STATED MEETING, HELD MAY 5, 1913.

GEORGE G. ROSS, M.D., in the Chair.

ACUTE GASTRIC DILATATION FOLLOWING OPERATION FOR HERNIA.

DR. JOHN SPEESE presented a man, fifty-six years of age, who was operated upon at the Presbyterian Hospital for an incomplete inguinal hernia by the Bassini method. The patient recovered consciousness readily and experienced but slight nausea from the ether. Thirty-four hours later, after drinking a small quantity of milk, he complained of nausea, which gradually became more severe, and was followed by vomiting two hours later. Temporary relief was experienced, and no alarm was felt as the temperature, pulse and leucocytic count were normal and the abdominal muscles relaxed. The vomiting recurred, the fluid was dark in color, odorless and was ejected in small amounts without effort every few minutes. The patient then developed considerable abdominal pain, and on examining the abdomen at this time, the stomach was found greatly dilated, extending to the umbilicus, and partially filled with fluid, as determined by a succussion splash. A stomach tube was passed and several quarts of dark fluid withdrawn, and lavage performed. An abdominal binder was applied and the patient turned on his abdomen, a position he could assume only for short periods.

He was entirely relieved of the pain and vomiting for six hours, when the latter recurred in a less severe form. The same treatment was carried out and permanently relieved the condition. Examination of the stomach twelve hours after the vomiting began, disclosed a normal state of affairs, the dilatation having perfectly subsided. The patient was given salt solution by rectum to relieve his thirst, and small quantities of water by mouth were allowed twelve hours later without causing any disturbance.

Dr. Speese remarked that acute dilatation of the stomach may follow any variety of operation, although it appears to be relatively uncommon as a post-operative complication of hernia. Lyle (*N. Y. Med. Jour.*, 1911, xciv, 932) observed a case follow-

ing the radical cure of a hernia, the operation was performed under local anæsthesia, and a large amount of omentum was resected. The dilatation began one hour after the operation, and in all probability was caused by the removal of the large amount of omentum which profoundly altered the circulation and possibly the nervous mechanism of the stomach, thus causing a temporary muscular paralysis.

The finding, at post-mortem, of constriction of the duodenum by the superior mesenteric artery at the point where it crosses the intestine, has naturally caused this factor to be regarded as of much importance in the etiology of the affection. That the constriction can be caused by an abnormally long mesentery or by ptosis seems unlikely, and is denied by Mathieu (*Arch. d. Mal. d l' App. Dig.*, 1911, v, 409) who regards the gastric dilatation as the primary factor. Distention of the stomach must take place in a downward direction, the intestines are compressed, the superior mesenteric artery is put on a stretch and the duodenum is thus constricted. The primary dilatation is caused by aërophagia, which is the result of the constant swallowing which follows the nausea and pharyngeal irritation induced by ether. This view seems feasible in the case reported as the patient complained of annoying thirst soon after his recovery from the ether and the constant swallowing must have been accompanied by the passage of a considerable quantity of air into the stomach. The dryness and irritation of the pharyngeal mucosa were moreover increased by a chronic nasal obstruction which made mouth breathing a necessity.

Dr. Edward B. Hodge said that recently, in the case of a woman, fifty-seven years of age, who developed pneumonia one week after operation for appendicitis, temperature was normal for two days, at which time she developed acute dilatation of the stomach; this was relieved after several lavages, but the patient finally succumbed to acute nephritis.

Dr. George G. Ross reported a case of dilatation of the stomach which occurred about the seventh day after an operation in an otherwise healthy young woman who had pus tubes. She had done very well until the night of the second day when she drank the contents of an ice-bag (about one quart). The next morning she had acute dilatation from which she promptly died in spite of all treatment; the water from the ice-bag being the supposed cause.

RETROPERITONEAL ABSCESS.

DR. SPEESE reported the history of a child, eight years of age, who was admitted to Dr. Jopson's service at the Presbyterian Hospital, complaining of pain in the right wrist and left thigh. One week before he had been struck over the left hip, and in several hours the leg became stiff. This increased to such an extent that in three days the boy could not walk, and the thigh became very tender on pressure. The wrist also began to pain considerably and was slightly swollen.

September 5, 1912, four days after the accident, when admitted he complained of abdominal distention, pain and swelling over the lower end of the radius. The region about the hip was very tender, there was slight swelling on the outer side of the femoral vessels, and pressure over the trochanter caused pain. The temperature was 103°, W. B. C. 15,000. Exploration over the trochanter by Dr. Speese revealed normal bone and no evidence of pus. The lower end of the radius was exposed and free pus was found under the periosteum which was raised, the cortex of the radius was chiselled and several drops of pus found in the medullary cavity.

September 7, 1912, the pain in the hip region continued, and the abdomen was markedly distended, tender, but not rigid. W. B. C. 21,000.

September 9, 1912, an area of fluctuation was detected in the left groin, below Poupart's ligament and to the inner side of the femoral vessels. A large amount of pus containing blood clot was evacuated, and on exploring the abscess cavity it was found to extend behind the peritoneum for a distance of four or five inches. The abdominal distention disappeared after evacuation of the abscess.

The cavity was drained, and closed slowly as the convalescence was prolonged by the formation of several pyæmic abscesses. The bacteriological examination of the pus showed staphylococcus aureus.

