes which separate the circulating blood from the peritoneal cavity. The complements then pass into the peritoneal cavity and there reactivate the normal amboceptors, which are either pre-existing in the normal lymph of the peritoneal cavity—for they are dialysable—or which arrive with the complements. The natural resistance of the animal to intraperitoneal infection is, therefore, increased by the natural accumulation at the site of the injection, of normal complements and normal amboceptors.

Bacteriolytic Serum Complements in Disease. As the study of serum complements progresses, it becomes more and more interesting to see what practical deductions will be evolved. The variation of these bodies during disease is at once full of interest. Longcope[1] repeats his observations as to variations of these bodies in health and chronic affections, terminal infections and in typhoid.

For the experiments, 25 to 150 c. c. of blood was used, the serum allowed to separate out in the ice-box, and in from eighteen to twenty hours after the blood was taken,

(1) Univ. of Penn. Medical Bulletin, Nov., 1902.

the clear supernatant fluid was drawn off by means of sterile pipettes. Control cultures were always made from this serum, but in no instance was a growth of bacteria obtained. The bacteriolytic action of the serum was then tested for two organisms—B. coli and B. typhi—the same strain of both organisms being used throughout the experiments. Emulsions from eighteen-hour agar slants were made in normal salt solution, and one *oese* (a standard loop) from each salt suspension plated for control. Six *oese* of each salt suspension were then added to 1 c. c. of unheated complement-containing serum, and 3 *oese* of this mixture plated immediately. The mixture was at once put in a thermostat at 36.5° C., and at intervals of one, five and twenty-four hours, 3 *oese* were again plated. The Petri dishes were kept at 36.5° C. for at least twenty-four hours, when the number of colonies on each plate was estimated by means of plate glass ruled into small squares, the colonies if numerous being counted under the 3 ocular. In spite of certain difficulties, such as the formation of a single colony by agglutinated bacilli, the method proved surprisingly accurate when as controls two or three sets of plates were made from the same tube of serum.

Table I. shows the condition in a healthy individual as regards the complement. See page 156.

The conditions as found in patients are divided into three groups: I. Bacteriolytic action of the serum much diminished; reactivation difficult; 10 cases, 7 deaths. II. Bacteriolytic action of serum very slightly reduced; reactivation fairly good; 5 cases, 2 deaths. III. No reduction in bacteriolytic power of serum reaction; 2 cases, 2 deaths.

Table II. is illustrative of the complement condition in an advanced serious case.

The following conclusions state very clearly the results of the observation, as these are a possible direct bearing as to the prognosis of cases they are all the more interesting.

Normal individuals show slight fluctuations in the bacteriolytic complement-content of their blood.

TABLE I.

Observation I. W. T. L., aged twenty-five years. No typhoid. Healthy.
Blood drawn June 11, 4 P. M. Serum used June 12, 3.30 P. M.

			Control.	*Immediate.*	*1 hour.*	*5 hours.*	*24 hours.*
1 c.c. of unheated serum	+	B. coli	38,000	6,820	3,180	2	Sterile
1 c.c. " "	+	B. typhi	45,000	10,000	5,350	15	"
1 c.c. of heated serum	+	1/20 c.c. unheated serum + B. typhi	45,000	9,950	5,180	270	"
1 c.c. " "	+	6/10 c.c. " " + B. coli	38,000	4,900	2,600	3	"

TABLE II.

Observation XXI. W. D., male, aged thirty years. Typhoid fever, ninth day of disease. Death, September 2.
Blood drawn September 15, 4 P. M. Widal, negative, September 15.
Serum used September 16, 12 M. Blood cultures September 15: B. typhosus.

			Control.	*Immediate.*	*1 hour.*	*5 hours.*	*24 hours.*
1 c.c. unheated serum	+	B. coli	40,000	11,000	1,300	27	Sterile.
1 c.c. " "	+	B. typhi	50,000	16,500	960	3	"
1 c.c. heated serum	+	1/20 c.c. unheated serum + B. typhi	50,000	16,800	4,500	5,500	Innumerable.
1 c.c. " "	+	1/10 c.c. " " + B. typhi	50,000	13,200	3,820	1,620	"
1 c.c. " "	+	2/10 c.c. " " + B. typhi	50,000	15,600	2,120	38	36
1 c.c. " "	+	6/10 c.c. " " + B. coli	40,000	9,000	1,480	220	Sterile.

In many prolonged chronic affections, such as nephritis, cirrhosis of the liver and diabetes mellitus, there is a marked decrease in the bacteriolytic blood complement, which becomes more marked toward the end of the disease.

Terminal infection in chronic disease is probably the direct result of the diminished state of the bacteriolytic complement.

The blood serum of certain individuals suffering from chronic disease does not show a reduction in complement; these individuals appear to escape terminal infections.

Hyperleucocytosis is frequently associated with high complement-content of the blood serum for typhoid and colon bacilli.

The blood serum of some typhoid fever patients shows a diminution in the specific complements for the typhoid bacillus.

Human blood serum contains a multiplicity of bacteriolytic complements.

Antihemolytic Action. Noguchi[1] reports some interesting observations as to the effect of blood sera, milk and cholesterin in delaying and preventing hemolysis. As to the active hemolytic agencies, agaricin, saponin and tetanolysin were used. The opposing action of tetanolysin and cholesterin are very typical. The following tabulation shows this result:

Tetanolysin	1 mg.	+	cholesterin	1 mg.,	+	human corpuscles	—marked hemolysis.
"	"	+	"	"	+	guinea-pig's "	— " "
"	"	+	"	2 mg.	+	human "	—trace of hemolysis.
"	"	+	"	"	+	guinea-pig's "	—marked hemolysis.
"	"	+	"	2.5 mg.	+	human "	—no hemolysis.
"	"	+	"	"	+	guinea-pig's "	—marked hemolysis.

The writer summarizes his results as follows:

1. Blood sera and milk exert more or less antihemolytic action against certain phyto-hemolysins and bacterial hemolysins, and their action is non-specific.

2. The manner of anti-hemolytic action of sera and milk agree with the manner of action of cholesterin and lecithin against these hemolytic agents.

(1) Univ. of Penn. Medical Bulletin, November, 1902.

3. It is highly probable that this anti-hemolytic action of sera and milk depend either wholly or in part upon their cholesterin constituent.

4. Agaricin contains a hemolytic principle of considerable activity.

Alkalinity and Hemolytic Power. Jordan[1] reports experiments which indicate that the alkalinity of bacterial filtrates and other fluids influence the occurrence of hemolysis to a marked degree. In view of the fact that certain fluids retain their hemolytic properties after heating to a point that destroys such agents in serum it would appear that some special condition is operative. One group of the author's experiments shows that normal salt solution which in itself has no hemolytic activity, develops this property for the blood cells of the dog, hog, cow, sheep and rabbit, depending upon the amount of normal sodium hydrate solution that is added, 1 per cent or 5 per cent giving strong or complete hemolysis. Cultures of B. pyocyaneus were studied with this end in view. Filtrates from cultures of this bacillus show an increase in hemolytic activity as they become older. The writer shows by titration that the alkalinity of such filtrates is more marked in older cultures. In the experiments the hemolytic activity follows very closely the degree of alkalinity of the filtrate. By passing a stream of CO_2 through such filtration the alkalinity is destroyed as well as the hemolytic activity. On heating, the CO_2 is expelled and a normal status is re-established.

The Precipitation of Bactericidal Bodies by Plant Jellies. Lingelsheim[2] reports experiments somewhat similar to previous observations, in which plant jellies, especially Iceland moss solutions, showed the capability of precipitating the bactericidal elements of immune serum. If a few drops of a 1 per cent to a 1.3 per cent solution of Iceland moss jelly are added to immune serum, a slight clouding will quickly become apparent and a fine gelatinous precipitate form. The serum will now be found to have

(1) Jour. of Med. Research, August, 1903.
(2) Zeits. f. Hygiene, Bd. 42, No. 2, 1903.

largely lost its germicidal activity. The reduction of alkalinity in a great measure facilitates this action. The ordinary albuminous constituents of the serum are not precipitated. When the serum is greatly diluted and contains very little salt and when the reaction is neutral or acid, this jelly will precipitate all albumins, and may become a very delicate test for them.

Typhoid Bacillus Immunization Against Agglutins. Mutter.[1] Typhoid was cultivated in immune serum 1:50 and 1:12,500 and 1:25,000 for periods of twenty-four hours and then suspended in bouillon and tested for agglutination. He finds that there is a great decrease in agglutinatibility after such treatment. In one case forty times more concentrated dilutions were required. Transfers to bouillon for twenty-four hours led to a disappearance of this property. The author does not find that distinct immunity can be acquired by bacilli that have been subjected to this procedure.

Lympho- and Myelotoxic Intoxication. The interesting record of experiments by Flexner[2] on the pathologic changes due to the injection of lymphotoxic and myelotoxic sera show that the changes in the lymph glands, spleen and bone marrow are unmistakable, often very rapid and at times profound. An absence of specially elective action in these organs as regards the various cytotoxins used, is undeniable. Lymph glands, intestinal lymphoid tissue and spleen respond markedly to marrow toxin; the response of the marrow to the lymphotoxins and splenotoxins is far less marked. The following account of the changes is interesting:

Rabbit Injected with Myelotoxin. "The enlargement of the *lymph glands* is due to an active increase in lymphoid elements. The cortices are enlarged and the germ centers are proliferating rapidly, while many eccentric areas of cell multiplication are found. Degenerations of all kinds are infrequent. The medullary portions of all glands

(1) Muenchener med. Wochens., No. 2, 1903.
(2) Univ. of Penn. Medical Bulletin, Nov., 1902.

show an increase in cells within the meshes and free within the sinuses. The peripheral sinuses are also rich in cells. While many of these cells partake of the type of lymphoid cells, a larger cell both as regards the size of the nucleus and amount of protoplasm is met with, especially in the sinuses. Its character has not been exactly determined. Granules were not discovered in the protoplasm. Giant cells are not infrequently seen, and in some glands they are relatively numerous. In type they differ from the giant cells of the bone marrow. The protoplasm is pale, the margin irregular and frayed in appearance, and the nuclei, which are usually peripheral in position, are discrete and pale. These cells differ much in size, and vary from a cell the size of two or three of the pale cells of the germ centers up to a dozen or twenty such; they contain finely granular, yellow pigment, which may be altered hemoglobin. Their origin is not made out.

The enlargement of the spleen is brought about especially through the increase in size of the Malpighian bodies. These structures encroach upon the pulp which is reduced in quantity. The Malpighian bodies are several times the normal size, and cells in all parts are undergoing the most active division. Mitoses in all stages, and many to the microscopic field, are encountered. Giant cells were not seen. Degenerations are uncommon.

The *bone marrow* is converted into an almost solid tissue composed chiefly of white cells. The latter consist, for the greater part, of granular mononuclear and polymorphonuclear cells. The former exhibit, for the most part, fine amphophile granulations; but the eosinophilic cells with coarse granules are also increased, and giant cells are strikingly common. Mitoses occur, apparently in the mononuclear white cells. Erythroblasts are abundant. Degenerations here, as in the lymph glands and spleen, are inconsiderable.

Lymphocytoxin and splenocytoxin were used in the same way, and the histologic changes in the organs are described. They do not differ widely from those in the case noted.

As to the primary change there can be speculation. Do they begin in the circulating blood cells? It would seem further that important changes in tissues will be later recognized and taken into account where the visible injury is not the only evidence of the process.

Bacterial Distribution and Pathogenesis. The two following observations are rather isolated studies of questions that were the leading subjects of investigation some years ago. It is evidently true that repetition of the older investigations will show many interesting facts that may be turned to practical account.

Anaerobic Bacteria of the Intestines. Friedman[1] reports finding anaerobic bacteria in the cecum in sixteen cases under examination as follows: Most frequently isolated was *B. aerogenes capsulatus,* separated 9 times. Next the drumstick bacillus of Rodella, separated 5 times. Third, an unidentified bacillus, "No. 5," separated 4 times. The next in frequency were three organisms each isolated 3 times, namely, Bacillus No. 6, *B. muscoides* of Liborius and *B. cadaveris* sporogenes of Klein. The writer found that upon removing pieces of intestine the facultative bacteria grow rapidly at first, and later, after four or five days, the obligate anaerobes increase rapidly. The same condition is produced in the intestine by constipation or strangulation. At times some of these species may cause pathologic changes or toxemia.

Pathogenesis. The generally recorded statements that certain bacteria are entirely without pathogenic properties, are receiving from time to time controverting evidence. Bacillus subtilis has been presented as a pathogenic species under circumstances. Very recently Bertarelli[2] has reported a closer study of this organism which shows that sometimes it is capable of causing toxemia and some inflammatory changes in experiment animals. It is recognized that prodigiosus is capable of increasing virulence as in association with streptococci, and on the other hand

(1) Trans. Chicago Path. Soc., Nov. 12, 1902.
(2) Centralb. f. Bakt. I Abth. Originale Bd. xxxiv, No. 3, 1903.

can decrease virulence as in association with anthrax bacilli. It may be that the low virulence of prodigiosus may have something to do with these facts. Young cultures are usually not pathogenic; it is only when the cultures have grown for 12 to 14 days that a sufficient quantity of toxin has been formed to disturb the experiment animals. The writer makes the interesting statement that in cases where the animals are not killed by the injected bacteria these may be found in the circulating blood 3 or 4 days after the injection. He also found that the passage of cultures through white mice augmented pigment production, and even sometimes as in one of his cultures the color was maintained when the cultures were maintained at 37° C.

The general results of inoculation experiments on guinea pigs show that prodigiosus can cause a fatal toxic septicemia when medium sized doses are used. However, at the same time there is no evidence of growth in numbers of the bacteria that are present in the blood or tissues. These facts call attention to the possibility of injurious results from infection with these non-pathogenic forms.

SECTION IV.

HYGIENE.

Food. Wesener[1] calls attention to the necessity of measuring food value in diet. The science that is proving of such value in animal feeding, must be taken into account in the further study of diets for man. The chemistry and physics of food have reached the point where their simple theoretic bearings should be turned to practical uses. The flavor of food is a most important factor, and without it, no diet can be expected to be well borne. The temperament of the individual is of importance in selecting a diet. Fruits especially, do not agree with nervous excitable individuals, and the most suitable proteid for him is cheese. The languid plethoric individual should receive less meat than he is usually found to be taking; a proper vegetable diet is more proper for him. The habit of overeating should be replaced by a rational consideration for the wants of the body.

The Albumens in Milk. Schlossmann and Moro have investigated the albumens in milk by biologic methods, and find that the animals immunized with a species of albumen retain a specific precipitating power for that species, but not for others. Thus, serum of guinea-pig I, immunized with cow lactoserum: Cow lactoserum plus cow casein solution, causes precipitation. Cow lactoserum plus cow lactalbumen causes precipitation. Cow lactoserum plus human lactalbumen, causes no precipitation.

Guinea-pig II, was immunized with human milk; the

(1) Illinois Medical Journal, June, 1903.

resulting human lactoserum did not cause precipitation with cow lactalbumen.

Guinea-pig III, was immunized with cow lactalbumen; the resulting cow lactalbumen serum caused precipitation with cow lactalbumen solutions, but not with human lactalbumen solutions.

This difference being apparent, it would seem that the child, of necessity, must convert or alter the constituent albumens of cow's milk before appropriating them, while those from the mother are ready for absorption.

BACTERIA IN INTESTINES.

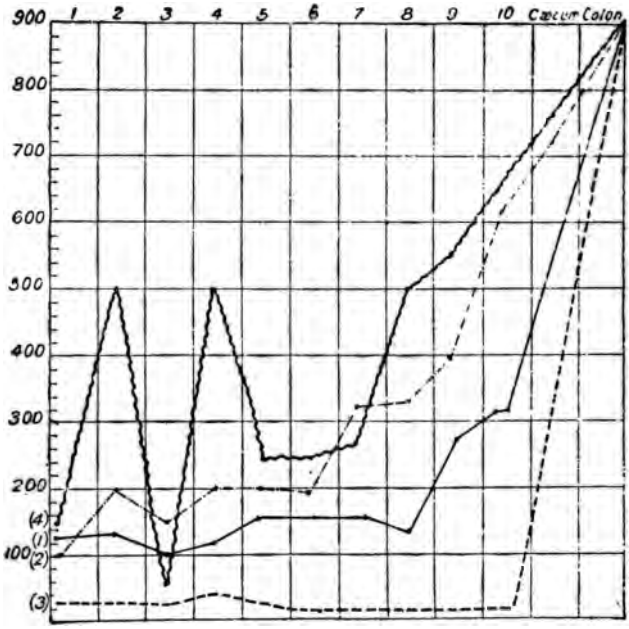

Fig. 22. The number of bacteria which grew on an agar plate inoculated with a loopful of intestinal contents taken at regular intervals.

(1) Continuous line = average results of ten dogs with healthy intestine.

(2) Dotted line = average result of six dogs infected with intestinal worms.

(3) Interrupted line = case of dog with normal intestine, showing very few bacteria throughout.

(4) Wavy line = case of dog in which the intestinal wall was congested.

Bacteria, Growth in Intestine. Smith and Tennant.[1] In a series of experiments on dogs and rabbits, these writers find that relatively little bacterial growth takes place in the upper part of the small intestine. In the lower part it increases greatly and is most active at the cecum. Intestinal parasites and congestion of the mucosa modify the normal bacterial content. See Fig. 22.

