very small incision. Immediately upon delivery the vaginal incision was closed with silver wire sutures, which took in the walls of the ureter. Some hemorrhage occurred in the anterior cul-de-sac, and as it was not desired to take any more time a few clamps were left on the vessels. A gauze drain was put in. The whole operation took ten minutes, and the stone was delivered in five minutes. The convalescence was not remarkable and there was no fistula. The ureter has remained pervious since the operation for both ureters have been catheterized since. The stone was a very large one and it could not possibly have been passed *per vias naturales*. It weighed in the dry state 7 dg. and it was 1 cm. long and nearly 1 cm. wide at the widest part. It consisted of phosphate of lime.

At the time this operation was done I was not aware that Doyen* had originated an almost similar operation for the removal of stones in the ureter. Doyen's operation, however, differs in a number of essentials. He incises the anterior cul-de-sac, and the ureter is exposed in the cul-de-sac. Then the stone is removed *through the cul-de-sac* itself and *not* through the vaginal wall. The operation terminates by suture of the ureteral walls and a drain is left in the cul-de-sac. Doyen's operation must take a great deal of time on account of the technical difficulties, and a fistula must be more serious in the anterior cul-de-sac than in the vagina, should one result. The advantages of the operation as performed upon my patient are obvious. The technique is simple. Hemorrhage is under perfect control. It is very rapid. I feel certain that very high stones can be pushed down and brought within reach. If fistula results the consequences will not be as serious as they are when the sacral, the rectal, or the Doyen method is employed, and operations for fistula are easier through the vagina than through the other routes mentioned. The ureteral and the vaginal walls can be sutured *en masse* with success. As the ureter in these cases is always dilated, this suturing is less difficult than it appears on first sight. If it leaks subsequently, the leakage will take place between the stitches and no harm will result. Of all the advantages gained, that with reference to time is the most important. These patients are almost always desperately ill, and time is an element sometimes not sufficiently considered.

Medical Progress.

REPORT ON PATHOLOGY.

BY JOSEPH H. PRATT, M.D., BOSTON.

THE PATHOLOGICAL ANATOMY OF PARATYPHOID FEVER.

Wells and Scott[1] report a fatal case of paratyphoid fever. Only four other instances have been recorded in the literature although nearly a hundred cases of the disease have been recognized during the past six years. The chief clinical features in the case studied by the writers was the continued fever which reached 104° on the day of admission to the hospital. The pulse was somewhat accelerated and dicrotic. A few rose spots appeared on the abdomen and the spleen was palpable. Intestinal hemorrhage occurred early in the disease. The patient died during the fifth week of illness. Agglutination tests with the typhoid bacillus were negative. Unfortunately the reaction of the blood with paratyphoid bacilli was not tested. At the autopsy a paratyphoid bacillus was obtained from the spleen and kidney and there can be little, if any, doubt that the case was one of paratyphoid infection. The organism produced a terminal alkalinity in milk and hence belongs in group β of Buxton's classification, although the authors place it in group α.

The spleen weighed 400 gm. and was very soft. The lymphatic apparatus of the intestine was not involved and the mesenteric lymph nodes preserved their normal size. Numerous irregular superficial ulcers were found in the ileum just above the ileo-cecal valve. There was no infiltration around the ulcers and they resembled those of dysentery much more than those of typhoid. Similar ulcers occurred in the case reported by Lucksch. Intestinal lesions were absent in the three other fatal cases.

The absence of proliferation and phagocytosis in the lymph nodules, which Mallory has shown to be the characteristic intestinal changes in typhoid fever, as well as the absence of any involvement of Peyer's patches in paratyphoid fever indicate, as the authors state, that there is some essential if obscure biological difference between the typhoid and paratyphoid bacilli.

The anatomical picture of paratyphoid fever is that of a septicemia associated with splenic enlargement and occasionally with non-specific ulcers in the intestine.

THE GENESIS OF BLOOD PLATES.

The opinion held by many pathologists that blood plates are products of red blood corpuscles is largely based on the observations of Wlassow made in Ziegler's laboratory some years ago. This investigator found that when blood was treated with a solution of mercuric chloride (saturated aqueous solution of mercuric chloride 10 parts, distilled water 50 parts) there were quickly formed from the erythrocytes red bodies with sharp outlines which gradually became transformed into colorless structures resembling blood plates. Other reagents acted similarly on the erythrocytes. Wlassow observed that these bodies like the blood plates were stained with certain aniline basic dyes and he regarded the two as identical and claimed that this experiment showed that the blood plates are not independent cellular elements of the blood but are formed from the erythrocytes.