Instances of retroperitoneal abscess of this type are uncommon, and may cause considerable difficulty in diagnosis because of the symptoms suggesting an acute inflammation of the abdominal viscera. The origin of the abscess is not easy to explain, it seems rational to suppose that the primary process consisted in the formation of a hæmatoma, the result of the

injury sustained. Subsequent infection occurred by bacteria derived either from the intestine or from the blood stream and abscess formation resulted. The same sequence has been observed in rupture of the liver or kidney, followed by hemorrhage into the retroperitoneal tissues.

Diffuse inflammation of the retroperitoneal tissues with pus formation may follow suppurative infections of the appendix, gall-bladder or pancreas. Sprengel (*Arch. Klin. Chir.*, 1912, c, 382) in a recent contribution has thoroughly reviewed the subject, and believes that the general peritoneal cavity in some instances may be uninvolved after appendicitis, for example, while a diffuse suppurative process may attack the retroperitoneal space. As a general rule the peritoneum acts as a barrier and prevents rupture of the pus into the abdominal cavity, but œdema of the mesentery and intestines, and a serous or blood-tinged effusion may result from the infection, and prove misleading when the abdomen is opened.

Sprengel refers to a case which resembles the one reported in many respects. The patient, a woman sixty years of age, sustained a fracture of the neck of the femur, and ten days later was seized with an attack of pain, vomiting and abdominal distention. The patient collapsed and death resulted on the eleventh day. The autopsy disclosed a suppurative para-proctitis which was regarded as the primary infection, and from this a fatal peritonitis arose. The rectal mucosa did not show any trace of injury from which the infection might have started.

The origin and formation of many of these infections is not clear. In children the diagnosis is difficult because of the possibility of an acute osteomyelitis of the femur, and because of the abdominal symptoms produced by pressure of the retroperitoneal effusion. That the disease is not so uncommon may be judged from the occurrence of this case, a second one a short time afterward in Dr. Rodman's service, and several of similar nature in Dr. Wharton's wards at the Children's Hospital.

Dr. J. Stewart Rodman recalled the particulars of a case which occurred at the Presbyterian Hospital, in the service of Dr. W. L. Rodman. The patient was a boy of ten who had been run over by a wagon three weeks previous to his admission to the hospital, thus giving a history of trauma more or less remote. The diagnosis was not altogether clear at first, as tenderness and

fixation of the left hip suggested an acute tubercular arthritis. Later on, however, an easily palpable tumor appeared above Poupart's ligament on the left side which was quite tender to touch and accompanied by a septic temperature. The diagnosis of retroperitoneal abscess was now quite clear. This was opened extraperitoneally and the boy afterward made a rapid recovery.

PERFORATION OF ILEUM BY A FOREIGN BODY FOLLOWED BY SYMPTOMS SIMULATING APPENDICITIS.

DR. SPEESE recited the history of a man, aged fifty-three, who was admitted to Dr. Jopson's service at the Presbyterian Hospital with the following history: Two days previously he experienced a sudden and severe pain in the right iliac region, this was accompanied by nausea and vomiting. The pain which continued until his admission to the hospital, gradually became more severe, the nausea persisted but the vomiting ceased; the bowels had not moved for two days. The leucocytic count was 24,500, pulse 92, temperature 99.4°. The abdomen was slightly distended, and on palpation the right rectus muscle was rigid, and pronounced tenderness was present over McBurney's point.

A diagnosis of acute appendicitis was made and the operation which was at once performed by Dr. Speese revealed many recent adhesions about the cæcum, a small amount of turbid fluid and fibrin, but a normal appendix. Believing that an inflamed Meckel's diverticulum might have caused the condition, the ileum was drawn out of the wound and examined. About four inches from the ileo-cæcal junction a foreign body was felt in the lumen of the bowel, one end being firmly embedded in the intestinal wall almost penetrating the serous coat. The body was extracted and was found to consist of a flat piece of bone, 4 x 1 cm., resembling a sequestrum, one end was splintered and as sharp as a needle. The intestinal wound was closed, examination of the surrounding coils of intestine did not reveal any evidence of perforation. The local peritonitis present was regarded as the result of a puncture of the intestine by the foreign body, the opening being small was soon closed by plastic lymph making its site difficult to discover. The abdomen was drained, convalescence was uninterrupted.

The patient when questioned was unable to remember that he had swallowed a piece of bone, but as he used alcohol to

excess, it seemed likely that the accident occurred during one of his frequent debauches, from one of which he was recovering at the time of his admission to the hospital.

Dr. George G. Ross reported a case seen by him which was diagnosed as acute appendicitis; there was tenderness rather higher up than usual. At operation he found the appendix inflamed, but also noticed that on the outer wall of the cæcum there was a great deal more inflammatory lymph than on the inner. In pulling up the cæcum a tooth pick floated up into the wound and was removed. It seemed as if the tooth pick had gone through the ileocæcal valve and had pinned itself on the opposite wall of the cæcum and had ulcerated through.