Typhoid Dissemination. Two epidemics of typhoid fever occurred during the year that have caused special comment. One was in Chicago, the other in Ithaca, N. Y. The Chicago epidemic has in it some facts of unusual moment, and was made the subject of a special investigation by residents of Hull House.[2] The epidemic occurred during July and August, 1902. It was shown by a spot map that between one-sixth and one-seventh of the typhoid deaths were located in the 19th ward, a district in which there lives only one thirty-sixth part of the city's population. The people are almost all of the working class. Water and milk may have had their place in the epidemic, but as these two supplies are general for all parts of the city they cannot account for the concentration of the infection in this one district. To those who studied the distribution of the cases it was soon apparent that the greatest number were on the streets where sewage removal was most imperfect. Many of the homes are below the street level, and ready opportunity is given for flooding of vaults and areaways by backing up in the sewers All the conditions in 2,002 dwellings were examined: of the entire number 967 were found with sanitary plumbing, 48 per cent; in 148 dwellings the plumbing was in bad order, 7 per cent; the number of vaults out of doors with water supply was 433, or 22 per cent; there were 218 dwellings with vaults with sewer connections, but no water supply, 11 per cent. The old fashioned simple vault was found in 236 residences, or 12 per cent. Spot maps are

(1) British Medical Journal, Dec. 27, 1902.
(2) An Inquiry into the Cause of Typhoid, etc. City Homes Assn., Chicago, 1903.

shown giving the locations of the cases and the character as to sewage removal stated above. From a study of these it is apparent that cases of typhoid were not more numerous in houses where the disposal method was worst. All that can be said is that the distribution follows into the places where the greater number of neglected arrangements exist, and here were most of the typhoid cases. Degree could not be considered, only the general existence of conditions and the cases. The possibility of flies spreading some of the infectious material from some of these vaults was investigated by Hamilton[1] and reported on at length. Flies were caught in two of the localities which were most suspicious and were then placed in bouillon. The tubes were examined for typhoid and allied bacilli. From one locality the typhoid bacillus was isolated in two of the inoculated tubes, and an intermediate organism from another. From the other locality, tubes, inoculated in the same way with flies, showed that typhoid bacilli were present in three tubes, and an intermediate bacillus in one other. These experiments were repeated several times. As near as can be learned, 50 per cent of the existing vaults are located in this ward, and this fact with the findings presented would lend color to the assumption that the concentration of typhoid during the epidemic in this district is due to the direct spreading of bacilli by flies from these cesspools. Details of the investigation are added. Engleman[2] describes a home epidemic in which it was shown that cockroaches were the intermediary agency in spreading the disease from flat to flat.

The Ithaca epidemic must remain as a classical example of water borne typhoid. The community remained blissfully ignorant of its polluted water until the first case of typhoid appeared, and then some 851 cases occurred. The special commissioners[3] report states that undoubtedly 183 cases occurred among the students of Cornell with at least 12 deaths. For a long time it was known that the

(1) Journal Am. Med. Assoc., Feb. 25, 1903.
(2) Journ. Am. Med. Assoc., Feb. 14, 1903.
(3) Jour. Am. Med. Assoc., May 14, 1903.

water derived from the Six Mile Creek was contaminated. The water supply of Ithaca is derived from several sources; all of these being creeks that flow partly through the town. Five districts can be recognized on this basis: No. 1, receiving straight Six Mile Creek water; No. 2, receiving a mixture of Six Mile and Buttermilk Creek water; No. 3, a district in which there is a mixed water as in No. 2, but with the Six Mile Creek much in excess; No. 4, receiving water from Fall Creek, and No. 5, a district dependent upon wells or other than the pumped supply. Upon investigation it was shown that most of the cases were in the area receiving the straight Six Mile Creek water, and most of the other cases in the areas where this water in mixture was supplied. There was only one case in District No. 4, and this person had drank water in other parts of town. Inspection of these creeks show that they are small rapid streams draining small water sheds and that numerous houses are located on their banks. The conditions about these houses show that infectious material could hardly fail to reach the water. An epidemic might have arisen from pollution of either under the conditions. Long before the present outbreak it was known that typhoid occurred frequently, that the character of the water sheds of the streams was unsatisfactory, but no special interest was taken in these facts. In fact, a mild fever present annually and known as Ithaca fever was considered peculiar to this locality. At first the cases were called paratyphoid, and on this account much confusion as to the true status was occasioned. It is remarked, with justice, that as far as sanitary precautions and regulations are concerned, it would not have changed the matter in the least if they had all been paratyphoid, and lastly that the water was unfit to drink as shown by the analyses. In any case water from above could reach the pumping station in about an hour's time. It may be that the large number of young adults among the student body, especially as most of them were non-residents, had much to do with the large number of cases

among this part of the population, 183 cases. As regards information concerning the precise source of infection of Six Mile Creek with the typhoid bacillus the investigation is uncertain. It was impossible to ascertain if typhoid was present in any of the houses in this water shed. At first the epidemic was thought to have had its origin from the discharges of a gang of 100 laborers who were working in a dam about a mile above the water works. The work was discontinued in the middle of December, which was considerably earlier than the time indicated by the incubation period of the first cases. As regards a thaw having carried infectious matter down, it would appear that the causal condition was existant for a considerable period, and was not simply a matter of being dislodged and swept into the stream. In a rapid stream such as this the water is changed very rapidly and frequently. It is now thought that some unrecognized case or cases gave origin to this epidemic. The management of the epidemic presented difficulties because of the relatively large number of cases. Special precautions were instituted to prevent secondary infection from the sick, and it is probable that few if any of the later cases originated in this way. In this connection the findings of Levy and Kayser[1] in a case of typhoid are interesting. In this particular instance the undisinfected dejecta from a case of typhoid were thrown into a cemented vault which was emptied five months later, and the contents used to manure a garden plot. These five months were during the winter period. The land samples of soil were subjected to bacteriologic examination, and as the writers say, "legitimate typhoid bacilli" were isolated. This result would support the view that typhoid bacilli can live a long time under natural conditions and remain as infectious agents in the soil. It shows the necessity for disinfection of dejecta, and the hidden danger that may exist where vegetables are cultivated on land manured with human excrement.

Colon Bacillus and Sanitation. Moore[2], in a rather in-

(1) Centralb. f. Bakt. Abt. I., Original. Bd. XXXIII. No. 7, 1903.
(2) Medicine, March, 1906.

dividual report presented to the Section of Bacteriology and Chemistry of the American Public Health Association, gives it as his opinion that most of the irregular varieties of B. coli that have been described are from polluted water, soil, or lesions of various kinds in man or animals. This fact of irregular habitat may have a bearing on the variations from the typical forms as cultivated from the intestinal contents. From the author's study of such organisms as he has isolated from the intestines of man and animals, he is led to believe that there are not many established varieties. Such forms as are undoubtedly species varieties should rank with the species in importance. Moore thinks that a closer discrimination of described species should be made, to determine differences between them and B. coli communis.

An excellent appendix of tables showing the action of colon bacilli isolated from the intestine, on sugars and milk, is added.

Pathogenicity of Bacillus Coli. Savage[1] takes up this question with a view of determining if a knowledge of virulence of cultures isolated from water would be an indicator of harmfulness. Bacteria from 22 different sources were tested. Of these 15 were from water, 2 from milk, three from excreta, and one each from sewage and a case of malignant endocarditis. The results of the guinea-pig inoculations were as follows:

Source.	Virulent.	Non-Virulent.
Pure water	1	2
Suspicious water	0	3
Contaminated water	3	6
Sewage or excreta	0	3
Malignant endocarditis	0	1
	4	15

These figures are not large, but they show that 1-3 of the

(1) Journal of Hygiene, July, 1903.

bacillus coli from both pure and contaminated sources were virulent: while the organisms from sewage and excreta were non-virulent. The pure water from which the virulent culture was obtained consisted partly of spring, and partly of surface water. These results lend no support to the view that the pathogenesis of isolated colon bacilli is of help in determining the potency for evil of a water under examination. The virulence of B. coli is a variable quality, and one which can be readily lost.

Bacillus Coli in Water. Prescott[1] concludes from an extended study as to the distribution of B. coli and allied forms:

Bacteria corresponding in every way to B. coli are by no means confined to animal intestines, but are widely distributed elsewhere in nature.

The finding of a few colonies of B. coli in large samples of water, or its occasional discovery in small samples, does not necessarily have any special significance.

The detection of B. coli in a large proportion of small samples (1 cubic centimeter or less) examined is imperatively required as an indication of *recent* sewage pollution.

The *number* of colon bacilli in water rather than their *presence* should be used as a criterion of recent sewage pollution.

Inasmuch as some red colonies other than those of B. coli appear on a litmus-lactose-agar plate, this test alone cannot be regarded as diagnostic.

In tests for the presence of B. coli by the usual methods, the possible inhibitory effect of lactic-acid-producing streptococci must not be overlooked.

Petruschky and Pusch[2] add to the suspected water bouillon in about equal amounts, or, in the case of very small amounts of water, more than the measured quantity. Flasks thus arranged and having 1 liter, 100 c. c. 10 c. c., 1 c. c., and dilutions to million parts, are placed

(1) Medicine. January, 1903.
(2) Zeits. f. Hygiene, 43d Bd., 2 Hft., 1903.

in the incubator at 35°-37°-39°-41° C. for twenty-four hours, or until clouding is determined. This clouding they recognize as the thermophiltiter as shown by the smallest quantity of water to cloud. From the cloudy flasks the colon bacillus is isolated if possible by streaking out on agar and studying the resulting colonies by further cultures. The smallest amount of water to show presence of B. coli is stated as the colontiter in the particular case. This does not always follow the thermophiltiter, but in badly contaminated waters it was found that it did. A scale of coli contamination in degrees of 100, 10, 1, .1, .01 is recommended.

Plague Transmission.—Calvert[1], in speaking of the manner of plague transmission, records a case of a Filipino boy who was brought to the hospital some five or six days after sexual intercourse. He had had a discharge for several days, and developed a right inguinal bubo. Gonorrhea had been diagnosed. He died soon after admission, and the autopsy showed plague bacilli in the urethral pus and in the buboes. Cultures confirmed the findings.

Tuberculosis-Spread. Great interest attaches to the possible relation of the influence of the tubercular individual as regards the infection of his surroundings. That tubercle bacilli occur in apartments occupied by such cases has been often demonstrated. The general question, however, always awakens renewed interest. Recently Romberg and Haedickel[2] in Marburg report observations on the influence of the dwelling on tuberculosis. That the most active warfare against this disease should be waged amongst the middle classes would seem to be required by statistical evidence. A study of this particular point in Hamburg shows that mortality from tuberculosis follows closely the income conditions of individuals. Thus amongst groups of persons 1,000 in number the mortality was found to rise 1.07, 3.93 and 5 per M. as the wage per

(1) American Medicine, Jan. 24, 1903.
(2) Deutsche Archiv f. klin. Med., Bd. 76, Heft 4 and 5, 1903.

individual was less. In Marburg the status shows 197 cases of tuberculosis among 18,000 inhabitants, or 1.1 per cent. If the population is divided into groups, according to financial circumstances, it was found that among the poor class, or one-fifth of the population, there were 167 cases or 4.7 per cent. The remaining four-fifths showed only 0.2 per cent. The writers remark that this may be unusual when compared with other cities, but do not think that any great discrepancy can exist. A canvas of the situation in Marburg shows that of 1,503 houses, as few as 2.6 per cent had been the homes of 34 per cent of all cases of tuberculosis. From 33.6 per cent of the houses, or those occupied by the poorer class, there occurred 59 per cent of all cases of tuberculosis. This would strengthen the view that tuberculosis is a disease of the home. A study of the character of the houses as regards situation, construction, light and air, did not seem to show a close bearing on the question. Neither could the conditions as regards cleanliness be brought to bear, although it must be said that in clean, well ordered homes, very little tuberculosis was found. As regards tuberculosis in children, the evidence of other influences beyond the home seemed to affect these findings. The results as regards older persons show that the home becomes more and more infected as time goes on, and the conditions become more deplorable in cases where the absence of knowledge and care of the sick individuals is less. The number of tubercular patients coming from such homes must necessarily increase under these circumstances.

Infectiousness of Apartments. The question as to the distribution of diphtheria and tubercle bacilli in rooms is reviewed and some facts added in a paper by Hill[1] presented at the New Orleans meeting of the American Public Health Association. Rooms in which there had been cases of infectious diseases were examined by cultures, stained specimens and animal experiments. It was found in these tests that in such rooms the bacillus of tuber-

(1) American Public Health Ass'n. Trans. XXVIII., 1902.

culosis could be demonstrated present more often than the bacillus of diphtheria. It is possible that the length of time of occupation of the room had something to do with this result. It was further shown that the bacillus of tuberculosis was less resistant to drying than diphtheria. In both cases the tests were for living bacteria. It would therefore appear that towards or at the end of diphtheria the living bacilli would be more particularly in evidence on the bedding and bed clothes of the patient, while there would be few to be killed by aerial fumigation. In tuberculosis, however, the number of bacilli expectorated is increasing constantly, and the number about the room is being augmented. The necessity for disinfection is logically then more advisable in the care of tuberculosis. This would agree with the growing feeling of the importance of disinfection after tuberculosis.

Vaccine. *Bacterial Impurities.* The inspection and control of vaccine virus has become a necessity since our knowledge of the defects that may be possible, and particularly because a clean and relatively bacteria free and efficient product can be produced. Rosenau[1] in an interesting bulletin has recorded observations extending over a year, and the examination of specimens from ten producers. The primary object of the experiments was to settle the point as to what part of the untoward clinical results, sometimes noted in the vaccinations, could be attributed to the vaccine virus. Glycerin has an important place in the preparation of vaccine. By a mild germicidal action the pus cocci derived from the skin of the calf are killed while the vaccine virus itself it preserved. It would appear from the general results that producers are depending very much upon the sterilizing effect, and possibly at times overlook real cleanliness in the necessary manipulations. A vaccine pulp that has been prepared as cleanly as possible, in the first place, and then glycerinated should be relatively free from bacteria. It is a

(1) Hygienic Laboratory. Public Health and Marine Hospital Service Bulletin No. 12, March, 1903.

necessity of this method that the glycerinated pulp be stored for several weeks in order to give opportunity for the sterilization to take place. Material that is distributed before the required period has elapsed may, therefore, show an excessive number of bacteria. The use of this green glycerin lymph has given rise to results that have discredited the entire method. The period of utility for this lymph is sharply drawn and must be recognized by the producer as to the time of its beginning, and by the user as to the ending of the period. The variable number of bacteria found in tubes, bought in the open market, shows that no definite rule is followed by producers. A comparison between the bacterial findings of points and tubes show that when properly aged the glycerin product uniformly contained a smaller number. When fresh there may be more than on points. As regards varieties of bacteria, the experimenter found that pus bacteria were abundantly present in fresh material. In aged glycerin product they were few in number or absent entirely. Staphylococci predominated, and streptococci were present quite frequently. The tetanus bacillus was looked for in many tubes of glycerinated lymph, and on many points, but none was found. After trying several methods, animal experiments were made and quantities of the specimens were injected into animals, all showing a negative result as regards tetanus. The writer found when experimenting with tetanus, that when added to glycerinated lymph no growth occurred, and a decrease in toxicity took place. This action, however, is not rapid enough to offer any protection should it happen that the vaccine becomes contaminated. It was further found that the tetanus spores would live a long time, over a year, in glycerinated vaccine. When tetanus was added to dry points it was found to dry out more quickly than in glycerin lymph.

Gehrmann[1] states the following as being the *standard requirements for the vaccine* used in the Chicago Health Department:

(1) Journal American Medical Association, April 25, 1903.

Deliveries are made in lots of 5,000 to 10,000 vaccinations. Only individual tubes are used. For each lot the following conditions must be fulfilled:

The lot must be from one operation; that is, it must bear the same laboratory number throughout.

The dates on the packages must not precede or exceed the time limit of efficiency. Sixty-day emulsion with 40 to 50 days for the period of use.

The physical conditions must show a uniform emulsion.

Cultures made must show an absence of pus bacteria.

Cultures made must show at least a relatively small number of saprophytic bacteria.

The injection of the mixed contents of 10 or 12 tubes into a guinea-pig must be negative.

The clinical test must give not less than 90 per cent in typical results in primary vaccinations.

Green[1] proposes to replace the glycerin used in *preserving and sterilizing vaccine virus by the use of chloroform water* 1:200. He finds that the bacteria are killed off much more quickly, only a few hours being required to sterilize samples. The vaccinal efficiency is not impaired to such an extent as to interfere in any way with the practical results. The entire period is, however, less than for the glycerin process. The following detail of the method of preparation is described:

Vaccine emulsion is first prepared by triturating vaccine pulp with distilled water. *The presence of the water is essential* in order that later chloroform may enter into solution with it. About three parts by weight of water should be mixed with one part by weight of pulp. Should a more viscid emulsion of vaccine be desired, glycerin may be added without interfering with the action of the chloroform.

The newly made vaccine emulsion to be subjected to the action of chloroform is dealt with in the following way. Sterile air is first passed through pure liquid chloroform, whereby this air becomes charged with chloroform vapor.

(1) The Lancet, June 20, 1903.

This mixture of air and chloroform vapor is then passed through the vaccine emulsion which is contained in a cylindrical glass vessel of test tube shape, and in size suitable to the quantity of vaccine to be treated. The mixed chloroform vapor and air can be passed seriatim through a number of tubes of vaccine before it finally escapes into the outside air, and it is efficient for all of them, provided that the current be sufficiently strong to keep the contents of each tube in active movement, and that a distinct smell of chloroform be apparent at the outlet of the last tube of the series. It is essential that no liquid chloroform be allowed to pass over into the vaccine, as its presence is strongly inimical to the potency of the lymph. To obviate the chance of such an accident, an overflow bottle, weighted with sterile sand, is interposed between the bottle of liquid chloroform and the tube or tubes of vaccine emulsion. By passage through it of chloroform vapor and air, the water of the vaccine emulsion quickly becomes saturated with chloroform, and this strength of solution is maintained so long as such passage is continued. When saturation is reached, all excess of chloroform immediately escapes automatically from the vaccine. Thus the vaccine is not at any time brought into contact with a stronger solution of chloroform than 1 in 200 in water.

Tetanus in Gelatin. Much interest has attached to this question because of the use of gelatin in the treatment of aneurisms. Several instances have been reported in which tetanus followed such injection, and these have brought the subject to special notice. Anderson[1] has taken the opportunity to investigate commercial gelatin for the presence of B. tetani. Seven samples were examined and in one tetanus spores were found. Levy and Breen[2] found the tetanus bacillus in four out of six samples that they examined. The method used by the writer was to take ten grains of each sample and place it in 100 c. cm. of glucose bouillon and heat at 50 C. until

(1) Hygienic Lab., Pub. Health and Marine Hos. Service. Bull. 9, September, 1902.

Deutsch med. Wochens., Feb. 20, 1902.

dissolved. The solution was neutralized and then heated to 80° C. for ten minutes to kill any sporing varieties that might be present. The flasks were now quickly placed in Novy jars and boiled *in vacuo* to expel all the air. They were then placed in the incubator and kept at 37° C. for one week. Of the seven specimens three remained sterile, and the other four showed end-sporing bacilli. Animal experiments proved that one of these was B. tetani. The practical suggestion is made that gelatin to be used for therapeutic purposes be sterilized by the fractional method.

Stream Examination. Report of the Sanitary District of Chicago.—The report of the special commission appointed by the Sanitary Trustees forms a volume of 140 pages and 196 tables. It is truly monumental in its bearing on the purification of polluted water courses. The director of the investigation was A. R. Reynolds, M. D., and the laboratories engaged were the University of Illinois, A. W. Palmer and T. J. Burrell; the University of Chicago, Prof. E. O. Jordan, and the Laboratory of the Chicago Department of Health, Dr. Adolph Gehrmann.