Sacerdotti[2] states that the bodies described by Wlassow are not blood plates and morphological differences exist between the two struc-

* H. Morris. Surgical Diseases of the Kidney and Ureter. London, 1901, Vol. II, page 530.

[1] Journal of Infectious Diseases, 1904, i, p. 72.

[2] Anatomischer Anzeiger, xvii, p. 249.

tures, although they appear similar at the first glance. The little knobs on the erythrocytes are never flattened, while the blood plates are flat. The former are homogeneous and frequently tinged with hemoglobin and thereby differ from the blood plates which present a granular appearance and are always colorless. Another difference was shown by treating a microscopical preparation prepared by Wlassow's method with a dilute solution of acetic acid, preferably a 5% solution. The small globular knobs and the red blood corpuscles vanish while the blood plates remain and their granular appearance becomes more distinct.

Sacerdotti performed another experiment. He defibrinated blood, in order to remove all the blood plates and then mixed the blood with sublimate solution. After the small globular bodies were formed acetic acid was added. The erythrocytes and globules disappeared and nothing remained which bore any resemblance to blood plates. If the action of the acetic acid was studied under the microscope it was seen that Wlassow's globules first swelled, gradually paled and finally disappeared. The erythrocytes were more resistant and for a time a crater marked the site of the dissolved knob. With such differences in form and nature existing between Wlassow's bodies and the plates, how can the view be entertained, says the author, that the blood plates are derived from the erythrocytes?

Schneider[3] under Arnold's direction has repeated Sacerdotti's experiments. When a drop of blood is mixed with Wlassow's fluid changes in the erythrocytes occur with great rapidity. At the moment of contact the erythrocytes become smaller and globular. They lose their elasticity entirely and appear completely fixed. A few of the fixed elements appear as bell-shaped bodies with a concavity on one side. The author does not agree with Weidenreich that the normal form of the erythrocytes is bell-shaped. The majority of the erythrocytes appear as spherical structures. A portion of many of the corpuscles is finally granular and colorless or faintly tinted with hemoglobin. The chief mass of the hemoglobin is collected at the other pole of the erythrocyte where the peculiar little knob is attached. This little body is usually spherical. If two are present they are rarely of equal size. They vary from one fifth to one third of the size of the erythrocyte. Most of the bodies contain hemoglobin and are homogeneous. Transitions were seen between these and quite colorless, slightly granular forms. Some were only attached to the erythrocyte by a thread-like connection, but no free bodies were seen, such as Maximow has described. Schneider calls attention to the fact that both Wlassow's bodies and blood plates stain similarly with methyl violet and gentian violet. But he agrees with Sacerdotti that the plates resist the action of dilute acetic acid while the structures formed from the erythrocytes by the mercuric chloride solution are quickly dissolved. Schneider points out that red blood corpuscles have been shown to contain nucleoid material and he asserts that a part of the erythrocyte is not dissolved by the acetic acid but remains as a shadow and concludes from this that Sacerdotti's experiment does not prove that blood plates cannot be formed from the erythrocytes.

On agar preparations, prepared by Deetjen's method and containing 2 to 10% Na Cl, the author states that blood plates are formed in large numbers from the red blood corpuscles and that some resist acetic acid. As the result of his studies he concludes that the blood plates are not independent cells but simply cell derivatives. The great majority of the plates of normal blood according to Schneider arise from the erythrocytes, but some are derived from the leucocytes. Most of the plates possess nuclear substance, which can be demonstrated by certain staining methods and the nuclear substance is the cause of their resistance when brought in contact with dilute acetic acid.

[3] Virchow's Archiv., 1903, clxxiv, p. 294.

AN EXPERIMENTAL STUDY OF THE SO-CALLED FATTY DEGENERATION.

Fischler[4] arrives at the following conclusions based on a study of the fat in kidney infarcts experimentally produced in rabbits.

The appearance of visible fat in cells, as well in the so-called fatty degeneration, as in fatty infiltration is dependent not alone upon a certain condition of the cell, but upon factors outside of the cell.