Dr. Edward B. Hodge said that he saw a patient last summer which resembled Dr. Speese's case very closely: A boy of four years came to the Children's Hospital. It was thought that he had appendicitis; he had the usual symptoms and had had two attacks previously of colicky pain which had been diagnosed as appendicitis by the family physician. Operation revealed some adhesions in the right iliac fossa and some free dirty fluid; no pus. Appendix looked all right. Further search detected a stricture of the ileum about one foot from the ileocæcal valve and a perforation above through which a piece of pickle was protruding. He did not think the pickle made the puncture but the boy had perforation and a very tight stricture. A resection was done and the patient eventually got well.

Dr. Morris Booth Miller raised the question of perforation from foreign bodies. This matter has never been definitely worked out, *i.e.*, why certain objects cause perforation and others do not. All are familiar with the fact that children often have passed through their intestinal tract extraordinary substances, such as coins, pins, buttons and various materials of similar type. The great majority of these pass through without disturbance, but occasionally serious trauma is inflicted.

Twice he had seen perforation caused by a piece of bone. Several years ago he operated upon a woman who had all the symptoms of acute appendicitis. When opened it was found that she had a perforation of the appendiceal tip due to a piece of chicken bone, which was hardly larger than one-half the size of an ordinary pin. Again, about a year ago he had an experience with a woman, age fifty-five, who had previously under-

gone a hysterectomy. Following this she had had an incisional hernia which was characterized by adhesions. She was suddenly taken ill with pain, local tenderness in the hernial sac and vomiting. At operation an irreducible hernia was found but not obstruction. Within the sac a piece of chicken bone over an inch long had perforated the gut with some local contamination. It was hoped she would get well, but as she was diabetic she died from coma in two or three days. There was no generalized peritonitis.

(1) PERFORATED MECKEL'S DIVERTICULUM, (2) TWO INDEPENDENT SACS IN AN INGUINAL HERNIA, (3) REMOVAL OF PARAFFINE FROM INGUINAL CANAL IN CASE OF INGUINAL HERNIA.

DR. A. BRUCE GILL reported the following cases from the service of Dr. Hodge at the Presbyterian Hospital:

CASE I.—A school-boy, aged thirteen years, admitted to the ward on the evening of March 4, 1913. For a week previous he had been having crampy pains about the umbilicus that occurred for the most part during the afternoon while the patient was in school. On the day of his admission about two o'clock in the afternoon while in school, he was seized with a sudden severe pain about the umbilicus and in the lower right part of the abdomen. He vomited repeatedly and was carried home from school.

On examination at ten o'clock P.M. there was present general abdominal rigidity which was most marked in the lower right quadrant. Pain and tenderness were chiefly in the same quadrant. The patient lay on his left side with the knees drawn up. On preparing the abdomen for operation a small red papule at the umbilicus bled slightly on being scrubbed.

The mother stated later that he had bled from the umbilicus in infancy, and the patient himself had observed a red spot at the umbilicus that would bleed on irritation.

Operation was performed at 10.25 P.M. The abdomen was filled with bloody serum. The appendix was long and kinked, but not diseased. A Meckel's diverticulum was found coming from the ileum about two inches from the ileo-cæcal junction. It was about two inches long and as large as the middle finger. A cord three inches long extended from the end of the diver-

ticulum to the umbilicus. The diverticulum was highly congested and was thickened. Near its base was an annular thickening and constriction. On the distal edge of the dense ring was a small perforation filled with a blood clot.

With the exception of enlarged glands in the transverse mesocolon there was no other evidence of abdominal disease.

The cord attached to the diverticulum was ligated and divided near the umbilicus. The diverticulum was excised and its stump was inverted by a purse-string suture reinforced by Lembert's sutures. The appendix was removed. The abdomen was freely flushed with hot salt solution and a drain was placed in the pelvis.

Recovery was uninterrupted and the patient was discharged on March 21, 1913.

CASE II.—Man, aged fifty-five years. Four years ago he suddenly developed a right inguinal hernia. He was stacking soap-boxes weighing 100 pounds each. While he had the seventh box above his head and was about to place it upon the pile, his right foot trod upon a potato and he slipped suddenly backward. The patient immediately felt a sharp nauseating pain in his right groin, and shortly afterward he noticed a soft swelling in the same region. The hernia has slowly increased in size to the present time. He has latterly worn a truss that did not restrain the hernia but did cause considerable pain. The hernia was reducible.

At operation a hernial opening large enough to admit the forefinger was found internal to the cord and one inch above the external ring. Protruding about two inches through the opening was a pouch of transversalis fascia which contained preperitoneal fat and a thin empty peritoneal sac. The sac was drawn down, ligated, and excised, and its stump was transposed upward and inward and fastened behind the rectus muscle. Upon examination of the cord a second hernial sac was found coming out of the internal ring. It was freed from adherent veins, opened, and found to be empty. The sac was excised and the stump disappeared into the abdomen. The operation was completed according to the Bassini method, but with the addition of one suture that fastened the edge of the rectus fascia to Poupart's ligament near the pubic spine, and a second suture external to the internal ring.

Recovery was uneventful. The patient was seen recently, which is more than a year since the operation. There has been no recurrence of the hernia, although he returned to his former work and has been doing heavy lifting.

CASE III.—Man, aged forty-eight years. He developed a right inguinal hernia seven years ago. Four years ago paraffine was injected at the side of the hernia by some person unknown. For eight months following this the hernia seemed cured, but at the end of that time it recurred and gradually increased in size. Several masses of paraffine could be felt beneath the skin and within the inguinal canal.