The summarized results show conclusively that purification to a remarkable degree is taking place in the Illinois River as it flows towards the Mississippi. Again and again, the evidence of pollution by the cities below Chicago disappears, until the point where the next lower pollution occurs. The totals of bacteria count and the colon bacilli both partake in this numerical decrease. Bacterial averages for the points of greatest pollution show as follows (August to December, 1899):

	Lockport, Ill. and M. Canal.	Joliet.	Wesley City.	Pekin.
August	3,215,000	3,220,000	2,355,000	1,928,000
September	669,600	217,000	484,500	391,250
October	1,359,375	1,145,000	2,373,750	1,047,500
November	582,000	1,023,000	705,000	812,000
December	746,250	1,026,250	20,600	69,000

The numbers are all very large. The variation usually is not more than is ordinarily anticipated, except those for

December at Wesley City and at Pekin. These, compared with the others given and with those usual for these stations, are very exceptional and must have some special explanation. Upon the latter, however, it is idle to speculate. Let it be remembered that there is no connection between the first two sets of figures and the last two sets, for Joliet and Wesley City are separated by a long line of the waterway, from which the samples show very low counts.

The Ottawa station is 48 miles from that of Joliet, and Averyville is 74 miles from Ottawa. Havana is 33 miles from Pekin and Kampsville is 81 miles from Havana. The average counts for these places for the same time as above were as herewith given:

With these are to be compared the figures at points below which indicate to what an extent the bacteria have disappeared.

	Ottawa.	Averyville.	Havana.	Kampsville.
August	7,500	4,000	5,700	6,200
September	40,100	3,100	6,137	3,420
October	33,050	3,770	12,420	1,717
November	24,750	2,800	164,000	6,850
December	261,760	9,300	201,300	28,850

From these and other figures, it appears that so far as Chicago's bacterial contribution to the pollution of the river is concerned, it is manifest for summer periods at least as far as Ottawa, while in winter it passes a short way further down stream. As regards the results of the chemical analyses, it is apparent that only the inorganic constituents pass along with the stream, namely, chlorin and the nitrates and nitrites that have been formed during the various processes of purification.

The opening of the main drainage channel on January 1, 1900, does not materially alter the conditions that existed in the Illinois River, except as regards the upper stretches, namely, from Lockport to Peoria. Throughout this part of the river, all the constituents as shown by the chemic and bacterial analyses are decreased to a greater or less extent, while in the lower stretches and at the mouth of the river any change that might be attributed

to the diluting effect of the channel is so irregular and slight that its significance is lost. In this lower part of the river the chlorin is the only element that can be attributed as coming directly from Chicago. The organic matter, even if it originally did start from there, has evidently passed more or less times through the bodies of plants and animals to change it to the degree observed.

The presence of the typhoid bacillus was sought by all of the investigators, and was not found in any of the samples. Taking into account the known vitality of this organism as compared with B. coli, it appears clear, even if it were demonstrated as being present, that it would disappear to a greater and more rapid degree than demonstrated for the colon bacillus.

When compared to the Mississippi or to other streams in Illinois, it is shown that the Illinois River, after a flow of 150 miles from Chicago, practically returns to normal for a river flowing through a cultivated region.

Ozone and Water Purification. Proskauer and Schneider.[1] The writers report a description of the ozone purification works of Siemens and Halske in Wiesbaden. These works are capable of treating 250 cubic meters of water per hour, but during average demands, 125 cubic meters is the amount purified. The plant consists of an engine house, ozone developing room and the rooms with the sterilizers. In the engine room are the motors, blowers and pumps. The ozone generators are in two groups, 24 circular cast-iron generators being in each set. They are arranged in 4 tiers of 6 each. In the sterilizing room there are two rows of sterilizing towers, four in each row. These towers are 4 meters high and divided by cross-partitions into four compartments. They are filled to a depth of 2 meters with crushed stone. About 40 cubic meters of water pass through each of these towers per hour, and at the same time about 80 cubic meters of ozone is forced through under slight pressure. The equipment is in groups so that any set can be disconnected, and the

(1) Zeits. f. Hygiene, Bd. 42, No. 2, 1903.

flow of water and ozone is all operated automatically. Under ordinary operation the following figures indicate the bactericidal effect exerted:

	Raw Water.	*Ozone Treated.*
June 24.....	298	3
June 25.....	682	11
June 26.....	290	2
June 27.....	250	2

Bacteria per cubic centimeter.

Artificially infected water gave these results:

	Raw Water.	*Ozone Treated.*
June 28.....	39,000	8
June 30.....	26,000	12
July 2.....	55,000	5

Bacteria per cubic centimeter.

It was found that an excessive amount of ozone had to be applied in order to accomplish complete sterilization, and that this would have to be determined specifically for each case. A detailed statement as to operation experiments and expense is shown.

SECTION V.

ANATOMY.

GENERAL.

Dissection at Home. Jackson[1] thinks that the general practitioner should keep up his anatomy better than he does. This will necessitate home study, and the author suggests that it is possible for him to dissect at his home. He should always ascertain the legal status of dissecting in his state. Then if he cannot secure the body of an adult he should use a fetus as near full term as possible. Any practitioner can inject a body. Jackson advises the following method: Having picked up and opened the blood vessel inject with any large syringe. The best fluid is made by mixing equal parts of 95 per cent alcohol, melted carbolic acid crystals, and glycerin. Inject slowly and with a fair amount of pressure. Use 6 to 8 quarts of fluid for the body of an adult. Consume an hour in making the injection. In 24 hours much of the fluid will have diffused into the tissue. Then inject the color mass for the vessels. This can be equal parts of plaster of paris and starch, colored with red lead. This is ground, sifted and then mixed with water, to the consistency of a thin paste. Watch the cadaver closely for several days. If any part of the body becomes discolored inject some formalin (25 per cent) with a hypodermic or aspirating syringe. Drying is prevented by wrapping in water-proof cloths.

New Method for the Demonstration of the Framework of Organs. Flint[2] has modified Spalteholz's method of

(1) Journal American Medical Assoc., Oct. 4, 1902.
(2) Johns Hopkins Hospital Bulletin, March, 1902.

digesting the parenchyma of organs so as to demonstrate the framework. He follows the following plan:

The tissues are cut thin, not over 3 mm. in thickness. They are then hardened in Van Gehuchten's fluid. This fluid is made as follows:

Glacial acetic acid	10 parts
Chloroform	30 parts
Absolute alcohol	60 parts

Very good results can be obtained with the corrosive acetic mixture, by the use of the graded alcohols, but tissue must not be fixed in formalin, in fluids containing chromic acid or its salts, or those in which osmic acid is an ingredient. Trypsin is powerless to digest tissue that has been hardened in fluids containing any of these reagents. After the fixation it is best to dehydrate the tissue gradually in order to avoid any possible shrinkage. Moreover, great changes in the strength of the alcohols seem to render the digestion of tissue more difficult. After the dehydration is complete the tissue is transferred to ether, placed in one of the filter paper cups and then dropped into the receptacle of a Soxhlet apparatus and is extracted for 5 or 6 days continuously. In Leipzig, the experience with the Soxhlet apparatus has not been altogether satisfactory, and several explosions led them to substitute chloroform for ether in order to escape this danger. The chloroform, of course, does not burn, but at the same time does not seem to extract the fats as well as ether. To overcome this difficulty the Soxhlet apparatus can be placed in a hood and operated by means of hot water obtained from a Fletcher heater situated in another part of the room, or the heater may be placed in the hood and the ether apparatus erected in a sink. In this way all danger of explosions can be obviated and the apparatus may be operated day and night with perfect safety. Prof. Spalteholz writes that he now uses an electric resistance plate to heat the ether flask, a method which is at once safe and

economical if the electric current can be obtained. Another method which involves the use of electricity has been devised by Prof. O'Neill, of the University of California, which is much simpler than the resistance method used in Leipzig. The ether flask is placed in a snugly fitting cylindrical roll of asbestos board. An ordinary electric light bulb, which generates sufficient heat to keep the ether boiling is then inserted in the cylinder beneath the ether flask. Most laboratories are, however, not wired, so that the use of the Fletcher heater in the manner described above offers the only escape from this embarrassing difficulty. [The Editor in similar work makes use of a Soxhlet apparatus with mercury joints. This is placed on a water bath which is heated by the steam of the building. This runs continuously day and night without any danger of fire or explosion.]

After the extraction has been progressing about a week and all of the free fat is dissolved, the tissue is removed from the apparatus and run down slowly through the graded alcohols. Owing to the well known fact that CO_2 is less soluble in a mixture of alcohol and water than it is in either of the two fluids alone, there is always an evolution of gas when they are brought together. It is well, therefore, to make up the graded alcohols sometime before the tissue is placed in them, as the gas bubbles become entangled in the meshwork of the connective tissue and often cause considerable annoyance. After passing through the alcohols the tissues are washed in running water for 24 hours, when they are ready for the digestive mixture. The pancreatin of Parke, Davis & Co. can be used, but the preparation manufactured by Grübler after Spalteholz is better adapted to this use. From comparative tests with weighed amounts of pancreatin and fibrin, it appears that Grübler's pancreatin is more potent and more soluble. The resulting solution, moreover, is almost colorless, which is an important consideration in this technic, as the latter quality of the German pancreatin renders the preparations more transparent while the for-

mer naturally makes the digestion quicker and more effective. Only a small quantity of the pancreatin need be used. In general enough to cover the end of an ordinary scalpel handle, dissolved in 100 c.c. of a 0.5 per cent solution of sodium bicarbonate makes a very effective mixture. Inasmuch as the products of digestion inhibit the action of the enzyme, it is better to use a dilute solution of trypsin and renew it often than to make a stronger mixture and allow it to act over a longer period of time. If the fluid is changed every 48 hours, good results, so far as digestion is concerned, are assured. To prevent putrefaction it is well to use enough chloroform to cover the bottom of the vessel in which the digestion is made. Thymol accomplishes the same result, but has the disadvantage of staining the tissue a dirty brown, which interferes considerably with the sharpness of the picture obtained under the microscope after the digestion and clearing are complete. During the progress of the digestion the heat of the thermostat volatilizes the chloroform and the digestion mixture becomes filled with small bubbles of gaseous chloroform, some of which often lodge in the mesh-work of the connective tissue and cause the digesting organs to float about in the fluid. To avoid this, a small elevated stand can be made out of thick paper which not only keeps the tissue raised from the bottom of the beaker where the debris of digestion collects, but keeps the volatilized chloroform away from the tissue. A porcelain filter plate elevated on bits of glass tubing serves this purpose quite as well as the paper stand.

The fats which are united to protein, of course, can only be extracted after the tissue has been submitted to the action of the digestive fluid so that it is necessary to extract and digest at least twice. Apparently the action of the enzyme is much more effective on some organs than others, for in some cases the digestion may be complete after a double extraction and double digestion, while in others it may be necessary to repeat each process four times before the connective tissue is completely free from cel-

lular matter. Among the organs that digest quickest are the thyroid, spleen, lymph gland and lung, while the salivary glands and pancreas and adrenals digest with much greater difficulty. The pancreas itself is apparently more resistant to its own ferment than any other organ which we have tried. This, of course, applies to the organ only after it has been fixed and hardened, as no experiments were made on the fresh pancreas. In slide digestions the enzyme apparently acts with great ease, since, owing to the thin sections, all parts are freely exposed to the action of the digestive ferment. In pieces, however, this distinct resistance to the enzyme is noted, and even after repeated extractions and digestions some debris remains imbedded in the connective tissue framework. In considering the effect of the nature of the tissue on the digestion we should note perhaps that the method works equally as well on pathologic as on normal tissue. The writer has, for example, digested a tumor of the thyroid which shows perfectly the changes in the framework resulting from the pathologic condition in the gland.

Flint[1] prepared *specimens of the thyroid* by the plan outlined above. These were cleared in glycerin and examined with a stereoscopic microscope. He finds that the capsule has an outer dense layer, and an inner layer that communicates freely with the interstitial tissue of the gland. The follicles are not clumped according to any arrangement. Each seems to be by itself. There was nothing to support the theory that the follicle ruptures and thus explosively discharges its secretion into the blood serum. The anatomic "findings" indicate that the thyroid secretion passes by some form of absorption from the lumen of the gland into the blood.

The Lymphatic System. Hannum[2] calls attention to the lack of accurate information, and the great abundance of misinformation on the subject of the lymph system. This arises from two causes. The first, the great difficulty

(1) Johns Hopkins Hospital Bulletin, February, 1903.
(2) Cleveland Med. Jour. Vol. 2, No. 6, p. 259.

in anatomic study of the lymph system; second, the lack of uniformity in the anatomy of the lymphatics.

Relative to the first point Hannum advocates the use of Leaf's method. This method consists in repeated injections of the vessels with formalin until the tissues become edematous. In spite of the anatomic irregularity of the lymph system, its medical and surgical importance is very great.

Hannum refers to the development of elephantiasis as the result of lymph stasis subsequent to removal of the lymph glands, and other operative procedures that obliterate lymph channels. That this is not the rule is due to the free anastomosis of the lymph vessels and to the rather prompt development of new lymph glands from fat. He quotes Beyer[1] and Warthin as having demonstrated such new growth of glands. He quotes Gelsner[2] as attributing a large part of the *vis a tergo* of lymph circulation to the contraction of the muscle fibers of the gland capsule.

Relations Between the Lymphatics and Connective Tissue. MacCallum[3] reviews the literature bearing on this mooted point. He gives the result of his work, and his estimate of the "findings" in the use of various methods. It is his opinion that the lymphatic vessels are definite structures, with continuous walls, and that they are not continuous with the tissue spaces. The tissue spaces are not entities. They are due to conditions in the tissue and are decidedly adventitious. There are no openings from the lymph capillaries. He agrees with Ranvier[4] and Sabin[5] that the lymph vessels start as buds from the blood vessels, beginning at the point of junction of the thoracic duct and the vein. This branching and budding continues to the blind endings of the lymph radicles. In another article MacCallum[6] demonstrates that there are no openings from the serous cavities into the lymph channels. As

(1) Prager Zeitschrft f. Heilk. Bd. VI., and Archiv f. Klin. Chir., Bd. 49.
(2) Archiv für klin. Chir., 1901, Hft. 1.
(3) Johns Hopkins Hospital Bulletin, January, 1903.
(4) Archiv de Anatomie Microscop. 1897.
(5) American Journal of Anatomy, Vol. I.
(6) Johns Hopkins Hospital Bulletin, May, 1903.

to the method of origin of new lymphatics, MacCallum has seen endothelial buddings result in new capillaries. Sometimes the protoplasm of the cell is tunnelled, and sometimes the column of cells divides to form two columns of cells and pressure opens a lymph path between these. Deficiency of this pressure is responsible for the difference in the usual course of *anlages* in the lymph as compared with those of the blood routes. He is of the opinion that the endothelial cells are specific, and do not participate in the formation of connective tissue.

THORAX.

The Transverse Sectional Anatomy of the Thorax. Berry[1] fixed the organs of the thorax with formalin in the cadaver of a middle aged man. The thorax was then segmented at six levels. Comparison is made with the similar sections made by Braune and by Symmington. Space will not permit a full reference to the findings. Berry thinks it would be much more accurate to speak of the anterior and posterior heart. A horizontal section passing posteriorly through the 8th dorsal vertebra divided all four cavities of the heart. Berry says that the heart is more nearly horizontal than vertical. The only portion of the heart not covered by lung was a portion of the right ventricle lying posterior to the sternum on a level with the 8th vertebra.

Malformation of the Tricuspid Valve. Griffith[2] reports a heart with the following abnormalities: The heart weighed 1,260 grms. The circumference of the aorta was $1\frac{7}{8}$ inches, of the pulmonary artery $1\frac{3}{8}$ inches. The foramen ovale was widely patent. There was well marked continuity of the Eustachian valve with the Thebesian as the coronary sinus opened into the same part of the auricle with the inferior cava. Both auricles were large. The left

(1) Edinburg Medical Journal, July, 1903.
(2) Journal of Anatomy and Physiology, April, 1903.

ventricle was disproportionately large and thick walled. The anterior flap of the mitral measured 2 inches from the attached to the free border. In the tricuspids the following was found: The septal flap was quite isolated from its fellows. The left or great flap was less fringed at its margins than normally, its upper commissure was fixed by confluent and rudimentary chordæ to the septum, quarter of an inch from the septal flap, while, when traced to the right, the ventricular aspect of this flap was found to be obscured by great columns of muscular tissue passing from the upper part of the ventricle downwards to the lower part of the anterior wall, and closely adherent to the valve. Between these columns, in some parts, the fibrous tissue of the valve was visible, and at two places small dome-like projections of this fibrous tissue were seen, with apertures at their apices, whose margins were provided with chordæ tendineæ attached at the other end to the above noted muscular bands passing to the ventricular wall. It would appear from a careful replacement of the cardiac walls where these had been cut that the free margin of the anterior flap had been directed upwards and forwards to the pulmonary artery, and the whole of this free margin would correspond in a normal valve to that part of the flap extending from its apex to its junction with the septal flap. This margin bounded, with the upper part of the septum and the septal flap of the tricuspid, a narrow strait through which most of the blood would pass on its way from the auricle to the ventricle. The rest of the margin of the great anterior segment was obscured by muscular bands as above described.

A probe passed from the pulmonary artery, therefore, could pass in two ways, viz., (1) to the right into a gradually narrowing portion of the ventricle, which became blind at the margio acutus, and into which the two funnel-like apertures above mentioned opened, or (2) down between the anterior and septal flaps into another part of the ventricle.

The deformity of the posterior flap may be best under-

stood by suggesting as a possible explanation of its condition that it had been destroyed at its auriculo-ventricular attachment, and become adherent along its entire free margin with the ventricular wall, a condition associated with complete disappearance of the chordæ tendineæ and musculi papillares, and also associated with a large deficiency in the upper part of the flap and a smaller one nearer the septum.

From the right auricle, therefore, the blood might have passed (1) into the left auricle through the patent foramen ovale, or (2) through the auriculo-ventricular opening into the second part of the ventricle referred to above; here the blood would in the first instance pass in part behind the posterior segment of the tricuspid valve, and reach the main stream by sweeping above its upper margin or going through the deficiencies in its substance, whence it would pass directly to the conus arteriosus and pulmonary artery by going between the free margin of the tricuspid valve and the septum, and indirectly by going through the two crater-like orifices described above as opening into the blind part of the ventricle, whence, of course, it would pass to the left and upwards to the pulmonary exit.