Some circulation must be maintained although it need not be complete, of the blood, lymph or diffusion stream, or fatty metamorphosis will not occur.

The most essential condition for the appearance of fat is the life of the cell. Dead cells do not undergo fatty metamorphosis. The experiments summarized in this study add nothing to the support of the theory that fat can originate from the cell proteids. Without doubt the presence of fat within the cell can be due to a great variety of conditions.

THE DIAGNOSTIC VALUE OF BLOOD CULTURES.

Hecktoen[5] records a case in which the concurrence of scarlet fever and typhoid fever was observed. The patient was suddenly taken ill with nausea, headache and pain in the limbs. Deglutition became difficult and painful. On the day following the onset a rash developed on the neck and body and spread to the face and extremities. When admitted to the hospital on the fourth day of the disease there was a deep subcuticular flush with raised points. The tongue had the "strawberry" appearance seen in scarlet fever and the tonsils were swollen. A culture from the blood made the next day yielded a pure growth of the typhoid bacillus and the blood gave a positive Grünbaum-Widal reaction. Typical scarlatinal desquamation occurred. The temperature fell to normal about the twenty-first day. The clinical features seem distinctive

[4] Virchow's Archiv., clxx, p. 100.
[5] Medical News, 1903, lxxxiii, p. 580.

of scarlet fever but, as the author admits, absolute evidence as would be furnished by the demonstration of the causative agent is lacking. The occurrence of desquamation is regarded as strongly supporting the diagnosis of scarlet fever. Two cases of typhoid fever associated with early angina and erythema were observed in which there was no exfoliation. In both instances the typhoid bacillus was obtained from the blood during the first week. The author emphasizes the almost constant presence of the typhoid bacillus in the blood in the early stages of the disease and the importance of blood cultures to settle promptly the diagnosis of typhoid fever in such puzzling cases as the ones reported.

OBLITERATION OF THE SUPERIOR VENA CAVA.

Although compression of the superior vena cava is not very uncommon in cases of aortic aneurism and mediastinal tumors, instances of complete obliteration of the vessel with the establishment of collateral circulation are, according to Osler,[6] very rare. In a search of the literature he found only twenty-nine cases of complete obliteration of the superior vena cava.

I. Thrombosis due to disease within the vein, 10 cases. Simple phlebitis seemed to have been the cause of the thrombus formation in 8 of these; one was a propagated thrombus from the periphery (Duchek), and one was due to tuberculous endophlebitis (Banti).

The majority of the cases belonged in the second class.

II. Disease outside of the vein, 19 cases, grouped as follows: (*a*) tuberculosis, 4 cases; (*b*) mediastinitis, 4 cases; (*c*) aneurism, 4 cases; (*d*) syphilis, 3 cases; (*e*) periaortitis, 2 cases; (*f*) carcinoma, 1 case; (*g*) fibroma, 1 case.

Osler divides the cases clinically into two groups. In one group after years of completely effected collateral circulation and good health the symptoms set in acutely. In the other group the symptoms of venous obstruction are constantly present although the individual may enjoy a fair measure of health.

Osler reports an instance of occlusion of the superior vena cava from compression by enlarged mediastinal lymph nodes in Hodgkin's disease and another case in which the diagnosis of fibroid obliteration of the superior vena cava was made during life. The second case presented many interesting and unusual features. When first seen the patient complained of dyspnea and swelling of the neck. There was gradual distention of the superficial thoracic and epigastric veins. The case was regarded during the first year as probably one of intra-thoracic tumor, but the absence of all signs of aneurism and of enlargement of the lymph nodes, the negative result of the x-ray examination, the slow course of the disease, led to the correct diagnosis. Symptoms of phthisis pulmonum developed and tubercle bacilli were found in the sputum. At autopsy the left jugular, both innominate veins, and the upper portion of the superior vena cava were found converted into a dense thick cord. The superior vena cava was patent within the pericardium from the point where it received the dilated azygos vein to the heart. There was tuberculous caries of the last cervical and the two upper thoracic vertebræ. The fibrous tissue about the diseased portion of the spine was continuous with that about the innominate veins.

[6] Bulletin of the Johns Hopkins Hospital, 1903, xiv, p. 169.

THE RELATION OF MYELIN TO FATTY METAMORPHOSIS.