At operation one mass of paraffine was found imbedded in the aponeurosis of the external oblique in front of the canal, another lay beneath the skin above the canal, several pieces, including the largest, were within the canal. The vas was firmly adherent to three separate masses of paraffine and was dissected free with difficulty. The aponeurosis in front of the canal was dense and thick, and structures were obscured by the fibrous tissue about the masses of paraffine. The canal was exposed by splitting the external oblique above the canal and not in front of it and dissecting the lower flap downward. The largest piece of paraffine was near the internal ring, and its fibrous capsule was continuous with a mass of dense tissue that appeared to be an old obliterated sac. All the pieces of paraffine were removed.

A thin, wide-mouthed, empty sac was found posterior and internal to the cord and to the old obliterated sac. It was excised and its stump was transplanted upward and inward and fixed behind the rectus. The conjoined tendon and the edge of the rectus were sutured to Poupart's ligament in front of the cord. The upper flap of the aponeurosis of the external oblique was also sutured to Poupart's ligament, and the lower flap overlapped it and was sutured above.

DR. ASTLEY P. C. ASHHURST said that Dr. Gill's case of two sacs in an inguinal hernia reminded him of a similar sac found this winter while operating on a cadaver in the University. There were two sacs in the canal and extending down beyond the external ring; each had a separate opening into the peritoneal cavity at the internal ring. They were excised and sent to Dr. C. Y. White at the Episcopal Hospital for examination. He reported that both sacs were lined with endothelium appar-

ently identical. The existence of adventitious bursæ which are not very rare in the wall of a hernial sac is easily explained, but he knew of no satisfactory explanation for two distinct sacs, unless both are congenital.

As regards Meckel's diverticulum, he had seen three cases. The first case was reported to the Academy some years ago. There was an internal hernia through a rent in the mesentery of the ileum. The patient was a child, aged twelve, and, as he had learned that if in children from thirteen to fifteen years of age, the umbilicus appears abnormal, one should suspect the existence of a Meckel's diverticulum, the opinion was hazarded before operation that this might be the origin of the obstruction.

Only a month later he saw a man of forty-six years with symptoms of intestinal obstruction. He also had an abnormal umbilicus; four spots could be seen where the fetal structures had come through. In this case the diverticulum was found adherent to the umbilicus on the inside and twisted on itself. Unfortunately, the man died, in spite of relief of the obstruction.

Another case of Meckel's diverticulum he saw in a patient with typhoid perforation. The perforation occurred in the ileum and in looking for more perforations he found a slightly inflamed but non-adherent diverticulum higher up. This patient, who was eighteen years of age, had an abnormal umbilicus.

Dr. Addinell Hewson said that recently, in his service at the hospital, a case came in with symptoms suggesting acute obstruction. Operation was performed and it was found that a Meckel's diverticulum was present and located about 2 feet from the ileocæcal valve, was twisted around the intestine and caused complete obstruction. This case was not in a young person, but in a man of mature age who made an uneventful recovery.

SUTURE OF THE HEART.

Dr. Arthur E. Billings reported the case of a man, thirty-one years old, who was brought to the Pennsylvania Hospital, at 2.55 A.M. on May 7, 1911, and was admitted to the service of Dr. Hutchinson, to whom the reporter was greatly indebted for the privilege of operating upon and reporting this case.

The patient's previous history was negative except for marked alcoholism for the last two years. Present condition: While

walking through the east side of Franklin Square he was attacked by a man who stabbed him and fled. He was found by a policeman lying in an alley between Fifth and Sixth and between Race and Vine Streets, from where he was brought to the Pennsylvania Hospital by the patrol wagon. He was unconscious at the time of his admission, presenting the picture of extreme shock with hemorrhage. Temperature was 95°, breathing labored and sighing in character, radial pulse was imperceptible, and skin was clammy and cold. On examination of his thorax, there was a wound about 2 cm. in length in the midclavicular line of the left fifth interspace which was bleeding quite freely with respiratory movements. Percussion and auscultation revealed signs of a marked hæmopneumothorax. The heart sounds were irregular and distant, rapid, and at times almost inaudible.

Operation.—Local preparation consisted of shaving and the application of tincture of iodine to the field of operation. A semilunar incision was made over the left fourth and fifth ribs with the convexity toward the sternum. The fourth and fifth ribs were then severed near the costal border and turned outward about 5 cm. away from the sternal border forming a trap door. The left pleural cavity was filled with blood and the pericardium showed a wound about 1.5 cm. in length and was distended with blood. This opening was increased in length exposing the heart which revealed a wound in the left ventricle between 2 and 3 cm. in length on the external surface and slightly triangular in character, which was bleeding profusely with each systole, and was partially controlled by digital pressure during suturing.

This was closed with five interrupted sutures of No. 1 iodized catgut. The pericardium was closed with a continuous No. 1 iodized catgut suture, without drainage. In the lower lobe of the left lung on its anterior surface, there was a ragged wound about 4 cm. in length which continued to bleed slowly and persistently. The sixth rib was then resected in the left midaxillary line and a small gauze pack was introduced to control bleeding from the lung. The trap door was then closed, the ribs being sutured in place with No. 5 iodized catgut and the skin closed with interrupted silkworm gut sutures without drainage. During the first part of the operation, an anæsthetic was not necessary, ether

being used during the latter part of it. During the operation the patient received twenty-four ounces of normal salt solution, intravenously, and strychnine sulph., grains 1/20 hypodermically. After the operation the reaction was fairly prompt and surprisingly good. Pulse one hour after operation was 88 and temperature 96.2°.