It is clear, therefore, that there must have been some silght obstruction to the passage of the blood through the right ventricle; and in harmony with this we note that the pulmonary artery was much smaller than the aorta.

Division of Cavity of Left Auricle Into Two Compartments by a Fibrous Band. Griffith[1] writes of a heart in which the left auricle was divided into two compartments by a broad fibrous band. This band sprung by several spurs from the tissue of the valve of the foramen ovale. The band then passed upwards and forwards below the upper right pulmonary vein, then along the anterior and left walls of the auricle, having now a downward direction, and passing just below and in front of the left pulmonary veins, and finally was lost on the pos-

(1) Journal of Anatomy and Physiology, April, 1903.

terior wall of the auricle about an inch above the auriculo-ventricular furrow. Thus two compartments were formed. The orifice between them would admit two fingers. The band was cribriform in places. The veins opened into the upper compartment. Griffith thinks that the band was due to an incomplete fusion of the two early divisions of the auricle, namely, the pulmonary vein auricle and the common embryonic heart auricle. The author reported a specimen of the same deformity in 1896. He refers to a similar specimen reported by Martin in 1899, and one by Fowler in 1882.

ABDOMINAL REGION.

Mechanism of Absorption from the Peritoneal Cavity. MacCallum[1] agrees with other investigators that the larger part of peritoneal absorption is through the diaphragm. He thinks that the pleura is a definite structure with a specific, complete, definite lining. The same is true of the peritoneum. Into the diaphragm at a relatively late period, embryologically, the lymphatics grow. These are blind canals with no definite openings into either serous sacs. The different cell layers are composed of delicate cells and transitory openings form between these cells. Through these openings particles, both free and in phagocytes, go. The largest force in this is the pumping action of respiration.

The Form of the Spleen. Shepherd[2] does not think that the shape of the soft organs can be determined with any degree of accuracy unless the organs are hardened before the body cavities are opened. He uses formalin and alcohol. He finds the convex or parietal side of the spleen much more regular in outline than the visceral. The convex surface is frequently bound to the diaphragm by a peritoneal ligament, called the lieno-phrenic ligament,

(1) Johns Hopkins Hospital Bulletin, May, 1903.
(2) Journal of Anatomy and Physiology, October, 1902.

which passes from the upper end of the organ. The visceral surface is subdivided into three: the renal, gastric and basal surfaces of Cunningham. The gastric surface is always sharply curved and it has a border which is frequently sharp and notched. The renal surface is usually flat. The basal or colon area is the most variable of the three. The shape is largely dependent upon the relative pressure of the viscera. The shape of the lower end is either pointed or tetrahedral.

In addition to the lieno-phrenic ligament, noted above, there is the lieno-renal ligament and the gastro-splenic omentum.

Topography of the Gall-Bladder. Carmichael's[1] studies were made on bodies preserved by injecting formalin into the rectum, bladder and stomach. He concludes that the text-book statement that the gall-bladder is opposite the ninth costal cartilage is wrong in 75 per cent of cases. The costal cartilage is very variable in its position. He thinks that any localization of the gall-bladder must be by a vertical and not by a horizontal line: that it usually lies external to Addison's lateral line. He says that a vertical line from the midpoint of the clavicle will cross the fundus in most cases.

Anatomy of the Perirenal Fatty Tissue. Keen[2] has found an arrangement of the perirenal fat that has helped him to locate the kidney in operative procedure.

He says: There are two distinct layers of fatty tissues surrounding the kidney: First, that which should be called the transversalis layer of fat and, secondly, the proper perinephric fat or fatty envelope surrounding the kidney itself. In operating on the kidney, so soon as the fat, bulges through the incision, if it is carefully cut or torn through, gradually deepening the incision or tear, a distinct interval between these two layers will be found, which is recognized by a layer of connective tissue, similar to that which is found, for example, between the internal ob-

(1) Journal of Anatomy and Physiology, October, 1902.
(2) American Medicine, Jan. 31, 1903.

lique and transversalis muscles. The second layer of fat then presents itself. If this is incised or torn through and drawn into the wound, the opening thus made in it becomes a sort of infundibulum or funnel-shaped opening, at the bottom of which the kidney is invariably found. Occasionally the first layer of fat does not extend so far posteriorly as the second layer, but commonly, so far as the operating surgeon is concerned, they lie superposed.

At his request, Dr. A. B. Craig made two dissections of the parts and describes them as follows:

"I find the two fatty layers of which you speak. The external layer is less thick than the internal and is continuous with the transversalis fascia. This fascia in passing posteriorly becomes thin, cellular, and reticular and the fat is embedded in its meshes. After passing through this layer there is a distinct interval before we come upon the thick fatty capsule of the kidney proper, which latter completely envelopes the kidney and is more or less adherent to the true capsule. The fatty capsule has a more or less distinct fibrous covering, which is continuous below with the reticular tissue which passes down with the ureter and surrounds the bladder, rectum, etc., while it is continuous above with the attenuated reticular structure which intervenes between the peritoneum and the caudad surface of the diaphragm, and is continuous across from one kidney to the other.

"It appears to me that this structure is morphologically continuous with that fatty-reticular layer between the transversalis fascia and the peritoneum, often fairly marked in the region of the inguinal rings, and denominated 'properitoneal fat,' and which, therefore, in the region of the kidney would be postperitoneal."

The Lumbar Segment of the Ureter. Robinson[1] describes that portion of the ureter reaching from the kidney to the point where the ureter crosses the external iliac artery. He describes its relations to the abdominal organs and abdominal walls as follows:

(1) The Cincinnati Lancet-Clinic, Jan. 10, 1903.

VISCERAL RELATIONS OF THE URETER.

A. THE VENTRAL visceral relation of the lumbar ureter is first and foremost with:

(1) *The Colon. Right Side.* The colon passes on the ventral surface of the lumbar ureter at its proximal end. This is the most important viscus ventral to the lumbar ureter. Practically there is no right meso-colon, hence the lumbar ureter courses dorsal to the insertion line of the internal blade of the peritoneum which projects ventrally to embrace the right colon. The colon is important because in exploration of the lumbar ureter the colon, with the peritoneum, is forced ventralward and medianward in order to expose the ureter lying on the dorsal surface of the peritoneum. However, in the great majority of subjects the ureter is located median to the line of the peritoneum, which is reflected to cover the right colon, especially in the distal end of the lumbar ureter. The right colon is the greater colon in volume. It frequently passes on the ventral surface of the kidney and covers considerable of the proximal lumbar ureter. In ureteral ptosis the ureter passes more and more dorsal to the right colon. *Left Side.* The LEFT colon passes to the left of the ureter between it and the lateral abdominal wall. Toward the distal lumbar ureter the left colon approaches closer to it until the meso-sigmoid in the region of the intersigmoid fossa crosses the ureter. The difference in colonic and ureteral relations on the two sides renders the exploration of the right ureter the easier.

(2) *The Duodenum on the right side* frequently covers the ventral surface of the proximal lumbar ureter as far as the fourth lumbar vertebra. Distal descent of the duodenal loop may extend to the fifth lumbar vertebra, but this is due mostly to duodenal dilatation, for the duodenum is the most fixed abdominal organ, seldom sharing in splanchnoptosis. On the LEFT the relations of the duodenum to the ureter are less fixed and less extensive; sometimes the fourth portion of the duodenum is imbedded in the

tissue between the aorta and ureter, which may extend to the fourth lumbar vertebra.

(3) The ventral surface of the distal end of the lumbar ureter is in relation on the right side with the distal end of the ileum and mesenteron. The distal mesenteron may be so short that the two ureters are in direct contact or the mesenteries may be several inches long.

(4) The lumbar ureter lies in close relation with the inter-sigmoid fossa and the sigmoid loop. It courses through the base of the inter-sigmoid fossa and emerges at the dorsal surface of the intersigmoid aperture.

(5) The cecum and appendix may rest on different segments of the lumbar ureter with changing conditions.

(6) Segments of the transverse colon (especially in splanchnoptosis) may have indirect relations with the ureter by resting on the peritoneum covering some parts of it.

B. LATERALLY the ureter has the colon more closely adjacent to the right than the left. The cecum may lie in contact with the right ureter.

C. MEDIALLY the lumbar ureter may have visceral relations irregularly with the duodenum and with enteronic loops on both right and left side.

D. DORSALLY the lumbar ureter is in relation with no viscus.

DICTIONARY

OF

NEW MEDICAL WORDS

BY

WILLIAM HEALY, A. B. (HARV.), M. D.

INTRODUCTORY NOTE.

The criterion of what words are new has been the last (1901) edition of Dorland's admirable dictionary. The general scheme of that work has been followed; thus, phrases are to be found under the head of the principal noun, *e. g.*, for Trunecek's serum, see Serum. If recent research has made certain older words much more exactly definable, they have been included; instance the various terms relating to cirrhosis, to mosquitoes, and to the antibody theory of immunity. Careful attention has been paid to the new remedies.

As a considerable record of recent medical progress these pages will repay direct perusal.

DICTIONARY OF NEW MEDICAL WORDS.

A

Abiotrophy. A gradual degeneration or failure of vitality as shown by various cells of the body under various conditions. This may be accompanied by overgrowth of other cells, as shown by connective tissue overgrowth when nerve elements decay.

Ablatio placentæ. Premature detachment of the normally situated placenta.

Acestoria. A local dental anesthetic.

Acetopyrin. Chemical compound of acetyl salicylic acid (aspirin) and antipyrin. White, crystalline, sparingly soluble in water. Antiseptic, analgesic, antirheumatic. Dose: 3 to 5 grains.

Acetozone. Benzoyl-acetyl-peroxide. Crystalline. Slightly soluble in alcohol, ether and oils. In water soluble from 1:1000 to 1:10000. A non-toxic germicide for internal and external use. Dose: 3 to 5 grains, the solution in proportion. As dusting powder or in ointment from 1 to 5 per cent strength.

Acholuria. Absence of bile pigments from the urine, characteristic of one variety of icterus.

Acid, Isanic a. A crystalline acid derived from isano tree. Strong purgative.

Acocantherin. African arrow poison.

Actinolite. An apparatus for generating the ultraviolet rays, actinic light. It consists essentially of an electric arc light and adjustable lenses.

Actinotherapy. Treatment of disease by chemical or actinic light, the ultra-violet rays.

Adnexopexy. Surgical elevation and fixation of the Fallopian tube and the ovary.

Adrenalin. The isolated active principle of the suprarenal gland. Used mostly as the hydrochlorid in weak solution. Local and general hemostatic. Raises blood pressure.

Adrenoxin. The organic compound or oxidizing substance which is said to form in the lungs from loose combination of the internal secretion of the adrenals with oxygen.

Agglutination. The clumping together of bacteria, evidence of detrimental effect upon the growth and virulence of the organisms.

Agglutinin. A specific uniceptor antibody elaborated by an infected or immune organism, which has the power of clumping together the corresponding bacteria in pure culture.

Agrafage (ag-ra-faj) . [Fr. clasping]. The method of suturing a cutaneous wound by means of double-pointed agrafes or clamps. The skin is not pierced by the points which bring the edges of the wound in firm apposition.

Agurin. Acet - theobromine - sodium. A white hygroscopic powder, incompatible with acids. For dropsical effusions with healthy kidneys. Dose: 7 to 15 grains.

Airol. Bismuth oxyiodogallate. Greenish, odorless powder, insoluble in water and alcohol. Moistened it decomposes into bismuth and iodin. A substitute for iodoform.

Akouphone. An electric apparatus on the principle of the telephone, for aiding hearing.

Albargin. Gelatose silver product containing 15 per cent silver. Light brown powder. For gonorrhea in solutions of $^1/_{10}$ to 2 per cent.

Albugineotomy. Incision of the tunica albuginea from pole to pole on the convex surface of the testicle.

Albumosuria, Myelopathic a. A disease characterized earliest by albumose in the urine and later by rheumatoid pains and softening of the bones.

Alexin [Gr. alexo, to ward off]. The same as complement, q. v.

Algophily. Desire for experiencing pain, a form of sexual perversion.

Alkalithia. A proprietary granular preparation containing in each dram caffein 1 grain, lithium carbonate 5 grains, bicarbonate of soda and potassium of each 10 grains.

Alkarhein. A proprietary name for alkaline elixir rhubarb compound with pancreatin.

Alkathymol. An alkaline antiseptic solution for use on mucous membranes.

Allamanda cathartica. A South American plant used as a cathartic. Dose: watery extract, 1 to 2 grains; of the juice, 10 drops.

Amboceptoid. An amboceptor which has lost its affinity, its haptophores, for receptors in cell molecules, or in complement molecules.

Amboceptor [L. *ambo*, both; *capto*, to take]. A thermostable chemical substance found in the blood serum as the result of immunization. It is the one of the two substances composing serum lysin which has specific action against certain invading cells. It is a sort of mordant or fixer, since its presence is necessary for lysis to occur. It has affinity both for the complement and the invading cell. Synonyms: intermediate body, intermediary body, sensitizer, copula, philocytase, desmon, preparative.

Amyloform. A white powder composed principally of starch and formalin. Used externally as an antiseptic powder and, mixed with rice powder, as a snuff for aborting or relieving coryza.

Analgesine. The proprietary name for a combination: Acetanilid 3 parts, chlorid of ammonium 1 part, citrate of caffein ½ part, sodium bicarbonate ½ part. Dose: 5 to 15 grains.

Anaplasia. The alterations in cell-character which constitute malignancy.

Anasarcin. A proprietary combination of the active principles of oxydendron arboreum, urginea scilla and sambucus canadensis. A heart tonic. Used for the treatment of dropsy.

Anesthesia, Central, Lumbar, Spinal, Sub-arachnoid a. See Cocainization, Spinal c. **Morphin-scopolamin a.** A method of general anesthesia by subcutaneous injections of morphin 0.01 gram and scopolamin hydrobromat 0.0012 gram, 4 hours, 2 hours and ½ hour before the operation. Said to be very safe and without disagreeable after-effects. **Schleich's infiltration a.** A method of obtaining local analgesia by subcutaneous injection of weak solutions of cocain. His formulæ include morphin, etc., and vary according to the amount of inflammation and hyperesthesia present at the site of the incision.

Anesthesin. Odorless and tasteless white powder. Non-irritating substitute for orthoform as local anesthetic. Said to be valuable on account of non-toxicity for injection in place of cocain.

Anesthol. A mixture for general anesthesia composed of chloroform, ether and ethyl chlorid.

Angina cruris. An affection characterized by paroxysmal pains usually in the calf of the leg, condi-

tioned by vascular changes. The same as intermittent claudication.

Vincent's a. An ulcerative, membranous inflammation of one tonsil, rarely both, accompanied by slight general disturbance.

Anguillula aceti. Vinegar worms. Occasionally found as human parasites in urine.

Ankylostoma. The same as uncinaria, q. v.

Anodynone. A proprietary name for ethyl chlorid.

Anopheles. A genus of the family Culicidae, order Diptera. The genus has 5 species in North America. They may be distinguished from Culex by the fact that while resting the body is held with proboscis, thorax and abdomen in a straight line. This genus is the sole carrier of malaria.

Antibody. A chemical substance of varying complexity formed in living organisms by the action of foreign proteid molecules upon the body cells. Antibodies exist in the fluids of the organism, and bear a specific relation to the substance under the influence of which they were formed. This relation under appropriate conditions amounts to nullification, hence the name, antibody. See Side-chain Theory.

Antidolorin. A proprietary name for ethyl chlorid.

Antiferment. A uniceptor antibody which prevents the action of ferments.

Antigermin. A preparation of copper used as disinfectant.

Antihemolysin. A substance which protects the blood corpuscles against the hemolysin.

Antilupia. A proprietary antipyretic and analgesic given in 5 to 10 grain doses.

Anti-phymin. A combination of formaldehyd, ozone, sulphur dioxid, etc., which is forced into water by high pressure. Used internally in ounce doses, and as inhalant from nebulizer. Dr. Cock's treatment for tuberculosis.

Antipuralgos. A proprietary antipyretic and analgesic coal-tar derivative.

Antipyrin camphorate. A salt of camphor used as antipyretic and for night sweats.

Antiserum. The serum of one animal (a) which has been injected with the serum of another species (b). Such serum (a) has specific precipitating powers for serum of the species (b).

Antithermoline. Proprietary plastic application used as anodyne and antiphlogistic.

Antithyreoidin. A preparation made from the serum of sheep from which the thyroid has been removed. Dose: 0.5 to 4.5 grams t. i. d. for exopthalmic goitre.

Antitoxin. A uniceptor antibody which has the power of combining with and thus rendering harmless the toxin to which it bears relation.

Antitussin. An ointment of difluordiphenyl, 5 per cent. Yellow, aromatic. Used externally by inunction into neck and chest for pertussis.

Aphasia, Puerperal a. Loss of the power of speech in pregnancy or the puerperium. Probably of central origin and caused by a variety of conditions. It is liable to recur more seriously in a subsequent pregnancy.

Aphthisin. A proprietary remedy for tuberculosis. Dose: 4 grains daily.

Appendicitis by contiguity. Inflammation of the vermiform appendix caused by infection from neighboring tissues, such as the uterin adnexa.

Appendicostomy. The operation of opening the normal appendix for the purpose of irrigating the lower bowel.

Aqua sedativa. A sedative application composed of ammonia water 2 ounces, spts. camphor 1½ drams, sodium chlorid 1 ounce, water q. s. ad. 1 pint.

Argentamin. A solution of ethylene-diamin-nitrate of silver, containing 10 per cent silver nitrate. A clear, non-caustic liquid. Used especially for gonorrhea in strength $^1/_5$ to 1 per cent.

Argyrol. A vitellin silver salt, containing 30 per cent of silver. Dark brown scales, readily soluble in water. Used for conjunctivitis, laryngitis, gonorrheal urethritis, etc., in from ½ to 25 per cent solution. Said to be non-irritating.

Arheol. Alcohol derived from oil of santal.

Aristochin or Aristoquin. Di-quinine carbonic acid ester. A white, tasteless powder, soluble in dilute hydrochloric acid, containing 96 per cent quinine salt. Dose: about the same as quinine salts.

Arrhenal. The same as disodium methylarsenate, q. v.

Aseptinol. A proprietary ointment containing resorcin, zinc oxid. etc., and a powder containing boracic acid, eucalyptol, menthol, etc.

Aspirin. Acetyl salicylic acid. White, crystalline needles. Incompatible with alkalies. Must be administered in dry form. As substitute for the salicylates is non-irritating and without after-effects. Dose: 5 to 15 grains.