According to Kaiserling and Orgler,[7] myelin drops resemble fat morphologically only. They are stained light gray by osmic acid, never coloring as intensely as fat. With sudan III and scharlach R they assume a red hue. The osmic acid preparations of myelin, unlike fat, are decolorized by xylol, chloroform and oil of bergamot. They differ from fat in being double refractive. In hardened tissues the double refraction of the myelin is lost. These myelin droplets were found in fatty metamorphosis of the cells of intima of arteries, particularly the aorta, in an amyloid kidney, and a great white kidney of chronic Bright's disease, in the epithelial cells of the pulmonary alveoli, in pneumonia and tuberculosis, and in the bronchial secretion, also in the corpus luteum of the ovary and in the cortex of the adrenal and the retrogressive metamorphosis of the thymus gland.

Myelin granules were not found in the heart, in fatty infiltration of the liver, in the normal fat deposits of the body, in colostrum, in functioning mammary glands, or in four cases of fatty metamorphosis of the kidney.

Schmidt and Muller obtained from the myelin drops of the sputum protargon, a highly complex body, but not a fat. By the breaking up of protargon, lecithin and cerebrin are formed.

ORIGIN OF EOSINOPHILIC LEUCOCYTES.

Opie[8] has made an experimental study of the eosinophile cells in the blood and tissues of guinea pigs and the relation of these cells to nutrition. This investigation lends support to Ehrlich's view that the eosinophiles are derived wholly from the bone marrow.

In the mucosa of the intestinal tract and air-passages and in other tissues of the guinea pig occur eosinophilic polymorphonuclear leucocytes identical with those of the blood. In the bone marrow alone mononuclear eosinophiles are found. These undergo mitosis, and transition forms between them and the eosinophiles of the blood can also be demonstrated in the bone-marrow. When the eosinophiles of the blood become increased the number of eosinophilic myelocytes was far greater than normal and proliferation of these cells was more active. The blood vessels of tissues in which accumulation of eosinophiles occurred contained in certain instances a demonstrable increase in the eosinophile leucocytes.

[7] Virchow's Archiv., clxvii, p. 296.
[8] American Journal of the Medical Sciences, 1904, cxxvii, p. 217.

Starvation caused a diminution in the number of eosinophiles in the blood. A temporary loss of weight was accompanied by an increase while a gain in weight led to a decrease of the eosinophiles.

THE PRIMARY ACTION OF THE TUBERCLE BACILLUS.

Baumgarten has maintained that the production of miliary tubercles and diffuse tuberculous tissue is the result of a direct irritation by the tubercle bacillus of the fixed cells of the part. This irritation causes the cells to proliferate.

According to Weigert's well-known theory such an explanation would not be tenable. The cause of the proliferation of the cells should be sought rather in an injury to the tissue which removes the inhibitory force that normally holds in check the power of growth of the cells. Neither Baumgarten nor the other writers have concerned themselves with the existence of such a primary injury.

Wechsberg [9] studied this problem in Weigert's laboratory. Suspensions of tubercle bacilli were injected into the ear veins of rabbits. The animals were killed at different periods ranging from six hours to twelve days and the lungs hardened and sectioned.

At the end of six hours the blood-vessels showed in places a loss of their endothelium as well as a marked destruction of their elastic fibers. Groups of bacilli lay in the lumina of the vessels surrounded by polynuclear leucocytes. After twenty-four hours epithelioid cells were found. The destruction of elastic tissue was the more extensive the longer the duration of the infection.

Wechsberg concludes that the tubercle bacillus destroys the fixed cells and the intercellular tissue by its toxic action. It then injures the new formed cells so that they are unable to produce connective tissue and blood-vessels. The formation of giant cells indicates that the protoplasm of the proliferated cells is only injured in part. Finally the new formed cells become entirely destroyed and the stage of caseation is reached.

Recently an elaborate monograph on the mode of action of the tubercle bacillus has been written by Herxheimer,[10] one of Weigert's assistants. It is, in a sense, a continuation of the study begun by Wechsberg. Tubercle bacilli suspended in fluid were injected into the trachea of rabbits and guinea pigs. The animals were killed at different intervals, from one-half hour to seven weeks after the injection. The staining method, which gave the best results, was a combination of Weigert's elastic tissue stain and anilin-water-methyl-violet. The tubercle bacilli were colored blue and stood out sharply upon the red background.