During the following twelve hours, temperature reached 102.2°, soon dropping to normal with pulse ranging from 88 to 120, and the patient rational. Thirty-six hours after operation, patient's temperature began to go up, reaching 101.1°, pulse 120 to 140, with much respiratory distress and increasing cough with gradual circulatory failure, death resulting about forty-eight hours after operation. Cardiac compression is suggested in this case because of the marked increase in bleeding after opening the pericardium and improvement in patient's condition at this time.

Autopsy revealed extensive pleurisy and complete solidification of the already collapsed left lung with slight pericarditis about the line of incision and wound.

SPLENIC ANÆMIA, WITH SPECIAL REFERENCE TO ETIOLOGY AND SURGICAL TREATMENT.

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AND

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(Experimental work from the Laboratory for Research, Medico-Chirurgical College.)

SPLENIC anæmia, although recognized as a pathologic entity even in the early years of the nineteenth century, presents to-day many unsolved problems. This condition has been described under various names during the past twenty-five years but at the present time splenic anæmia is the generally accepted term. By it we mean a disease of long duration, generally lasting several years, characterized by an anæmia of the chlorotic type, considerable enlargement of the spleen without known cause, an absence of leucocystosis and a tendency to gastro-intestinal hemorrhage. In the advanced stage, cirrhosis of the liver, ascites and jaundice are also present. Banti, Osler and others have fully described the course of the disease and divide it into three distinct stages.

The first stage lasts two to ten years, with splenomegaly, anæmia, gastro-intestinal hemorrhage and pigmentation of the skin as its prominent symptoms. The second stage lasting only a few months adds to these symptoms kidney insufficiency with scanty high-colored urine. The third and terminal period presents the stage of liver involvement in which appears a cirrhosis of the Lænnec type with its accompanying ascites.

The name, Banti's disease, has been applied incorrectly to all three stages but it is the symptom-complex of the ascitic

stage alone that should bear the name of the man who did so much to clear up its symptomatology and pathology.

During the first stage, there are usually marked fluctuations in the severity of the symptoms. Months may pass in which slight enlargement of the spleen is the only demonstrable lesion. Again during the exacerbations, the spleen may enlarge enormously, with coincident increase in the anæmia and icterus.

Etiology.—The etiology of splenic anæmia has proved baffling. Much confusion has arisen and the main purpose befogged in endless discussion as to whether or not the splenomegaly is primary or secondary. The mere fact that we have first this splenomegaly is important, but not necessarily convincing that it alone is responsible for the anæmia, the cirrhosis and endophlebitis of the splenic and portal veins. Rather would it seem more reasonable to us that the splenomegaly was a link in the chain which resulted from the chronic irritation of some as yet unknown toxin, probably of gastro-intestinal origin. Having once acquired splenomegaly, we believe that it is responsible for the anæmia by reason of an exaggeration of its normal hæmolytic functions. In other words, the spleen finds itself much in the same position as the thyroid, which, as the result of some irritation, increases its activity and thus brings about hyperthyroidism. It is logical to assume this because we know that splenectomy, if done in the first stage, stops the anæmia with an early return of the blood picture to normal. Here again, comparison with the results of partial thyroidectomy are apt, since there too an excessive physiological function is checked. But we do not have to depend on splenectomy alone to feel sure this is so. Banti in his latest paper states that the anæmia is due to increased hæmolysis chiefly, and secondly to an actual increase in the blood destroying units of the spleen.

Banti, Joannovico, Lemarre and Gaucher have shown that splenectomized dogs have an increased resistance to certain hæmolytic poisons (pyridine, etc.). F. Bartazzi has also shown increase in the red blood corpuscle resistance in

splenectomized dogs. Pagliesi and Tuzzati, later Charrin and Moussie, and later still Gaucher, have shown that splenectomized dogs have a considerable lessening of biliary secretion. The logical conclusion is that the spleen has a great influence on hæmolytic and bilogenic processes not only because it diminishes red cell resistance and prepares them for destruction in other organs, but because it destroys them directly and forms thereby the necessary substances for biliary secretion.

The part that the enlarged spleen plays in the production of the anæmia has given rise to other theories. Harris and Hertzog believe that by reason of an erythrolytic enzyme secreted by the endothelial cells of the hyperplastic spleen there is a destruction of red blood corpuscles, and cite splenectomy with its cure to substantiate them in their position. Barr thinks that an increased blood supply to the spleen, with its consequent fibrosis, causes increased hæmolysis. The congestion is explained on a theory of vasomotor paresis of the splanchnic area from disease of the visceral sympathetic ganglia. Lintvarev believes that the anæmia is due to an increased destruction of the red cells by the erythrophages of the spleen, which cells have been produced in excessive numbers because of certain poisons (exogenous as well as endogenous). Rolleston would explain the anæmia upon an inhibition of blood formation due to the endothelial hyperplasia found in the enlarged spleen. So, while opinions differ as to the actual manner of accomplishment, it seems reasonably certain that the spleen causes the anæmia. Banti's theory of increased hæmolysis brought about by lessened resistance of the red blood corpuscles so that they are destroyed in greater proportion in the liver, lymph nodes and spleen itself, seems to us the most satisfying, since it is based on reliable experimental evidence and clinical findings. In these cases we have icterus without decoloration of the fæces, and we find that in periods of aggravation of the disease a further diminution of the red blood corpuscles and hæmoglobin is coincident with augmentation of icterus and urobilinuria.