Atoxyl. Meta-arsenite of anilid. White, odorless and tasteless crystals. Non-toxic form of arsenic. Daily hypodermic dose: 1 to 3 grains.

Atrabilin. A derivative of the suprarenal gland with formalin for preservation.

Atropin methylbromid. White crystals readily soluble in water. Mydriatic and antihidrotic. Dose: 2 drops one per cent solution in the eye. Internally, $^1/_{10}$ to $^1/_8$ grain. The mydriasis is said to disappear much quicker and heart to be less affected than with atropin sulphate.

Autolysis. The destruction of cells in a living body by its own serum or fluids.

Autoserotherapy. Treatment of disease by serum taken from the diseased organism, e. g. treatment of pleuritis by injections of fluid withdrawn from the pleural cavity.

Azurin. Double acetate of sodium and theobromin. Diuretic. Dose: 10 to 15 grains.

B

Bacillus oolyticus. A bacillus which acts especially on egg albumin and which is capable of causing fermentation in the stomach.

Bacteriolysin. An antibody with the power of dissolving bacteria. Specific bacteriolysins are developed in the serum under appropriate conditions.

Bana-diastase. A starch converting ferment derived from the banana.

Benzozone. The discarded name for acetozone, q. v.

Bilberry. The European huckleberry, *vaccinium myrtillus.* Said to be a valuable intestinal astringent and antiseptic.

Bigerminal. Arising from two ova. Applied to twins which have originally separate placentæ and membranes. Sometimes these may later fuse, but vascular connections are always separate. The sex may be alike or different.

Biltong. A dried meat obtained from a species of South African buck. Highly nutritive and easily digestible.

Biodal. A chemical combination of iodine, bismuth and cresol used as a dressing powder.

Biogen. Proprietary name for magnesium dioxid. A white, odorless powder, insoluble in water. Decomposed in the stomach acids, forming Mg. salts, and liberating oxygen which is said to be diffused through the system. Used in conditions,

such as anemia, where there is deficient oxidation. A diuretic, diaphoretic and laxative.

Bioplasm. A proprietary nerve tonic containing nuclein, enzymes and an alkaloid. A cream-colored powder, unstable if exposed.

Bismuth borophenate. An antiseptic dusting powder.

Blepharo-sphincterectomy. Relief of pressure of the eyelid upon the cornea by removal of some fibres of the orbicularis which pass over on the tarsus of the upper lid.

Body, Intermediary b. The same as amboceptor, q. v.

Boracil. Compound of oxy-benzoic acid, meta-dioxybenzol, acetanilid and boric acid. Antiseptic dusting powder and for making antiseptic solution.

Boro-chloretone. A dressing powder of boric acid and chloretone combined.

Borophene. A proprietary antiseptic and astringent powder.

Brometone [Bromoform + acetone]. A white crystalline substance, camphoraceous odor and taste. Soluble in alcohol, ether, etc., sparingly in water. Antiseptic and sedative. Used for headache, cough in chronic cases, gastric fermentation. Dose: 5 grains, 4 or 5 times a day, best in capsule.

Brominol. The same as bromipin, q. v.

Bromipin. An organic compound of bromid and oil of sesame containing 10 per cent bromin. Passes stomach unchanged. Dose: one to three teaspoonfuls daily.

Bromocoll. A combination of tannin and gelatin with 20 per cent bromin. Yellow, odorless and tasteless powder, split up in alkaline fluids. A non-irritating substitute for potassium bromid. Dose: 15 to 75 grains a day. Used in ointment form for pruritus.

Bromofarina. A mixture of bromides with flour.

Bromopan. A bread containing bromides, 15 grains to the loaf.

Bronchitis obliterans. A variety of bronchitis in which fibrinous exudate in the form of nodules fills the smaller bronchi. Clinically and at autopsy there is resemblance to miliary tuberculosis.

Bufonin, Bufotalin. The active principle of the secretion from skin of lizards. Physiologic action similar to digitalis.

C

Cacodyl (kak-o-dil) [Gr. kakodes, ill-smelling]. An organic preparation of arsenic, $As_2(CH_3)_4$. In various chemical combinations used as substitute for non-organic preparations of arsenic. **Cacodylate of iron** (ferricodyle or marsyle). Dose: $^1/_6$ grain. **Cacodylate of sodium,** 48 per cent arsenic. Dose: $^1/_6$ to ½ grain. **Cacodylate of magnesium.** Dose: 15 to 40 minims of 10 per cent solution.

Calcusol [Calculus + solvent]. Proprietary preparation of piperidine parasulphamine benzoate and potassium carbonate. Used for gout and some forms of rheumatism.

Camenthol. Colorless fluid formed by the union of refined camphor and Japanese menthol. Used in 3 per cent solution in atomizer for inhalation in diseases of respiratory organs.

Camphacol. A condensation product of camphoric acid, formaldehyd and guaiacol. Used in phthisis, other respiratory ailments and cystitis. Dose: 5 to 20 grains several times a day.

Camphoric acid. Obtained by action of nitric acid on camphor. Odorless, white, scaly, soluble crystals. For night sweats of phthisis. Dose: 15 to 24 grains.

Carcinoma, Epibulbar c. Cancer which begins, resembling phlycte-

nular ulcer, at the margin of the cornea where it joins the sclera, and spreads over the cornea and conjunctiva. Two varieties: luxurians and planum.

Cascarenna. A combination of cascara sagrada, senna, potassium and sodium tartrate, etc., especially adapted as a laxative for children.

Cassia bearcana. An African remedy for black-water and bilious fever.

Casumen. A proteid food made from casein.

Catheter a demeure [Fr. a demeur, stationary]. A catheter held fixed in the urethra.

Celiohysterotomy. Opening the uterus through an abdominal incision, Cesarean section. Distinguished from celiohysterectomy which is excision of the uterus by the same route, the Porro operation.

Cell, Nussbaum's c's. Small functionless cells of the pyloric glands of the stomach, the analogue of the acid cells of the glands of the fundus.

Cerevisine. Dessicated yeast.

Chaparrin. A proprietary combination of the active principles of *chaparro amargoso* with salicylic acid, phenol and camphor. Used for parasitic skin affections.

Chielen. Extract of tulip. Recommended for skin diseases.

Chinin lygosinate. A salt formed by the union of quinin and lygosin. A yellow powder, insoluble in water. An antiseptic dusting powder.

Chinotropin. Quinate of urotropin. Uric acid solvent and urinary disinfectant. Dose: 30 to 60 grains daily.

Chlor-anodyne. An antispasmodic and anodyne preparation containing morphine, chloroform, etc. Dose: 15 minims.

Chloroform - Anschuetz. Chloroform from which the impurities have been eliminated by combining it with salicylide to form crystals and afterwards distilling off the chloroform. Said to produce less unpleasant effects in narcosis.

Chloroma. Lympho-sarcoma of a peculiar green color, probably derived from modified hemoglobin, which runs its course with clinical symptoms of leukemia or pseudo-leukemia.

Chologen. A combination of mercury with podophyllin, melissa, camphor and caraway. Used in varying combinations for the treatment of gall-stones.

Chondroform. A proprietary antiseptic lubricant composed of chondrus (Irish moss) and antiseptics.

Chorditis cantorum. Inflammation of the vocal cords occurring in singers.

Chorea gravidarum. A rare disease of pregnancy. Symptoms same as chorea of childhood with greater mental disturbance.

Chromium sulphate. Green, amorphous, metallic tasting, soluble salt. Recommended for various neuroses. Dose: 1 to 4 grains.

Chromoradiometer. Holzknecht's instrument for measuring X-ray dosage by means of color changes produced in test slides which are placed next the skin exposed to the rays. A color-scale indicates degrees of ray absorption.

Chymosin. The ferment from gastric juice which coagulates casein. The same as rennin, lab, lab-ferment.

Chymosinogen. The compound in the gastric juice from which chymosin is formed.

Ciliectomy. Removal from the eyelids of the tissue which contains the roots of the eyelashes.

Cinnamylquinin hydrochlorate. A salt of cinnamene and quinin used as antipyretic.

C. M. Chirurgiæ magister. Master in surgery.

Cirrhosis, Biliary c. Cirrhosis in which the proliferation of connective tissue, characteristic of cir-

rhosis, follow upon lesions of the bile-ducts. Includes Hanot's and obstructive biliary cirrhosis. **Capsular c.** The form of cirrhosis following upon lesions of Glisson's capsule, especially chronic perihepatitis. Also called Glissonian cirrhosis, lymphatic cirrhosis. **Fatty c.** Hypertrophic alcoholic cirrhosis, like Laennec's, only remaining large. **Hanot's c.** Hypertrophic cirrhosis. A form of biliary cirrhosis in which there is proliferation of the bile ducts, enlargement of the liver, perhaps only very slight jaundice, and rarely ascites. Sometimes called Charcot's cirrhosis. **Laennec's c.** Atrophic cirrhosis resulting from obstruction to the portal circulation. Also called portal cirrhosis, alcoholic cirrhosis, hob-nail liver. The atrophic stage may be preceded by pseudo-hypertrophy. **Obstructive biliary c.** A form of biliary cirrhosis caused by chronic retention of the bile; characterized by early deep jaundice and enlargement of the liver. **Stasis c.** The form of cirrhosis arising from obstruction to the outflow of the hepatic vein. Also known as cyanotic induration of the liver, cardiac liver. **Vascular c.** The general term for cirrhoses following upon obstruction to the hepatic vein, the portal vein, or the general hepatic circulation as in arteriosclerosis.

Clausena anisata. An African plant used by the natives as a remedy in fevers.

Coagulin. A uniceptor antibody which has the power of coagulating the substance under the influence of which it was formed.

Cocainization, Spinal c. Sub-dural injection of 8 to 20 minims of 2 per cent sterile solution of cocain. Injected in fourth or fifth lumbar interspace. Produces analgesia extending usually as high as the thorax. Ordinarily the tactile, muscular and temperature sensibility is retained, so the terms anesthesia and narcosis are incorrect as applied to this procedure.

Colasaya. A tonic preparation containing cola, calisaya bark, coca, iron, phosphates, etc.

Colica scortorum. Occasional acute colicky pains felt in the region of the Fallopian tubes. Occurs in some cases of salpingitis, but for the pain itself there is no satisfactory explanation. So named on account of frequency among prostitutes.

Commotio retinae. Concussion of the retina from a blow on the head. Opaque gray patches appear on the retina and vision is reduced.

Complement. A thermolabile ferment-like substance normally present in cell protoplasm and found in many serums. It is the non-specific body in serum lysin. Synonyms: alexin, cytase, end-body.

Complementoid. A complement which has lost its affinity, its haptophores, for receptors in cell molecules or in amboceptor molecules.

Constipation, Spastic c. A form of constipation found in neurasthenics, and characterized by a palpably constricted section of intestine. Defacation is difficult, the intestinal spasm is painful. The feces are hard, dry and of small caliber.

Contractio praevia. A contraction of the lower segment of the uterus in front of the fetal presenting part. In contrast to Bandl's contraction ring, contractio praevia is a lack of expansion in the entire lower segment, thus including, of course, all of the cervix. Generally it disappears with the escape of the amniotic fluid, if not it may remain under the influence of labor pains as a spastic stricture of the lower segment, leading to prolonged labor and perhaps to dangerous tears.

Contracture, Volkman's c. A traumatic deformity of the forearm and

hand. Pronation and flexion of the hand, forearm muscles hard and shrunken. Exact cause unknown.

Cruria. Quinoline - bismuth - sulpho - cyanate. Yellowish, pungent, insoluble powder. Stimulating dusting powder for ulcers.

Cryoscopy [Gr. kryos, cold]. Study of the freezing point of various solutions, particularly of the blood and urine. The freezing point varies according to the number of molecules dissolved in the solution.

Crystallose. A synthetic sweetening agent said to be 500 times sweeter than cane sugar. Recommended in diabetes, corpulence and fermentative dyspepsia.

Culex. A genus of the family Culicidae, order Diptera. The genus has 3 species in North America. They may be distinguished from anopheles by their peculiar humpbacked appearance while resting. This genus is only known to transmit to man the embryo of filaria.

Culicidae. A family of insects of the order Diptera, comprising mosquitoes. The genera of peculiar medical interest are Anopheles, Stegomyia, Culex.

Cypridol. Proprietary name for a one per cent solution of mercuric iodid in an aseptic oil. Given internally in capsules containing $^1/_{32}$ grain, or hypodermatically, 8 minims equalling $^1/_{12}$ grain, for syphilis.

Cystogen. A proprietary name for the urinary antiseptic, hexamethylen-tetramin, $C_6H_{12}N_4$. Dose: 5 grains 3 or 4 times daily.

Cyst, Paranephric c. A cyst of the perirenal cellulo-fatty tissue, of congenital or unknown origin. It may have an opening into the pelvis of the kidney or into the ureter.

Cytodiagnosis. Diagnosis by means of study of the cells found in a specimen obtained from the patient. Particularly applied to study of withdrawn pleuritic effusion.

Cytolysin. An antibody which causes dissolution of cells, particularly invading micro-organisms. Those cytolysins with specific action for certain organs or cells are named accordingly, e. g., spermatolysin, hemolysin, etc.

Cytoryctes vaccinia. A protozoan, class sporozoa, about one micron in diameter, the intracellular or asexual cycle of which causes vaccinia, the intranuclear or sexual cycle causing small-pox.

Cytotoxin. An antibody which injures cells, not dissolving them (see cytolysin). Cytotoxins with specific action for certain organs are named accordingly, thus, nephrotoxin, etc.

D

Decapsulation. Stripping or removal of the capsule. **Renal d.,** an operation performed for chronic nephritis.

Decortication, Renal d. Removal of the proper capsule of the kidney, an operation lately recommended for some cases of nephritis.

Dementia præcox [L. præcox, premature]. That form of degenerative psychosis in which dementia occurs independently of any other form of insanity. It includes the ordinary primary dementia, hebephrenia and katatonia.

Dermogen. The name given to zinc peroxid. A yellowish, odorless powder, insoluble in water. Mixed with tartaric acid and water it liberates hydrogen dioxid, and is thus used externally as an antiseptic and stimulating application.

Diastasis recti abdominis. Separation of the abdominal recti away from the median line. A not uncommon disorder after pregnancy. Operative repair often effective.

Diastin. The proprietary name of a vegetable digestive preparation.

Diathesin. Formaldehyd phenol. Fine

leaflets, bitter taste, soluble in water. Anodyne, antipyretic. Supersedes the salicylates, no by effects. Dose: 7½ to 15 grains.

Dichotomy. A division into two parts. Applied to the division of fees between physician and surgeon.

Diosmal. An extract of buchu leaves. Used in affections of the urinary organs. Dose: 2 grains t. i. d.

Dioxogen. The proprietary name for a 3 per cent solution of hydrogen peroxid.

Diplacusis binauralis dysharmonica. Different pitch of the same sound as heard by the two ears. **D. b. echotica.** A sound is heard later and weaker in one ear. **D. monauralis.** A sound heard as two sounds by one ear, the other ear being closed.

Diplococcus of Class. A species of usually large size, but with varying morphological characteristics, obtained from the throat and said to be the cause of scarlet fever.

Discission. A cutting into two parts. Particularly applied to an operation for relief of stenosis of the cervix uteri. The cervix is divided on each side down to the vaginal attachment, wedge-shaped pieces excised, and the cut surfaces sutured.

Disease, Banti's d. An affection in which for some years there is anemia with splenic enlargement finally followed by ascites and a tendency to hemorrhages. At first differentiation from splenic anemia cannot be made. **Blue d.** A local name for Rocky mountain spotted fever, q. v. **Duke's d.** Rubella scarlatinosa, q. v. **Fourth d.** Rubella scarlatinosa, q. v. **Hookworm d.** Uncinariasis, q. v. **Reichmann's d.** The continuous secretion, even in the fasting stomach, of gastric juice. **Stokes-Adams d.** A chronic disease characterized by slow pulse, paroxysms of vertigo and epileptiform or apoplectiform attacks. **White-spot d.** A degeneration of the skin limited to the papillary body and upper portion of the reticular layer. The snow-white spots are arranged like beads on the chest.

Disodium methylarsenate. An organic compound of arsenic. $AsCH_3O_3Na_2$. Recommended as a substitute for arsenic and the cacodylates. Dose: 1 to 3 grains a day. The same as stenosin, arrhenal and neo-arsycodyl.

Diuraxin. A powder containing theobromine 36 per cent, salicylic acid 55 per cent, and formaldehyd 6 per cent. Soluble only in the alkaline secretions. An antiseptic, stimulating diuretic. Dose: 6 grains in capsule every 2 hours.

Dormiol. Dimethyl - ethylcarbinol - chloral or amylene chlorid. A rapidly acting only slightly toxic hypnotic. Dose: 1 to 4 drams of the 10 per cent solution.

Drain, Mikulicz d. A single layer of gauze with a number of thick wicks of gauze packed into its cavity as it is pushed down middle first into the wound cavity.

Dymal. Dydimium salicylate. Siccative, antiseptic, odorless powder. Used for skin affections, eczema, herpes, hyperidrosis, etc.

Dyspragia [Gr. prasso, to do]. Painful functioning. **D. intermittens angiosclerotica intestinalis.** A painful disease of the intestines corresponding to intermittent claudication.

Dystrypsia. Faulty intestinal or pancreatic digestion, in contradistinction to dyspepsia, faulty gastric digestion.

E

Eidoptometry [Gr. eidos, form]. Measurement of the acuteness of the visual perception of form.

Electron. An ultra-atomic corpuscle the presence of which on an ion gives its difference of character from a neutral atom.

Elkossan. An astringent preparation from the seeds of *Brucea sumatrana.*

Endometritis, Bacteriotoxic e. A form of inflammation of the endometrium caused by toxins of bacteria which inhabit the uterine secretions, in contradistinction to infectious endometritis which is caused by bacteria actually present in the tissues themselves.

Energin. A food product prepared from the proteid of rice.

Enterococcus. A capsulated streptococcus isolated from cases of dysentery.

Enterokinase. An enzyme secreted by the intestinal mucosa, having the power of transforming the inert proteolytic ferment, trypsinogen, of the pancreatic juice into the active digestive agent, trypsin.

Enzymol. A concentrated extract of the gastric juice, used as an antiseptic and sedative dressing for infected wounds and lesions.

Eosolates. Salts of creosote esters containing 25 per cent creosote. **E. of calcium.** Soluble gray powder. Dose: 5 to 10 grains, for phthisis and diabetes. **E. of silver.** Powder. Used in gonorrhea.