(1) Stain the sections in lithium carmine several minutes.

(2) Differentiate in 1% hydrochloric acid alcohol several hours.

(3) Stain in Weigert's elastic stain one hour.

(4) Wash rapidly in acid alcohol.

(5) Differentiate quickly in 96% alcohol.

(6) Stain in anilin-methyl-violet several hours in the cold.

(7) Differentiate in acid alcohol and in 96% alcohol.

(8) Dehydrate in absolute alcohol.

(9) Clear in xylol.

(10) Mount in balsam.

In order to remove the violet color from the celloidin, the sections after treating with absolute alcohol were dipped in an alcohol-ether mixture which dissolves away the celloidin.

The bacilli, as soon as they reached the lung, were taken up by epithelial cells lying free in the alveoli and by cells still attached to the alveolar wall. Desquamation of the epithelial cells soon followed the ingestion of the bacilli. At the same time the bacilli exert a destructive influence upon the elastic fibres. A result of the injurious action upon the cells and the intercellular substance is a proliferation of the fixed elements, a growth of epithelium occurs as well as of endothelium and connective tissue cells.

TUBERCULOSIS CONVEYED BY BOOKS.

A careful study has been made by Mitulescu [11] in Koch's Institute in regard to the possibility of the spread of tuberculosis by means of library books. A large number of old, soiled books, chiefly works of fiction, were examined. Of the books which had been in use over two years, one third were infected with tubercle bacilli. The pages were frequently stuck together by filth and the retained moisture prevented the tubercle bacilli drying. On clean paper drying quickly killed them. None of the books in use less than two years harbored tubercle bacilli, and the covers of the infected books were free from these microorganisms. The presence of the tubercle bacilli was determined by inoculating guinea pigs. The pages and covers of the books were thoroughly cleaned with saline solution, the washings centrifugalized and the sediment injected into guinea pigs.

CHRONIC MYOCARDITIS IN CHILDREN.

Acute myocarditis may develop into chronic myocarditis, or the latter condition may develop independently of the acute process. The acute infectious diseases are the most frequent cause of acute myocarditis, and on account of the greater frequency of these diseases in children, acute myocarditis is more common among them than adults. But it is far from common, even among children. In the autopsy records of the Children's Hospital in Vienna during the last ten years are only five cases of acute myocarditis; three followed diphtheria, two occurred after apparently trivial superficial ulcerative processes, in which other signs of sepsis were lacking. Observations at the bedside indicate that acute myocarditis, especially after diphtheria, is not rare, and recovery occurs even from the

[9] Ziegler's Beiträge, 1901, xxix, p. 203.
[10] Ziegler's Beiträge, 1903, xxxiii, p. 303.
[11] Zeitschrift fur Hygiene, 1903, xliv, p. 397.

severe forms. Under what condition chronic myocarditis is established we do not know, but it is certainly one of the rarer diseases in childhood, and plays a far less important rôle than in the cardiac pathology of adults. This is probably due to the freedom from arterio-sclerosis which so frequently in later life cripples the power of the heart; and chronic alcoholic intoxication, with its resulting injury to the heart, rarely has to be considered.

Two cases are reported by Zuppinger.[12] In the first, signs of acute myocarditis developed during diphtheria, and death occurred from chronic myocarditis six weeks later, with symptoms of cardiac insufficiency. The left ventricle measured 2 cm. in thickness. It was dilated and in the wall were many areas of fibrous tissue. In the second case there was great hypertrophy and dilatation and a mural thrombus was situated at the apex of the right ventricle. At this point the wall contained fibrous tissue, but it was not demonstrable elsewhere. The boy aged ten had an attack of measles four years before and pneumonia one year after the measles.

THE NATURE OF PERNICIOUS ANEMIA.

Bloch [13] has been able to add no experimental evidence in support of the modern theory, so widely held, that pernicious anemia is the result of absorption of toxins from the alimentary tract. Injection of fecal extracts into animals caused only slight anemia. A special toxicity of the urine or blood serum in pernicious anemia could not be demonstrated.

The buccal lesions which Hunter regards as an etiological factor are secondary to necrosis from subcutaneous hemorrhage and hence without significance.

Reports of Societies.