The question of the primary intoxication is by no means so definitely settled. Senator was perhaps the first to suggest that the toxin was absorbed from the gastro-intestinal tract. Others have followed accepting this theory, some offering clinical data to substantiate their position. Ostrowski, who states that in ten cases of splenic anæmia in children all had had an antecedent diarrhœa, and Ungar and Neuberg believe that the gastro-intestinal tract must be looked into. It would seem not illogical to assume that the gastro-intestinal tract might furnish the toxin because of the somewhat similar clinical picture that is found in protracted auto-intoxication and in the beginning of splenic anæmia. Banti, however, adhering to the infectious theory, does not believe that the irritant comes from the gastro-intestinal tract, reasoning that the atrophy occurring in the liver from cirrhosis due to auto-intoxication is not produced for years, whereas that occurring in the liver during the third stage of splenic anæmia occurs in a few months. Conflicting ideas also are advanced as to casual relation existing between splenic anæmia and certain diseases, as tuberculosis, syphilis, Kala-Azar, malaria and gout. Banti again thinks that his syndrome, when occurring as the third stage of splenic anæmia at least, is a disease *sui generis*, but does not deny that enlarged spleen, cirrhosis and ascites may be found in the terminal stages of other diseases, as is pointed out by Hultgen, Rolleston and others. Osler thinks that when secondary anæmia and splenomegaly are found in other conditions the differential diagnosis may be made, since (*a*) the cause is usually apparent as malaria, tuberculosis, syphilis, etc., (*b*) the anæmia and splenomegaly usually yield to appropriate treatment, (*c*) the spleen is not so large, (*d*) sequences not so characteristic, (*e*) the blood, while showing secondary anæmia, does not usually show the exaggerated chlorotic type, and (*f*) leucocytosis is more common than leucopænia.

We know that the spleen is not rigidly controlled by nervous influences from the central or sympathetic nervous system, but that its stimulation depends largely upon the

circulation of systemic blood which comes in direct contact with the parenchyma of this organ. Further, that a part of its physiologic function is to sift out from the circulating blood bacteria, toxins and other detritus. These facts offer additional theoretical evidence of the probable toxic nature of the primary irritant.

Certainly one of the most interesting problems to be solved is the connection between endophlebitis of the portal and splenic veins and the enlarged spleen. Is the thrombosis primary or secondary? That some relation exists is reasonably certain, because of the frequency with which thrombosis of the portal and splenic is found at autopsy in cases dying of splenic anæmia. The reports of Dock and Warthin, Oettinger and Fiessinger, Banti, Rolleston and many others amply attest this fact.

It was really to attempt to shed additional light on this phase of the question that led the authors to carry out experiments on animals.[1] Our aim was first to produce thrombosis of the portal and splenic and later to study the splenic changes and blood picture. It seemed easy to ligate the splenic vein and cause a passive congestion of the spleen which would, in a measure, at least reproduce what actually occurred in cases of portal and splenic thrombosis. This we did in seven dogs, and found that in every case the immediate enlargement of the spleen was enormous, but in none of our cases did this increase in splenic dimensions last longer than one month, to be followed by permanent atrophy of the organ. The results coincide with Warthin's earlier ligations of the splenic vein. In another series of three dogs and two rabbits ligation of the splenic veins and injection of aleuronat directly into the splenic pulp were tried. Here again, in every instance, temporary increase in the size of the spleen followed with local necrosis of the splenic pulp at the injection sites. The splenic congestion with its consequent enlargement gave way to permanent atrophy. The aleuronat was used to supply an irritant and was chosen because we wished to produce an inflammation that would stop short of pus formation. Feeling that ligation, with its sudden congestion, did not reproduce the actual pathological condition, we attempted partially to occlude the lumen of the splenic vein. This we did by metal clips and loosely tied ligatures in a series of three dogs. Slight enlargement of the spleen occurred temporarily, to be followed in three weeks by a return to the normal size. Having failed with the splenic we next tried, in a series of five dogs, to produce portal thrombosis by exposing the vein, breaking the intima by compression and partially occluding the lumen

[1] All experimental work on animals done under ether anæsthesia

by means of a silver wire passed around the vein, so as to constrict, but not totally obstruct it. In no case were we able to produce by this means portal thrombosis, although slight temporary enlargements of the spleen were again noticed to measurements.

The pathological studies of the removed spleen following ligation showed passive congestion in the early cases, that is, those removed not later than one month after operation, but no real hyperplasia of the splenic substance. In the later cases, those removed after one month or longer had elapsed, there was atrophy of the splenic pulp and an increase in connective tissue. The blood pictures during the period of splenic enlargement showed no characteristic change beyond a leucocytosis following operation, which returned to the normal after an average period of two weeks had elapsed.