Epibulbar. A term applied to tumors upon the eyeball.

Epicarin. Reddish powder, acidulous odor, dissolving readily in alcohol, ether, vaseline, etc. A condensation product of creosotinic acid and naphthol. Used as an ointment for scabies and prurigo.

Epinephrin. The active principle of the suprarenal gland isolated according to a special method.

Epiosin. An organic base obtained from morphigenin, analgesic and hypnotic. Dose: 1½ to 2 grains.

Epitheliolysin. An antibody which causes the destruction of epithelial cells.

Erotopath. A person with mind disordered on the subject of love.

Erythroderma, Congenital ichthyosiform e. Generalized redness of the skin with papillary hypertrophy about neck and joints. Dry and bullous forms.

Eubiose. Hematogen carbonated and free from glycerin.

Euphorbia heterodoxa. A Brazilian plant used locally in cancerous affections and ulcerous lesions.

Euphrasia officinalis. A plant, "eyebright," of Northern U. S. Recommended for catarrhal conditions of eyes and respiratory mucous membranes. Dose: 10 to 60 drops of fluid extract.

Euquinin. Quinin carbonic ether. White needles, tasteless, sparingly soluble in water, readily in alcohol. Used for same purposes as quinin and in same doses.

Europhen. Di-iso-butyl-cresol-iodid. An odorless substitute for iodoform.

Eutrepisty [Gr. eutrepes, prepared well]. The giving of remedies, such as potassium iodid, before an operation in order to lessen the dangers of septic infection.

Eutrophic [Gr. eutrophia, healthy, nourishing]. Causing healthy nutrition.

Extract, Jex's e. An extract made from the organs of animals injected 2 or 3 days previously with typhoid bacilli. Given by the spoonful every 2 hours, or subcutaneously injected.

F

Fang'hi di sclafani. Sulphuraceous volcanic earth used in acne rosacea.

Felsin. A proprietary combination of fel bovis, pepsin, pancreatin, and oleo-resin capsicum. Recommended as a carminative, digestive and hepatic stimulant.

Fergon. An organic iron compound.

Ferissol. A preparation of cinnamic

acid and guaiacol. **Used for tuberculosis. Dose: 15 to 45 grains** daily; intramuscular injections of 15 to 45 minims of 10 per cent solution.

Ferratogen. Organic iron made by growing yeast in a ferruginous medium, Yellowish powder, insoluble in water. Dose: 5 grains.

Ferro-phosphata. A proprietary preparation of tincture of chlorid of iron, 5 minims, and sodium phosphate, 40 grains, to the fluid dram.

Fever, Black f. A local name for Rocky mountain spotted fever, q. v. **Pretoria f.** A fever observed in South Africa, lasting from 5 to 12 days and accompanied by abdominal symptoms. Considered by some to be abortive typhoid. **Spotted f.** A name applied to typhus and cerebro-spinal meningitis, but designating more particularly an infectious disease peculiar to the Rocky mountain regions of the northwest. It is marked by continuous high fever, mental symptoms, and spotted red macular eruption which may become confluent and turn blue and dark.

Fibrinoscopy. The same as inoscopy, q. v.

Firolyptol. A proprietary restorative tonic containing eucalyptol, cotton seed oil, and firwein.

Firwein. A proprietary respiratory tonic prepared from the bark of the fir tree with iodin, bromin and phosphorus.

Fistula cervico-vaginalis laqueatica [L. laquear, a paneled ceiling]. An abnormal opening connecting the uterine canal with the vagina in the vaginal portion of the cervix. The result of disease or trauma.

Fluoroformol. Watery solution of the gas CHF_3 (2.8 per cent). Tasteless, odorless. Antiseptic, alterant. **Used in incipient phthisis, lupus, pneumonia. Dose: 1 tablespoonful.**

Fluor-rheumin. Ointment of fluorphenetol (5 per cent). Anodyne for sciatica, rheumatism, etc.

Folie du doute (fo-le du doot). Doubting insanity. Hesitation over the doing of acts which may be repeated many times to make them right.

Folliculoma. A tumor of the ovary derived from the epithelium of Graafian follicles. It contains alveoli filled with epithelium in which structures like mature follicles are included.

Formacetone. A proprietary disinfectant.

Formaldene. A solution of formic aldehyd gas, 40 per cent. The same as formalin.

Formasal. A chemical combination of formaldehyd and salicylic acid which may be chemically combined with various metallic and alkaline salts. These have the therapeutic properties of their various constituents and are designated by abbreviations of their constituents, such as caf-forma-sal (caffein + formasal), bis-forma-sal (bismuth + formasal), etc.

Formaseptol. A proprietary liquid disinfectant composed of ½ per cent formaldehyd with thymol, benzoic acid, etc.

Formula, Dreser's f. A formula giving exact expression to the work performed by the kidney by comparing in graminutors the molecular concentration of the blood with that of the urine.

Frosted. A term applied to the liver, heart, etc., in imitation of the German "Zuckergussleber" or frosted liver where there is thickening of the hepatic or splenic peritoneum or of the pericardium, and which resembles the frosting on cake. See hyaloserositis.

G

Gabianol. An oily liquid obtained from shale, used in pulmonary diseases. Dose: 4 minims several times a day.

Gallogen. A derivative of gallic acid, used as intestinal astringent. Dose: 5 to 30 grains.

Gasterine. Gastric juice of the dog. Used in France for hypochloridia.

Gastroptosia. Downward displacement of the stomach. A word proposed as being more correct etymologically than gastroptosis. The same correction applies to nephroptosia, for nephroptosis, nephropexia, for nephropexis, etc.

Gastrosia fungosa. The growth of mold in the stomach.

Gelone. A preparation of gelatin, etc., used in making bandages.

Geosot. Ester of guaiacol with valeric acid. Oily fluid, not soluble, sweetish odor. Intestinal antiseptic and antitubercular. Dose: 2 to 4 grams a day, best in capsules.

Germiletum. An alkaline antiseptic solution.

Glands, Hemolymph g's. Glands with blood sinuses found in contiguity with lymph glands, distinguished from the latter by appearing redder and smaller. Function is probably the formation of leucocytes and destruction of erythrocytes.

Glutannol. A preparation of vegetable fibrin and tannic acid used as intestinal astringent. Dose: Adults, 15 to 30 grains; children, 5 to 10 grains.

Gluton. A gelatin food product.

Glyceroplasma. A proprietary putty-like antiphlogistic preparation composed of glycerin, a mineral silicate, and antiseptics. Used externally to deplete the tissues.

Glycosal. The ester of glycerin-salicylic-acid. Used as substitute for salicylic acid. Dose: 45 to 90 grains daily.

Glykaolin. A proprietary name for a preparation of aluminum silicate, glycerin and antiseptics. Used externally as antiphlogistic and anodyne substitute for poultices.

Gorit. Peroxid of calcium. Used as gastro-intestinal disinfectant.

Guaiacamphol. Camphoric acid ester of guaiacol. Tasteless, insoluble white powder. Anhidrotic in phthisis. Dose: 3 to 15 grains, an hour before bedtime.

Guaiacophosphal. A proprietary name for neutral phosphite of guaiacol. Contains 92 per cent of guaiacol, 7 per cent of phosphorus.

Guaialin. An odorless and almost tasteless greenish powder containing 60 per cent of guaiacol, 30 per cent benzoin and 7 per cent formaldehyd. Antipyretic, antitubercular, diuretic, antiseptic. Dose: 60 to 100 grains daily in divided doses.

Gynecine. A proprietary remedy for the treatment of dysmenorrhea.

H

Hagiotherapy [Gr. hagio, holy]. Healing by a miracle or by a holy man.

Haptophore [Gr. hapto, to fasten, phora, a carrying]. An atomic group of a molecular constituent of a toxin or food which group exhibits affinity for other atomic groups, receptors, present in cell molecules.

botomy. The same as pubiotomy. Severing of the os pubis lateral to the median line for obstetrical purposes. Used as a substitute for symphyseotomy on account of better union of the pelvis.

Helmitol. Methylene citronate of urotropin. White powder, decomposed in an alkaline solution. Dose: 15 grains. A urinary antiseptic.

Helthin. Amidonaphthol-K-acid. A

reagent originated by Erdmann for testing the purity of water. The reaction depends upon the presence of the nitrous acid or nitrites which accompany any organic substance of detrimental character.

Hemaboloids. A combination of organic iron compounds with bone marrow, beef peptones and nuclein. Tablespoonful doses.

Heminal. A soluble powder containing iron albuminate and salts from fresh blood. Dose: 8 to 16 grains.

Hemolysin. An antibody having the power to dissolve erythrocytes.

Hemoquinine. A proprietary combination of iron, manganese, arsenic and quinin. Used especially in malarial cachexia and chlorosis.

Hemosozic [Gr. sozo, to preserve]. Antihemolytic. Preventing dissolution of red blood corpuscles.

Hermophenyl. Mercuric-phenol-disulfonate of sodium. White, soluble powder. Antiseptic in solution of $^1/_{10}$ to 1 per cent.

Heromal. A proprietary combination of malt extract and heroin.

Heroterpine. A proprietary elixir combining heroin and terpin hydrate.

Heterochylia. The condition where there is a marked change in the chemical character of the stomach contents when taken under the same conditions on successive occasions.

Heterolysis. The destruction of cells of an animal by natural or immunized serum from another species.

Hetoform. Bismuth cinnamate. A remedy introduced for the treatment of tuberculosis.

Hetol. Sodium cinnamate. Injected intravenously, hypodermatically or given internally for phthisis. Dose for injection: 0.05 gram; internally, 0.5 to 1.5 gram.

Histogenal. A preparation of nucleinic acid and sodium methylarsenate used as a remedy in tuberculosis.

Hook-worm. Uncinaria, q. v.

Hopogan. Another name for biogen, q. v.

Horismascope. An instrument for testing for albumin in urine.

Hyaloserositis, Multiple progressive h. A rare chronic inflammation of the serous membranes with overgrowth of fibrous tissue and hyaline changes. The same as "frosted liver," etc. See frosted.

Hydrargyrum oxycyanatum. Said to be of much use and practically non-irritating in urethritis. In acute cases used in solutions of from 1:3000 to 1 per cent.

Hydroa gestationis. A variety of dermatitis herpetiformis appearing in the puerperium or during pregnancy.

Hydrocerin. A preparation of wax, petrolatum and water used as an ointment base.

Hydroperinephrosis. A collection of fluid of traumatic origin in the retroperitoneal connective tissue and opening into the pelvis of the kidney.

Hydrops tubae profluens. A variety of salpingitis in which there is recurrent retention and discharge through the tube of fluid inflammatory products.

Hyperkeratosis lacunaris. White hard excrescences firmly attached within the tonsillar crypts.

Hypertrophy, Marie's h. A chronic enlargement of the joints resultant upon chronic periostitis. Most of the swelling consists of the soft parts, the bones being only moderately enlarged. Amenable to treatment.

Hypnopyrin. A quinin derivative. Antipyretic, analgesic. Dose: 4 grains.

Hyrgolum. Metallic mercury in an allotropic form soluble in water. Small, shining black crystals. Given internally and by inunction for syphilis.

Hysterocervicotomy. The so-called vaginal Cesarean section. Incision through the anterior or posterior

or both walls of the cervix and lower uterine segment to gain space for delivery of the ovum in cases where through pathologic condition or lack of time the cervical canal cannot be dilated by the usual methods.

Hysterokataphraxis [Gr. kata-phrasso, to fence in]. Supporting the uterus by metallic ligatures which, following the method of Catterina, are carried around the uterus, through the abdominal walls, and are tied just beneath the skin.

I

Iboga. The plant abona. Used as tonic and aphrodisiac.

Ibogain. An alkaloid from iboga. Hypnotic.

Ichthargan. A combination of ichthyol and silver, containing 30 per cent of the latter. Used in the strength of 5 per cent as solution or bougie for gonorrhoeal endometritis, etc.

Ichthosot. A combination of ichthyol and creosote. Antitubercular.

Ichthyodin. The same as isarol, q. v.

Ichthyoform. Product of action of formaldehyd on ichthyol. Brown, tasteless, insoluble powder. Given in gradually increasing doses for phthisis.

Ichthyosis uteri. A condition in which there is transition of the columnar epithelium of the endometrium into stratified epithelium which remains superficial.

Icterus, Acholuric i. Jaundice in which no bile pigment is found in the urine. **Xanthochromic i.** A variety of icterus in which there is yellow coloration of the palms and soles and slight coloring of the mucous membranes, but without trace of biliary pigment in the urine.

Igazol. Compound of formalin with an iodin body. Tried by inhalation for phthisis.

Immunization. The conferring on an organism of protection against foreign proteids bodies by treatment of the serum. Small doses of the foreign material are generally given repeatedly, the organism reacting to it by the production of antibodies.

Inoscopy [Gr. inos, of fibre]. Jousset's method of diagnosing bacterial diseases by artificial digestion of the fibrinous matter in sputum, blood or effusion. Especially adapted to tuberculous affections.

Insufficiency, Hepatic i. Inability of the liver to perform its functions, especially its anti-toxic function, resulting in various systemic infections and hepatic lesions. The condition may be congenital or acquired later in various ways. **Uterine i.** Atony of the uterine musculature and consequent weak contractile power, causing venous stasis and a train of symptoms.

Iodia. A proprietary combination of various herbs with potassium iodid and phosphate of iron, of which there are 5 and 3 grains to the dram, respectively.

Iodipin. A combination of iodin and sesame oil, in strengths of iodin 10 and 25 per cent. Does not produce iodism. Dose: 15 to 60 minims internally. Subcutaneously up to one dram for tertiary syphilis. Also used for asthma, bronchitis, etc.

Iodocacodylate of mercury. A compound of arsenic, iodin and mercury used internally for syphilis.

Iodoferratin. A preparation of iodin and ferratin (blood-iron). Used as a general hematogenic.

Iodoferratose. The syrup of iodoferratin. Dose: 3 or 4 tablespoonfuls a day.

Iodoformogen. Iodoform albuminate. A yellowish, nearly odorless powder. Insoluble in water. A substitute for iodoform.

Iodolin. A preparation of iodol and albumin. Used in place of iodids and iodoform. Internally, dose: 30 grains.

Iodomuth. An odorless and tasteless bismuth powder containing 25 per cent iodin, 5 per cent formaldehyd. A nonirritating antiseptic, desiccant and deodorant. Used externally, and internally for gastro-intestinal ailments. Dose: 5 to 90 grains.

Iodo-nucleoid. A reddish brown granular powder containing 9½ per cent iodin in organic combination with nuclein. Insoluble in acids, alcohol, ether; slowly soluble in alkaline fluids. Antisyphilitic, nontoxic. May be given in very large doses without producing iodism.

Iodophilia. The staining brown of particles in leucocytes by the use of iodin in potassium iodid solutions. This occurs in a considerable number of clinical conditions, but never in normal leucocytes and only when leucocytosis is present.

Iodosyl. An amorphous, garnet colored, odorless, non toxic, analgesic and antiseptic powder containing 65 per cent of iodine. Used as powder or in ointment.

Iodyloform. A compound of iodin with a gelatin preparation. A substitute for iodoform.

Ion. An atom or group of atoms accompanied by an electron, that is, charged by electricity, the combination being obtained by electrical dissociation of molecules.

Isarol. The same as ichthyodin. The ammonium salt of sulphonic acid from an insoluble sulphur-bearing mineral oil. A dark brown viscous fluid. Properties the same as ichthyol; astringent, siccative, antizymotic.

Islands of Langerhans. Irregular masses in the pancreas, composed of smaller cells than the ordinary secreting pancreatic cells. These islands control in some measure carbohydrate metabolism, and their degeneration is one cause of diabetes.

Isolysis. The destruction of cells of an animal by a natural or immunized serum from another member of the same species.

Ixal. An oil derived from the distillation of coke. Disinfectant and antiseptic. For phthisis used internally or by inhalation. Intestinal antiseptic. Dose: 15 minims a day.

J

Jennerization. The method of producing immunity to a disease by repeatedly injecting cultures of bacteria similar to those producing the disease, but without their virulence. The term introduced by Behring to designate his method of immunizing cattle to bovine tuberculosis by injections of cultures of human tubercle bacilli.

Jequiritol. A preparation of definite strength from *abrus precatorius*. Used in one minim doses of 4 strengths as substitute for jequirity in treatment of pannus and trachoma.

K

Karnoid. Meat powder without preservatives, made by an English process.

Karyolysis. Disorganization of the nucleus of a cell.

Kataphraxis. The fencing or caging

in of an organ with metallic supports to keep it in place. A method introduced by Catterina and used for the displaced kidney and uterus. See hysterokataphraxis.

Katharmon. A proprietary antiseptic and astringent solution containing hydrastis, hamamelis, boric acid, etc.

Keimol. A proprietary antiseptic prepared as powder and as soap.

Kinescope. An instrument for determination of the ocular refraction by observation of a fixed object through a slit in a moving disk.

Koilonychia [Gr. koile, a hollow]. Spoon-nail. The concave appearance assumed sometimes by the nail plate. The condition has various causes.

Kreso. A disinfectant coal-tar product consisting largely of cresols and higher phenols. Used to disinfect bowel discharges, to sterilize instruments, etc., in dilution 1 to 3 per cent.

L

Lanikol. A proprietary ointment containing acetanalid, salicylic acid, aristol, ichthyol, lanolin, bismuth, zinc oxid.

Lathyrism (lath-ir-izm). A disease affecting chiefly the nervous system, manifested by paralysis and other nervous disturbances, caused by poisoning from ingestion of different species of the genus *lathyrus*. *L. sativus*, of teora, is a food grain largely eaten in India. Poisoning results from improper cooking of it.

Lavoline. A colorless, odorless oil from petroleum.

Law, Wolff's l. Every change in the form and function of the bones is followed by certain definite changes in their internal architecture and external conformation. Wolff denies that pressure produces atrophy of bone.

Leucolysin. An antibody which dissolves leucocytes.

Leucotoxin. An antibody which injures white blood corpuscles.

Ligamentum infundibulo-pelvicum. The superior and free border of the broad ligament, which reinforced by fibrous tissue carries the ovarian vessels from the lateral pelvic wall to the ovary. Also called the suspensory ligament of the ovary.

L. interuretericum. The ridge of tissue seen by cystoscopy running laterally and projecting just back of the trigone and between the ureteral openings.

Linea nigra. The narrow band of pigmentation which develops during pregnancy in the skin on the median line between the pubes and the ensiform cartilage.

Linitis plastica. Inflammation of the connective tissue surrounding the vessels of the stomach resulting in hypertrophy of the connective tissue, a gastric sclerosis.