THE MEDICAL ASSOCIATION OF THE GREATER CITY OF NEW YORK.

Stated meeting, March 14, 1904. The President, Thomas E. Satterthwaite, M.D., in the chair.

Dr. Robert Coleman Kemp read a paper entitled

OBSERVATIONS ON DILATATION OF THE STOMACH AND ON GASTROPTOSIS,

which was illustrated by colored diagrams made from forty cases in the Manhattan State Hospitals, Ward's Island. It had unfortunately too often been the habit, he said, to depend on the examination of the stomach contents alone, and to neglect the investigation of the motor functions and the position of the stomach itself. The latter is necessary in order to arrive at a correct prognosis and undertake proper treatment.

Atony of the Stomach. — This must necessarily first occur before dilatation of the stomach is produced. Atonia gastrica may be defined as a loss of tone or contractile power in the muscles of the stomach, so that the organ becomes distended and does not contract about its contents, with a resulting motor insufficiency. While the dilatation of the stomach in gastroptosis is disputed by some, his own observations have shown that dilatation does exist, and with it, necessarily, a varying degree of motor insufficiency.

Dilatation of the Stomach. — Boas has demonstrated that an apparently dilated stomach may really be in a condition of compensatory hypertrophy. It may, therefore, be stated that as long as the functions of the organ are normal, the condition met with cannot be regarded as pathological. In dilatation the lesser curvature maintains in general its relation to the diaphragm, and this is the differential point between dilatation and gastroptosis. A number of forms of acute dilatation have been described. The first is the result of acute inflammation of the gastric mucous membrane. It is uncertain whether it depends on pyloric spasm or paralysis of the gastric muscles, or on both these causes. The second is a post-operative dilatation, following abdominal section especially. It would seem to be due to some shock of the sympathetic system, either from the operation or the anesthetic, or possibly, later, from uremia or sepsis. Among the other types of acute dilatation mentioned were the following: In typhoid fever; during attacks of migraine and just preceding epileptic seizures in certain instances; during the course of pneumonia and other pulmonary diseases; in convulsions in infants and young children from over-loading the stomach; in attacks of pseudo-angina pectoris from indiscretions in diet.

Chronic Dilatation. — Over-feeding and chronic indigestion are undoubtedly causes of dilatation of the stomach in infants and young children. Holt believes intestinal putrefaction exciting convulsions in young children to be an important factor in the production of epilepsy. Dr. Kemp regards acute dilatation of the stomach with convulsions as an equally powerful factor, when frequently repeated, in producing the convulsive habit, and thinks that some cases of epilepsy can certainly be attributed to this cause. Moreover, chronic dilatation of the stomach is itself one of the causes of intestinal putrefaction and disturbance. Among other etiological factors of chronic dilatation are chronic gastritis, atony of the stomach, rapid bolting of food, spasm of the pylorus due to gastric ulcer, and benign and malignant pyloric stenosis. Each case should receive the special treatment called for by the existing condition. Dr. Kemp washes out the stomach often only in cases in which there is chronic gastritis with the production of considerable mucus, or where there is much retained food with fermentation.

Gastroptosis. — If gastroptosis exists, some enteroptosis necessarily accompanies it. The determination of the position of the stomach, in order to secure the best kind of treatment, is of great importance, and trans-illumination of the stomach he considers the ideal method for accurate diagnosis. The treatment consists in the following measures:

(1) The correction of the functional derangement of the stomach by appropriate diet and medication.

(2) Regulation of the bowels.

(3) Mechanical support to increase the intra-abdominal tension, such as (*a*) various silk abdominal supporters; (*b*) bandages (as the Van Valzah-Hayes method); (*c*) Gallant's corset; (*d*) Rose's plaster belt. He had seen some brilliant results from the last named. Recently he had employed for this belt rubber plaster and moleskin. The sweat evaporates through it on account of its loose texture, and it can be worn for quite a long time with practically no irritation.

Cases at the Manhattan State Hospitals, Ward's Island. — The drawings Dr. Kemp presented were made from forty epileptics in these institutions. The anatomical regions were marked out in blue pencil

[12] Archiv. fur Kinderheilkunde, 1903, xxxv, p. 381.
[13] Deutsches Archiv. fur klin. Medizin, lxxvii, nos. 3 and 4.