Other experimental work has been done; thus Solowieff produced a fibroid condition of the liver by producing occlusion of the portal branches in dogs. Osler cites a case of his own, however, in which there was obliteration of the portal but no true cirrhosis, although the liver was atrophied and had undergone fatty degeneration to some extent. This case showed enormous enlargement of the spleen with the thrombosis of some of the splenic veins and with that of Cruveilhier, shows that obliteration of the portal can take place in man without cirrhosis of the liver. Banti reports the results of an extensive bacteriological study. Believing that the disease is bacterial in origin, he cultured the peripheral blood, splenic substance, blood of splenic vein and bone marrow in fatal cases without result. Aërobic and anaërobic cultures were both negative. In addition, guinea pigs, rabbits, dogs and rats were injected with the blood, splenic juice, liver and bone marrow without result. Fragments of spleen, liver and bone marrow have been directly injected under skin and into the peritoneal cavity still without infection. Finally Banti grafted pieces of the diseased spleen into the normal spleen of dogs, also without result. He states that similar bacteriological studies by others have always yielded negative findings. Although these experiments have been thoroughly carried out the parasitic origin of the disease has by no means been disproved. The cellular hyperplasia of the spleen in this condition is suggestive of an irritation due to bacterial or at least protozoal growth. Again, in the diseases most closely simulating splenic anæmia, such as tuberculosis and syphilis of the spleen, malaria, etc, specific organisms have been isolated.

Pathology.—Many observers have carefully studied the pathology of splenic anæmia. In the main they agree as to the macroscopic and microscopic picture of the disease. In brief, the essential pathology is first an anæmia of the chlorotic type, the microscope showing marked aniso- and pœcilocytosis, polychromatophilia and often basophilic degeneration of the red cells, with the occasional finding of

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normoblasts and myelocytes. Secondly, an enlargement of the spleen which weighs on an average 61 ounces. It retains its normal shape, is dense and firm and under the microscope shows a general fibrosis, especially of the malpighian bodies, which are sclerosed and atrophied and often show hyaline degeneration. In addition, the blood spaces show marked hyperplasia of the endothelial cells and many large phagocytic cells, spoken of by Lintvarev as macrophages. In the third stage the liver shows a varying degree of atrophic interlobular cirrhosis, closely corresponding to that seen in chronic alcoholism. In addition, in a large majority of cases, there is reported an obliterating thrombophlebitis in various stages of degeneration of the splenic and portal veins, which often show almost complete obliteration of the lumen.

Treatment.—In the treatment of splenic anæmia, as in its etiology, a great diversity of opinions is met with. Medicine and surgery have had their advocates and opponents. With the rapid advance of surgical technic in the past few decades, the operative treatment now offers the greatest chance for permanent recovery.

In the medical treatment, iron and arsenic stand preeminent. They are of undoubted value in combating the anæmia and in improving the general condition, but at the best they are only temporary aids and in no way counteract the causal factor. Up to the present time literature shows no cases of permanent cure by medical treatment alone.

As our knowledge of the etiology of this condition advances, the medical treatment will of a certainty become more valuable, especially in the early stages. If we can but discover the primary intoxicant and attack this before the spleen is permanently damaged, our therapeutic agents will become powerful factors in what may be called the prophylactic treatment of splenic anæmia. At the present time, the early eradication of any chronic source of intoxication, especially of the alimentary tract, must be of general if not of specific benefit to the patient. As an example, a case of one of the authors may be cited.

A man, forty-six years of age, suffering from mild anæmia of two years' duration, with its usual symptoms and with a spleen reaching to the level of the umbilicus, was examined early in the year. His past history was negative with the exception of occasional attacks of gastro-enteritis and chronic nasal catarrh. An examination showed chronic frontal sinus trouble and marked gingivitis with symptoms of gastric and intestinal stasis. After several months of treatment for these conditions there has been a decided improvement as to the blood and spleen, and marked betterment of the patient's subjective symptoms. The case has been under observation for too short a time to definitely decide whether the improvement is to be more than temporary.

The intravenous injection of salvarsan has been used for this condition and its results parallel those of internal medication, *i.e.*, improvement in symptoms without permanent recovery.

Treatment by X-ray has been tried at various times. Lucatello reports a marked improvement in general condition in the blood and in the spleen for a time, but the relief from this, as from medicine, is only temporary.

In taking up the surgical treatment it may be wise to review the topography of the spleen. We are dealing with an organ of the upper abdomen, lying well within the vault of the diaphragm and overlaid anteriorly by the fundus of the stomach and posteriorly by the ribs. It is securely held in place by its ligaments and in the disease under discussion is often additionally fastened to the surrounding organs and parietes by firm adhesions. Its blood-vessels are large and extremely thin-walled, making ligation a serious matter. The splenic pulp is brittle and tears and bleeds easily under rough handling.

With these points in mind, the difficulties of operative procedures can readily be estimated. Proper exposure is the first essential. Mayo, Meyer, Treves and others have suggested various methods of approach, all of which have many points of merit. We would like to call special attention to a modification of the incision described by Dr. Edward Martin

for exposure of the liver and which, in the dissecting room at least, has given us the best access to the upper left quadrant of the abdomen.

The incision used is a reversed " J " (see Figs. 1–5). The vertical portion starts one cm. to the left of the middle line, at the tip of the ensiform cartilage and is continued downward to the level of the umbilicus. It is then carried outward across the rectus and outward and upward toward the tip of the seventh rib. The rectus muscle is cut transversely, preferably at the linea transversa. The external oblique and the transversalis muscles are separated in the direction of their fibres; the internal oblique fibres are cut across. The flap can then be turned up and out giving free access to the splenic area.