Lipiodol. The same as iodipin, q. v.

Lipolysis. Dissolution of fat.

Lithabol. The proprietary name for a combination of the soluble double salts of lithium and sodium nitrites, borates and benzoates.

Lithiasis, Pancreatic l. The condition in which concretions, mostly of phosphate and carbonate of calcium, exist in the pancreas. Symptoms: colicky pain, passage of stones, fat diarrhea, emaciation, diabetes.

Lithona. A combination of salts of lithia with mild saline laxatives. Dose: 1 dram.

Lymphoids. A proprietary preparation of testicular extract, phosphide of zinc, nux vomica, iron, aloin, bovis pulv.

Lysin. An antibody which has the power of causing dissolution of cells, bacterial or other. It is composed of two definite and sep-

arable chemical proteid substances, complement and amboceptor.

Lysulfol. Thick black liquid combination of lysol and sulphur. Used in skin diseases.

M

Maizo-lithium. A proprietary name for a solution of maizenate lithium, which is the chemic union of maizenic acid, from green corn silk, with lithium. Genito-urinary sedative. Dose: 1 to 2 drams.

Maltsanta. A combination of maltzyme and extract of yerba santa concentrated to a confection. Used freely as a tonic and sedative to the respiratory mucous membranes.

Mangasol. Chloro-phenol-sulphonate-manganese. A brown, odorless, tasteless powder containing 10 per cent permanganate of potassium. Antiseptic. Internally dose 10 to 30 grains.

Manoia. A proprietary tonic said to contain the active principle of cod liver oil, china officinalis, coca, extract of mano nut and phosphates.

Marsyle. Cacodylate of protoxide of iron. A combination of an organic form of arsenic with iron.

Mediglycin. Fluid glycerin soap used as vehicle for dermic remedies.

Melachol. A laxative combination of phosphates with nitrates of sodium.

Membrane, Cargile m. Sterilized peritoneum of the ox, used in surgery for covering surfaces denuded of peritoneum in order to prevent adhesions forming.

Mercurivanillin. Antisyphilitic remedy containing 40 per cent of mercury.

Mesometrium. The uterine structure lying between the endometrium and perimetrium. A better term than myometrium because this structure often consists more of connective tissue than muscle fibers.

Mesotan. An ester of salicyl. Used in the local treatment of rheumatic affections and gout. An almost odorless and easily absorbed substitute for oil of gaultheria. Mixed with olive oil, lightly rubbed in t. i. d.

Meta-icteric [Gr. meta, after]. Following after jaundice, a variety of cirrhosis and of splenomegaly.

Metaplasia. The direct transformation of one tissue into another without the formation of an intermediate embryonal tissue, as when connective tissue becomes myxomatous tissue.

Metasol. Trade name for 1 and 2 per cent solutions of metacresol anytol, which in turn is a 40 per cent solution of metacresol in anytin. Metasol is a germicide indifferent to instruments and does not lose its powers in the presence of albuminoids.

Methylbenzaconine. Derivative of aconitine. Action upon motor nerves resembles curare.

Methylene diguaiacol. Other names: Pulmoform, Guaiaform, Geoform. Used as other guaiacol preparations.

Metranoikter. An instrument for dilating the cervix. Consists of halves joined by a strong spring which is released by special introduction forceps.

Metreurynter. An inflatable bag for dilating the lower segment of the uterus and inducing uterine contractions. The bag is aseptically introduced empty into the uterus and blown up through the connecting tube.

Metreurysis. The method of dilating the cervix and starting uterine contractions by use of the metreurynter.

Micrazotol. An antiseptic solution containing boric acid, eucalyptol, resorcin, menthol, etc.

Micrococcus neoformans. A coccus forming chains of 6 or 8, difficult to cultivate, isolated from various tumors by Doyen and suggested by him as their cause.

Mittelschmerz [Ger. middle-pain]. Periodic intermenstrual pain of undetermined origin resembling closely the pain of obstructive dysmenorrhea.

Mixture, Bonain's m. Equal parts of menthol, carbolic acid and cocain used for anesthetizing the membrana tympani before doing paracentesis. **Coley's m.** A mixture of toxins, q. v.

Monogerminal. Arising from one ovum. Applied to any two fetuses in a multiple pregnancy which occupy a single chorionic sac and are nourished by one placenta. They are always of the same sex.

Morrhuvin. A proprietary mixture of wine of cod liver oil and peptonate of iron.

Mosquitoes. See Culicidae.

Myasthenia, Angiosclerotic m. Abnormal muscular fatigue conditioned by local vascular changes. The same as intermittent claudication.

Myclosene. Bone marrow used by instillation for treatment of deafness.

Myelorrhaphy. Suturation of the spinal cord, performed for injury which has divided the cord.

Myofibrosis. A degenerative replacing of muscle fibre by fibroid tissue, illustrated by myofibrosis cordis, uteri, etc.

Myogen. A food product prepared from meat.

Myolysis cardis toxica. The degeneration of cardiac muscle fiber caused by systemic intoxication, such as may occur in diphtheria.

Myorrhaphy. Sewing together of muscular tissue.

Myxidiotie. A type of myxedema in which the characteristic symptoms are not very evident with the exception of defective mental development.

Myxoneurosis intestinalis. A non-inflammatory disease characterized by the passage of shreds of mucus, the symptom of an intestinal neurosis. Generally associated with enteroptosis.

N

Nandhiroba. A South American seed used as purgative, febrifuge, emetic.

Narcissus pseudo-narcissus. The daffodil. Emetic in doses 45 to 75 grains.

Narcosis, Medullary n. See Cocainization, spinal c.

Narcotile. Bichlorid of methylethylene. Transparent, colorless, highly volatile, inflammable, not decomposed by light. A general anesthetic.

Nargol. A chemical combination of nucleinic acid and metallic silver. Non-irritating, non-coagulable. Contains 10 per cent metallic silver as compared with 63.5 per cent in silver nitrate, 8.3 per cent in protargol, 30 per cent in argyrol.

Neo-arsyocodyle. The same as disodium methylarsenate, q. v.

Neodermin. Ointment of 5 per cent fluor-pseudocumol. Local application to wounds, burns, ulcers.

Neoferrum. The proprietary name for the combination of a maltopeptonate of iron and manganese, a small quantity of arsenic, with maltine and sherry wine.

Nephrospasis. Abnormal mobility of the kidney.

Nercibus. A syrupy preparation of glycero-phosphates, iron, strychnin, etc. Dose: 1 to 4 teaspoonfuls.

Nervocidin. The alkaloid of an In-

dian plant, gasu-basu. Used as dental local anesthetic.

Neurenergen. A substance supposed to be appropriated by the neurons from the vital fluids and which is utilized in maintaining their latent or active energy.

Neurilla. A proprietary remedy said to be a nerve-tonic and to contain the active principle of scutellaria with aromatics.

Nicofebrin. An antipyretic.

Node, Singers' n's. Circumscribed hypertrophy of the margin of the vocal cords which results from protracted local irritation.

Nori. A food product made from sea algae.

Nuclease. An enzyme which has bacteriolytic power on the organism which produced it and sometimes on others also. Special enzymes are named according to the producing bacteria, thus pyocyanase, cholerase, etc.

O

Obesity, Hyperplasmic o. Obesity characterized by increase of the body density from increase of protoplasm, especially of muscles. The type of obesity is important for treatment and is determined by estimation of the body density and its factors. **Hypoplasmic o.** Obesity characterized by diminution of the amount of protoplasm, the increased body weight being produced merely by increase of fat or water, or both.

Obliquity, Naegele's o. The presentation of the anterior parietal bone in the middle, the axis, of the pelvic entrance.

Oil, Margosa o. An oil derived from the Indian lilac or neem tree. Used for leprosy, rheumatism, mange in dogs, etc.

Olecco. A proprietary laxative preparation containing 80 per cent castor oil with the odor and taste disguised.

Omunono. A local name for yaws or frambesia, a contagious disease of the skin occurring in the tropics.

Oophoropeliopexy. The same as adnexopexy, q. v.

Oophoropexy. The same as adnexopexy, q. v.

Operation, Talma's o. Artificially inducing adhesions between the liver and spleen, the omentum and abdominal parietes for the treatment of ascites from hepatic cirrhosis.

Oresol. Glycerinic-guaiacol ether. Used for tuberculosis.

Orthodiagraph. An instrument for drawing exact reproduction of the boundaries of the heart or other organs as seen by the fluoroscope.

Osmosum. A proprietary name for a preparation of aluminum silicate, glycerin and antiseptics. Used externally as antiphlogistic and anodyne substitute for poultices.

Ossin. Combination of the oleates of cod-liver oil with albumin and sugar.

Osteotabes infantum. An affection of the first year dependent upon faulty function of the bone marrow which results in internal atrophy of bones with anemia and hemophilia.

Ovoferrin. A salt of organic iron $C_{47}N_{17}SH_5Fe_3O_{22}$, produced in liquid form. One tablespoonful is equivalent to one grain of elementary iron. Dose: ½ to 1 tablespoonful. Said to be very readily assimilable.

Oxaphor. Oxycamphor in 50 per cent alcoholic solution. Allays excitability of respiratory centers. Dose: 15 to 45 minims per day.

Ozonoform. A liquid disinfectant, antiseptic mouth-wash and gargle.

P

Palmiacol. $C_{28}H_{40}O_2$, a proprietary derivative of beechwood creosote. Used for respiratory diseases, etc.

Pancreon. Combination of pancreatin with tannic acid. A digestant unaffected by gastric juice. Dose: 5 to 8 grains.

Pani-ghao. Water-itch. A disease of the lower extremities found among laborers in certain tropical regions. Due to larval ankylostoma duodenalis introduced through the soil.

Panniculitis. A disease of the abdominal walls characterized objectively by stiffness and lack of elasticity in the belly wall, and subjectively by intense pain whenever the wall is so manipulated as to throw the peritoneum into folds.

Panzyme. A proprietary name for a combination of the digestive ferments diastase, pancreatin, pepsin and rennin, with aromatics and carminatives.

Parapsoriasis. A chronic disease of the skin with resemblances to lichen, psoriasis, and erythroderma pityriasis.

Peptonoids. A concentrated predigested food made from beef, milk and gluten. Prepared as liquid or powder.

Perdynamin. A hematogenic preparation.

Periendothelioma. A tumor derived from the endothelium of the lymphatics of the perithelial layers of the blood vessels.

Perineotomy. Surgical incision of the perineum antero-posteriorly for obstetric purposes. Distinguished from episiotomy which is, technically, the cutting of lateral incisions through the vulva for the same purpose.

Perioneal (per-e-o-neal) [Gr. oon, egg]. A designation of the cavity and fluid of the deciduae. In early pregnancy these may be greater than the amniotic cavity and fluid. The fluid escaping first in abortions is often the perioneal fluid. With the union of the reflexa and vera in the 4th month the perioneal cavity and fluid disappear.

Perityphlitis. A word used abroad somewhat to designate inflammation of the vermiform appendix, that organ being thought of as something which is upon or touching the cecum. With this nomenclature inflammation of the cecal peritoneum must be paratyphlitis.

Peroscols. A liquid antiseptic containing 3 per cent peroxid of hydrogen with salicylic acid and other antiseptics.

Persodine. A 12:1000 aqueous solution of sodium persulphate. Antipyretic, tonic. Dose: 1 to 2 drams.

Peruol. The active constituent of balsam Peru, 25 per cent, dissolved in oil. Non-irritating, colorless, almost odorless. Used as application for diseases of the skin.

Peruscabin. Benzoic acid benzyl ester. Artificial product representing the active constituents of balsam of Peru. Odorless, non-staining. For scabies. Dilute with 3 parts castor oil.

Pes gigas. Macropodia. Congenital hypertrophy of the foot.

Phaselin. The active principle of the *dilkos mexicano* bean. Used as a digestant, antiseptic and sedative internally and as a dressing for wounds, etc.

Phenadul. A proprietary antiseptic and analgesic preparation. Used externally and internally.

Phenosol. Salicylic acid paraphenetid. Sparingly soluble needles. Antipyretic, antirheumatic. Dose: 5 to 10 grains.

Phlebarteriectasia. A general diffuse dilatation of the veins and arteries without communication between them. Pains and threatened hem-

orrhage in this rare affection often demand operative interference.

Phosferrol. A preparation of cod-liver oil, ext. malt, glycerophosphates, iron, strychnine, etc. Dose: 2 drams.

Phosphotal. A proprietary name for neutral phosphite of creosote. Contains 90 per cent of creosote, 9 per cent of phosphorous.

Phytoroides. A proprietary combination of thyroid extract, phytolacca, fucus vesiculosus, etc., used as a remedy for obesity.

Pince-ciseaux (pin-s-se-zo) [Fr. forcep-scissors]. Cutting forceps for iridotomy.

Pitchblende. A pitch-black mineral consisting chiefly of the oxid of uranium. The source of radium and polonium.

Placenta circumvallata [L. *circumvallo*, to completely wall in]. The highest grade of placenta marginata; a bowl-shaped placenta. The surrounding wall is thick, removed 5 or 6 cm. from the margin of the placenta, and is the remains of a thickened, fibrinous degenerated decidua, the result generally of endometritis decidua.

Placentolysin. An antibody which has the power of destroying placental cells.

Plague, Septicemic p. A variety of bubonic plague in which there are severe constitutional symptoms with lack of local phenomena, following upon unobserved lymphatic enlargement or gastro-intestinal infection.

Plasmolysis. 1. The disorganization of the achromatic part of the cell in distinction from karyolysis. Achromatolysis. 2. Destruction of red blood corpuscles. Erythrocytolysis.

Plasmon. A very readily digestible food prepared from the albumins of milks. Contains 77 per cent mostly soluble casein.

Pneumagalactocele. A tumor of the puerperal breast containing milk and gas.

Pneumocephalus. Air within the cranial cavity. Induced as a surgical measure for the relief of hydrocephalus.

Polonium [L. Polonia, Poland, named for the birthplace of the discoverer]. A very rare metal obtained from pitchblende. Emanates rays of light and heat in manner similar to radium, but less active.

Polymazia. The existence of supernumerary mammary glands.

Precipitin. A uniceptor antibody produced by the methods of artificial immunization, which has the power of precipitating from solution the specific proteid which was used to produce the precipitin.

Precipitum. The precipitate formed by a precipitin.

Propol. Disinfectant for wounds and instruments.

Protan. Combination of tannic acid, 50 per cent, and nucleo-proteid. Brown, tasteless powder, insoluble in water or acids; broken up in alkaline fluids. Used as intestinal astringent in diarrhea. Dose 15 to 30 grains.

Proteinum pyocyaneum. An extract of the cultures of bacillus pyocyaneus. A lotion for ulcerous affections.

Prunol. A proprietary cough sedative containing ammonium chlorid, senega, etc., and heroin $^1/_{24}$ grain to the teaspoonful.

Pseudohydronephrosis. A paranephric cyst, q. v.

Pseudopepsin. A proteolytic enzyme secreted by certain glands of the stomach. It acts in alkaline solution and is otherwise different from pepsin.

Punctumeter. An instrument for ascertaining the range of accommodation.

Purgatol. An acetyl ether of anthrachinon. Yellowish insoluble powder. Slow-acting cathartic. Col-

ors feces and urine red. Dose: 7 to 15 grains.

Purin. A chemical body containing the nucleus C_5N_4, which includes the xanthins, uric acid, guanin, etc.

Puritas. A proprietary antiseptic powder for use dry or in solution. A compound of sodium borate, carbolic acid, eucalyptus, etc.

Puroform. Disinfectant preparation.

Pyocyanase. A nuclease derived from *bacillus pyocyaneus*. It has a bacteriolytic action upon typhoid, cholera, pest, diphtheria and its own organisms.

Pyraconitine. A derivative of aconitine. Physiologic action similar, but less toxic.

Pyridin tannate. Uric acid solvent and intestinal astringent.

Pyroferrine. A proprietary tonic containing iron pyrophosphate, strychnin and phosphoric acid.

Q

Quinoliv. A proprietary powder combining quinin sulphate and olive oil. Said to be tasteless and to cause no unpleasant after effects.

R

Radio-praxis [Gr. praxis, action, practice]. The therapeutic use of light rays including the x-ray and ultra-violet rays.

Radium [named for its properties]. A very rare and costly metal obtained in minute quantities from pitchblende. Its properties include continuous emission of light, of heat, of x-rays, of photographic rays, etc.

Rays, Becquerel r's. The light rays emitted from uranium, a mixture of cathode and x-rays.

Reagent, Obermayer's r. Two grams of $FeCl_3$ in 1 liter HCl.

Receptor. An atom group of a cell molecule which group exhibits either in the cell or when cast off into the serum a definite affinity for atomic groups, haptophores, of toxins or foods.

Reflex, Achilles r. Striking the tendo Achilles sharply, foot off the floor, causes normally a quick plantar flexion of the whole foot. Much the same significance as the knee-jerk reflex. **Babinski's r.** Dorsal extension of the great toe upon irritating the sole of the foot. Its presence means interference with the function of the pyramidal tract on the corresponding side. Found normally in young infants.

Renaglandin. A hemostatic solution prepared from the adrenals.

Renoform. A preparation of the adrenals used for nasal application.

Resaldol. Prepared from chlormethyl-salicyl and resorcin. Brown, astringent powder insoluble in water and acids. Intestinal astringent and antiseptic. Dose: 20 grains.

Respiton. A proprietary remedy prepared from asclepias and berberis. Said to be indicated in bronchial, catarrhal and cutaneous affections.

Rheumasan. A salicylic acid soap used in gout.

Roborat. A vegetable albuminoid nutrient preparation. Given mixed with other food in tablespoonful doses.

Rubella scarlatinosa. An exanthematous disease with the eruption, sore throat and desquamation resembling scarlet fever, but in mildness, and other features resembling German measles. Differentiated by some observers as a disease entity to be separated from German measles which more closely resembles measles.

Rubidiol. Solution of iodo-hydrargyrate of rubidium and potassium in oil. External application as a resolvent.

S

Saccharomycetolysis. The disintegration or splitting up of the saccharomyces or yeast-fungi.

Salacetin. Phenylamine aceto-salicylate. Analgesic, antiseptic, lithia solvent.

Saloquinine or Salochinin. The salicylic acid ester of quinin. Crystals, insoluble in water, soluble in alcohol and ether. Tasteless, non-toxic, non-irritating internally. Dose: 15 to 30 grains. Combines therapeutic properties of quinin and salicylic acid.

Salpingitis profluens. Inflammation of the oviduct in which the secretions after being locked up for a time in the tube are discharged, usually through the uterine ostium. Requiring a definite number of days for accumulation the discharge may be periodic and cause intermenstrual pain.

Salpingostomatomie. Conservative operation on the oviduct. The tube is resected and a new abdominal ostium formed by uniting the mucosa and serosa surfaces.

Sand, Intestinal s. Formed mostly in the upper colon, is usually accompanied by pain and symptoms of mucous colitis. Consists of brownish granules, composed of bacteria, oxides of calcium and phosphorus, bile pigment, etc. Distinguished from false intestinal sand which is composed of vegetable cells mostly from pears and bananas.

Sangogen (san-go-jen). A proprietary powdered preparation of predigested albuminate of iron and manganese in combination with arsenic and strychnia.

Sanguestine. A preparation of the active principle of the suprarenal glands used either in powder or in weak solution.

Sanosin. A preparation of flowers of sulphur, powdered charcoal and pulverized eucalyptus leaves impregnated with essential oil of eucalyptus. The fumes are inhaled for phthisis.

Sarcomatosis cutis. Sarcoid growths of the skin resembling in clinical history granulomata.

Satyria. A proprietary name for a genito-urinary tonic said to contain saw palmetto, phosphorus, bitter sweet, muira-puama, etc.

Secretine. The fluid obtained by acid maceration of the duodenal mucosa from a fasting mammal. Said to exert a remarkable influence upon the secretion of the pancreatic juice and bile when injected into other animals.

Senasal. A proprietary cathartic prepared from senna, phosphate of sodium and aromatics.

Septoform. A condensation product of formaldehyd. A disinfectant used in 3 to 10 per cent solution.

Serum, Haffkine's s. A serum used by inoculation as a prophylactic against the bubonic plague. **Inorganic s.** A mixture of the alkalin salts in the proportions in which they exist in normal blood serum. Used hypodermatically in doses of 1 to 5 c.c. to combat cerebral arteriosclerosis. Formula: Sodium sulphate, grams 0.44; Sodium chlorid, 4.92; Sodium phosphate 0.15; Sodium carbonate 0.21; Potassium sulphate 0.40; Distilled water q. s. ad. 100. **Maragliano's s.** Anti-tubercular serum. **Marmorek's s.** Derived from streptococcus pyogenes. Used as preventive and curative of streptococcus infections. **Trunecek's s.** The same as inorganic s., q. v. **Wlaeff's s.** Obtained by inoculating birds with pathogenic blastomycetes from cancer. Used by injection for the treatment of cancer. **Yersin's s.** A serum employed by inoculation in cases di-

rectly exposed to or suffering from the bubonic plague.

Siccolea. Preparations in which remedies with a bad taste, such as castor oil, santal oil, etc., are exhibited in dry form.

Side-chain. See Theory, Side-chain t.

Sidonal. Quinate of piperazin. White powder, very soluble in water. Uric acid solvent and excretant. Dose: 75 to 120 grains per day.

Sidonal New. Quinic acid anhydrid. Uric acid solvent. Dose: 75 to 120 grains a day.

Sign, Anterior tibial s. Involuntary overtension of the tibialis anticus when the thigh is forcibly flexed upon the abdomen. Seen best in spastic paretic conditions. **Koplik's s.** The appearance upon the mucous membrane of the cheek or lips of small dark-red spots surmounted by minute bluish white specks. These appear from one to five days before the cutaneous eruption in measles and are pathognomonic.

Silver fluorid. A surgical antiseptic used in solutions from 1:100 to 1:10,000. The same as tachiol.

Sirikaya. The tree *Anona squamosa*. The bark is said to be purgative and the leaves surodific.

Sirolin. A 10 per cent solution of thiocol in orange syrup.

Sodium persulphate. A salt which readily liberates oxygen. Used in 3 to 5 per cent solution wet dressing for lupus and ulcers. Internally tonic and antipyretic. Dose: 1½ grains.

Sodium sozoiodolate. White crystalline powder. Injected subcutaneously or given internally for phthisis. Dose: 0.025 gram for injection, 0.25 to 0.5 gram internally.

Somnoform. A rapidly acting general anesthetic composed of ethyl chlorid, 60 parts; methyl chlorid, 35 parts; ethyl bromid, 5 parts.

Somnos. Chloraethanal alcoholate. Said to be free from depressing effects, non-irritative to mucous membranes, without unpleasant after-effects. A sedative and hypnotic. Dose: Dessertspoonful to tablespoonful in water or milk. Repeat if necessary.

Sonnin. An antiseptic preparation; a compound of boric acid and phenols.

Spirits, Columbian s. Deodorized methyl alcohol, wood alcohol.

Sporidium vaccinale. Bodies, probably protozoa, discovered in certain lesions of vaccinia. See cytoryctes v.

Stain, Wright's blood s. A very easy and reliable stain for blood. Alcoholic solution of methylen blue and eosin, specially prepared. Stain on coverglass one minute, add water to stain until translucent, with metallic scum. Leave on 3 minutes. Wash. Dry.

Status lymphaticus. A condition characterized anatomically by enlarged thymus and spleen, hyperplasia of the lymphatic tissues and hypoplasia of the heart and aorta. Characterized clinically by lowered vitality and unstable equilibrium of the vital forces. Hardly to be diagnosed during life.

Stegomyia. A genus of the family Culicidae, order Diptera. The genus has 3 species in North America, found rarely above 40° North latitude. This genus, particularly the species fasciata, is the intermediate host for the parasite of yellow fever.

Stenosin. The same as disodium methylarsenate, q. v.

Stereoskiagraphy. The making of a picture or photograph by the Roentgen rays so that the natural appearance of solidity or relief is given to the objects.

Stomatitis, Erythematopultaceous s. A variety of uremic stomatitis in which the reddened mucous membrane is covered by a thick, sticky covering.

Strongyloides stercoralis. A species of small nematodes, one generation

of which is parasitic and occurs in the human intestine. Symptoms: not necessarily any, perhaps dysentery. Embryos .3 to .6 mm. long passed in great numbers in feces. Infection probably from water. Adult worms 2 mm. long. Also named, S. intestinalis, Anguillula intestinalis, etc.

Sublamin. Ethylenediamine-sulphate of mercury. White needle-like crystals, readily soluble in water with alkaline reaction. Equal in toxicity and bactericide power to corrosive sublimate, and used in same strengths. Penetration greater and irritation less than corrosive sublimate. Used also as antisyphilitic.

Succus alterans. A proprietary antisyphilitic and alterative containing stillingia, sarsaparilla, phytolacca, lappa minor, and xanthoxylum.

Suprarenalin. The isolated active principle of the suprarenal gland. Used in powder or 1:1000 solution. Local and general hemostatic. Raises blood pressure.

Syncytiolysin. An antibody which has the power of destroying the syncytium, the outermost fetal layer of the placenta.

Synergia. A proprietary aromatic digestant preparation containing nearly all of the digestive ferments.

Synthol. A chemical synthetic substitute for absolute alcohol.

T

Tachiol. A name for silver fluorid, q. v.

Tannin-aleuronat. A combination of tannic acid and albumin. A mild astringent nutrient for dysentery, etc.

Tannochrom. A yellow dressing powder, 50 per cent resorcin-chromium bitannate.

Tanocol. Brown, tasteless and odorless powder containing equal parts of tannin and gelatin. Dissolved in the intestine after passing through stomach. An intestinal astringent. Dose: 15 grains.

Tegone. A preparation of glycerin and agar-agar used for making bandages.

Test, Bell's t. (for approximate quantitative estimate of free HCl in stomach contents). To 4 c.c. filtered chyme add solution of dimethylamido-azo-benzol, drop by drop, until the resulting pink color no longer deepens. Quickly compare with Bell's color-scale for the approximate percentage. **Ewald's t.** (for motility of the stomach). After light meal 15 grains of salol are given in capsules. Urine is then passed frequently for 3 hours, and the specimens preserved separately. The salol normally is passed into the intestine where it is decomposed into phenol and salicylic acid so that salicyluric acid appears in the urine 40 to 75 minutes after ingestion. Weak solution of ferric chloride added to even trace of salicyluric acid gives a purple color. **Hay's t.** (for bile in urine). Sublimed sulphur added to urine quickly falls to bottom if bile is present. Otherwise the sulphur does not sink or does slowly. Reaction due to alteration of surface tension. Will occur if other substances rarely in urine, such as alcohol, chloroform, phenol, etc., are present. Reaction quite sensitive. Urine must be cold. **Knapp's t.** (for lactic and organic acids in stomach contents). Extract 1 c.c. filtered chyme with 5 c.c. ether. Float the ethereal extract in narrow test-tubes on dilute iron solution (1 drop 10 per cent ferric chlorid solution to 2 c.c. water). Various colored rings changing under certain conditions indicate the various acids. **Nitropropial t.** (for sugar in urine).

Heat ortho-nitro-phenyl-propiolic acid with the urine and alkali. Test striking and delicate. **Penzold's t.** (for stomach absorption). A 3 grain capsule of potassium iodid is given on empty stomach and followed by glass of water. Dried strips of starch paper are moistened with the patient's saliva and touched with a drop of fuming nitric acid. A violet or blue color results normally in 6 to 15 minutes. **Phloridzin t.** (for renal insufficiency). Phloridzin, 5 to 10 milligrams with sodium carbonate same quantity is given hypodermatically just after bladder is emptied. If kidney epithelium healthy sugar will appear in urine within half hour. If none appears by that time serious disease should be suspected; if only small quantity of sugar renal insufficiency is probable. **Pollacci's t.** (for albumin in urine). Solution A: tartaric acid 1 gm., mercuric chlorid 5 gm., sodium chlorid 10 gm., dissolved in 100 c.c. water. Solution B: Solution A + 5 c.c. formaldehyd (40 per cent). Solution B added to urine without admixture causes coagulation of albumin in a white zone. **Ruhemann's t.** (for uric acid in urine). Based on the principle that iodin is neutralized by uric acid until the brown color disappears. Requires definite quantities and a graduated scale. **Safranin t.** (for sugar in urine). Urine to which an equal quantity of normal sodium hydrate solution is added will decolorize safranin if heated to 180° F. and sugar is present. **Sahli's t.** (for estimating the digestive and motile power of the stomach). A soup prepared of definite quantities of flour, butter, water and salt is ingested and after an hour the stomach contents removed. Estimating the quantities, the acidity and the amount of fat shows how much has been passed on and how much liquid the stomach has secreted. **Serum t.** (for human blood). Into a rabbit human blood serum is injected several times. Physiologic salt solution of human blood, even from an old stain, added to serum of a rabbit so treated causes cloudiness of the serum. **Storch's t.** (for the detection of human milk). Hydrogen peroxid is split up by a ferment in human milk. **Trousseau's t.** (for bile in urine). Tincture of iodin diluted 1:10 with alcohol is poured into test-tube containing urine. In presence of bilirubin a green ring forms where the fluids join. **Uhlenmuth's t.** See Serum test.

Test-meal, Boas' t-m. (for accurate test of lactic acid in stomach). One ounce rolled oats boiled in quart of water down to one pint. Salt to taste. Take this for breakfast. If atony wash out stomach night before. Two shredded wheat biscuits or pint bowl of granose will substitute if taken plain. **Ewald's t-m.** (for general estimate of stomach functions). Two slices of light white bread and glass of water or cup of plain tea, ingested after fasting overnight and withdrawn one hour later.

Thearoma. A preparation of aromatics to be added to cod liver oil and castor oil for the purpose of rendering them more palatable. Used in the proportion of 2 drops to the teaspoonful of oil.

Theocin. An alkaloid first obtained from tea-leaves, theophyllin, later made synthetically. Colorless crystalline plates, soluble in water. An efficient diuretic. Dose: 4 grains.

Theory, Side-chain t. A theory advanced by Ehrlich to explain antibodies and immunity. In systemic cells there are, apart from the functionating center, "side-chains" or groups of molecules, receptors, which normally take up food, and which in the presence of appropriate groups, haptophores, of toxin molecules are joined thereto.

Side-chains of the cell having been thus used up, new ones even in excess may be formed and these when thrown off into the serum constitute antitoxins. For the effective union of any side-chains with invading cells, such as bacteria, the presence of a third body, amboceptor, is necessary. Side-chains are thus concerned in the development of all antibodies.

Thermofuge. A proprietary name for a preparation of aluminum silicate, glycerin and antiseptics. Used externally as antiphlogistic and anodyne substitute for poultices.

Thermolabile. Altered by moderate heat.

Thermostable. Not altered by moderate heat.

Thiocol. Guaiacol-sulphonate of potassium. White, odorless powder, readily soluble in water. Used for tuberculosis and respiratory diseases. Dose: 10 to 30 grains.

Thymol-urethan. An anthelmintic.

Toxaphore. An atomic group of a toxin which becomes destructive to the body cells when the toxin haptophores have united with the cell receptors.

Toxins, Coley's mixture of t. An unfiltered mixture of erysipelas and prodigiosus toxins used in minim doses for the treatment of malignant tumors.

Triacol. An aromatic solution of sodium-, potassium-, and ethylmorphine-guaiacol. Indicated in respiratory affections. Dose: 2 drams.

Triangle, Langenbeck's t. An isosceles triangle, the apex corresponding to the anterior superior iliac spine, the base to the anatomic neck of the femur, and the external side to the external face of the great trochanter.

Trichotoxin. An antibody which has the power of injuring epithelial cells.

Tricocephalus trichiurus. A species of widely distributed parasitic nematodes found in the human cecum and colon. Adult forms 50 mm. long. Eggs of microscopical size passed in feces. Nourished at least somewhat from the blood. Symptoms: only when parasites present in large numbers, depression, cardiac weakness, etc. Infection from surface drinking water and uncooked vegetables. Also named, T. dispar, Trichuris trichuria, etc.

Triferrin. Paranucleinic acid iron. Made from casein. Contains 22 per cent iron, phosphorous 2½ per cent. Yellowish brown powder, odorless, tasteless, soluble in alkaline solution. Dose: 5 grains.

Trikresol. A combination of ortho-, meta- and para-cresols. Not so toxic and stronger germicide than carbolic acid. Used as disinfectant and germicide in ½ to 1 per cent solutions.

Trinophenon. An aqueous solution of picric acid used for burns.

Trophedema. A disease occurring in a chronic, hereditary form, characterized by a permanent primary edema of one or both feet, legs or thighs.

Tropon. A nutritive powder, cheap and readily digestible, prepared from vegetable and animal albumins. Useful in phthisis, etc. Dose: 25 grams a day in soup or baked.

Trypanosomiasis. The disease caused by infection with the flagellate parasitic protozoon, trypanosome. Common in animals, rare in man. Chiefly tropical disease. Symptoms: Fever, erythema, anemia. Infection probably through bites of insects.

Tua-tua. A plant, probably *Jatropa gossypifolia*, used in leprosy.

Typhoin. A sterile preparation of typhoid bacilli used hypodermatically in typhoid fever. Dose: 0.5 to 3 c.c. daily.

U

Uncinaria. A genus of small nematode parasites two species of which, U. duodenale and U. americana, occur in the human intestine. The source of infection is earth or clay containing the larvae which are of microscopical size. Symptoms; intestinal disturbances and progressive anemia. Ova may be microscopically detected in the feces. Adult worm ¼ to ⅜ inch long. Also named, hook-worm and ankylostoma.

Uncinariasis. The disease caused by uncinaria, q. v.

Uniceptor. A chemical substance found in blood serum as the result of immunization. It has a single bond of affinity which unites it with a toxin. In this capacity it is the antibody called antitoxin.

Urasol. A condensation product of acetic, salicylic acids, and formaldehyd. Antiseptic, diuretic, uric acid solvent. Dose: 5 to 8 grains every 2 or 3 hours.

Urethritis petrificans. The condition in which calcareous deposits occur in the urethral wall. Generally results from devitalization by long-continued gonorrhea.

Uriseptin. A proprietary diuretic and genito-urinary antiseptic, a solution of lithium methaminate, containing formaldehyde and lithia.

V

Valvotomy. The operation of cutting the rectal folds known as Houston's valves.

Valyl. Diethylamid of valerianic acid. Colorless liquid, burning taste. Sedative to nervous system. Dose: 2 to 4 grains in capsule.

Varoma. A proprietary disinfectant. Used for vaporizing. Said to be efficacious for whooping-cough.

Vasa previa. Presentation at the uterine outlet, preceding the fetus, of the blood-vessels of the cord where they branch off to enter the placenta. It only occurs with a low-lying placenta and insertion of the vessels at one edge, velamentous attachment of the cord.

Veins, Vortex v's. Four venous trunks into which gather tributary whorls in the superficial layer of the choroid.

Velvril. A mixture of nitrocellulose with nitrated oil, soluble only in acetone. As an elastic film which is impervious to all ordinary liquids and which may be sterilized in boiling water it is used for sealing operation wounds. The edges are fastened to the skin with the acetone solution.

Ventroscopy. Examination of the abdominal cavity through vaginal incision. Illumination from head mirror or, better, from incandescent bulb attached to one of the retractors.

Veronal. A white powder, faint bitter taste, sparingly soluble in water. Derived from urea. A hypnotic. Dose: 4 to 15 grains at bed time.

Vibratode. The terminal apparatus with which vibrations generated by the vibrator are directly applied.

Vioform. Iodochloroxychinolin. Odorless, non-irritant substitute for iodoform.

Virilin. A proprietary aphrodisiac containing yohimbin, strychnin, glycero-phosphates, etc.

Viskolein. A proprietary antipyretic and stimulant preparation containing a coal-tar product, an alkaloid from kola, boric acid, menthol, etc.

Vitalizon. A proprietary tonic containing phosphorus, nux vomica, etc.

Volesan. A proprietary combination of creosote carbonate, heroin, camphor, balsam tolu.

W

Wismol. A dressing powder, substitute for iodoform.

X

Xanol. Sodio-caffein salicylate. Heart tonic. Used hypodermatically.

Xanthone. A drug subsequently named brometone, q. v.

Y

Yohimbin. An alkaloid from a tropical tree. Aphrodisiac. Hydrochlorid given in doses $^1/_{10}$ to $^1/_6$ grain in considerable water.

Z

Zenoleum. A proprietary disinfectant.

Zomol [Gr. zomos, juice of flesh]. A desiccated preparation of meat plasma. Dose: at least 10 grams daily.

Zomotherapy. The treatment of disease by (1) meat as diet or by (2) injections of muscle-plasma.

Zymin. A sterile yeast used as a germicide, especially in preparation for vaginal operations.

INDEX.

INDEX OF AUTHORS.