We consider that this incision not only gives us the best operative exposure but also greatly lessens the dangers of post-operative hernia. The blood supply of the flap is not interfered with on account of the free anastomosis with the vessels of the chest. The nerves supplying this area, coming as they do from the lower intercostals, run parallel with the transverse portion of the incision and therefore are not cut across. The transverse incision of the rectus has already proven satisfactory and the splitting of two of the three layers of the lateral wall adds greatly to the post-operative strength.

Having obtained the proper exposure, the greatest remaining difficulty lies in the efficient ligation of the vascular pedicle. Mayo and Sutherland both strongly urge the use of a temporary rubber covered clamp on this pedicle before ligation and the thorough testing of the ligatures before the complete removal of the clamp.

Extirpation of the spleen has been performed for various conditions by many operators and the consensus of opinion is that while the operation is a serious one with high mortality, it is entirely justifiable under certain conditions. The statistics for splenectomy from any cause compiled by Bessel Hagen in 1900, and by Johnson in 1908, show a mortality of 36.3 per cent. in a series of 353 cases and of 18.5 per cent. in a series of 355.

FIG. 1.

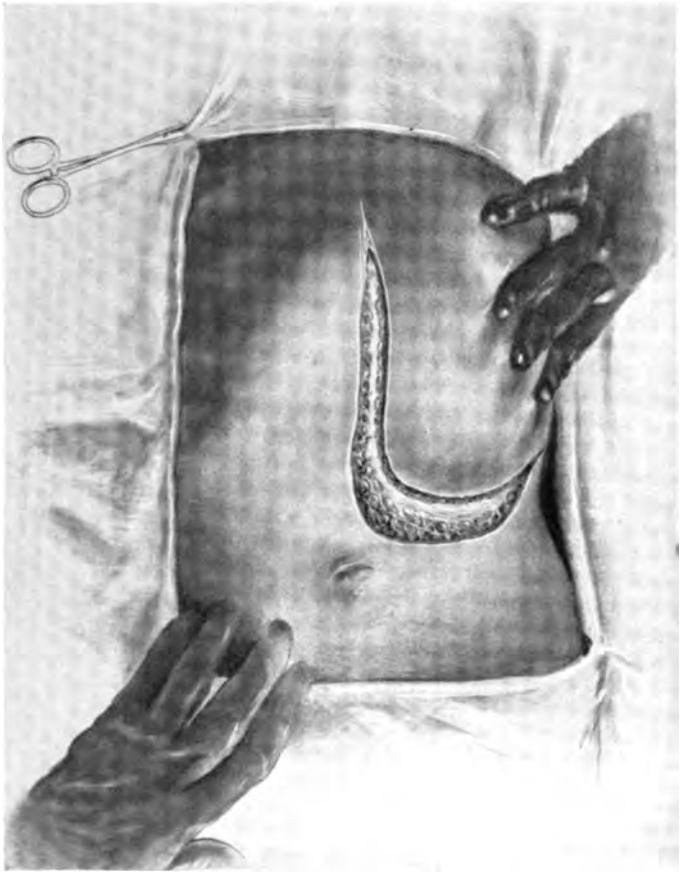


FIG. 2.

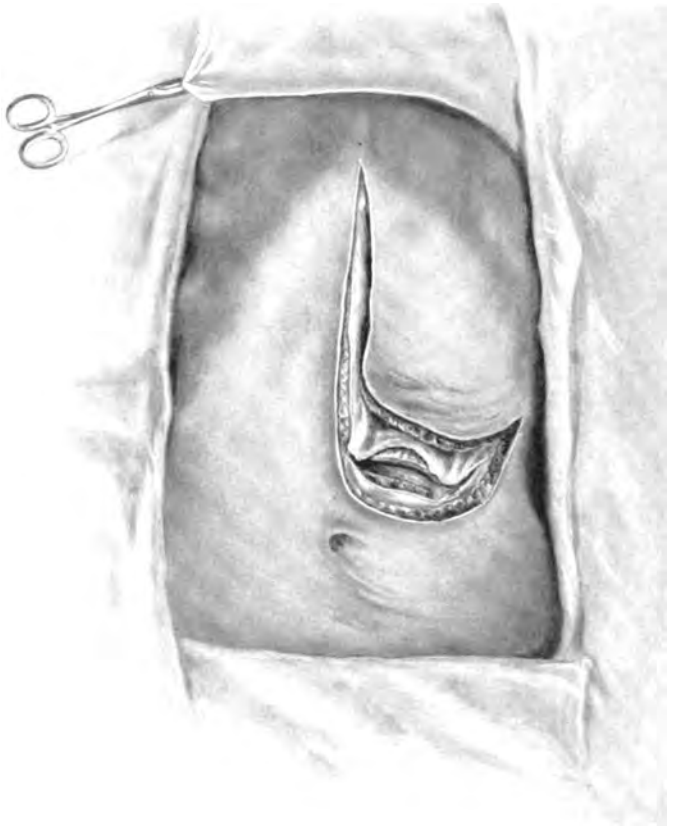


FIG. 3.

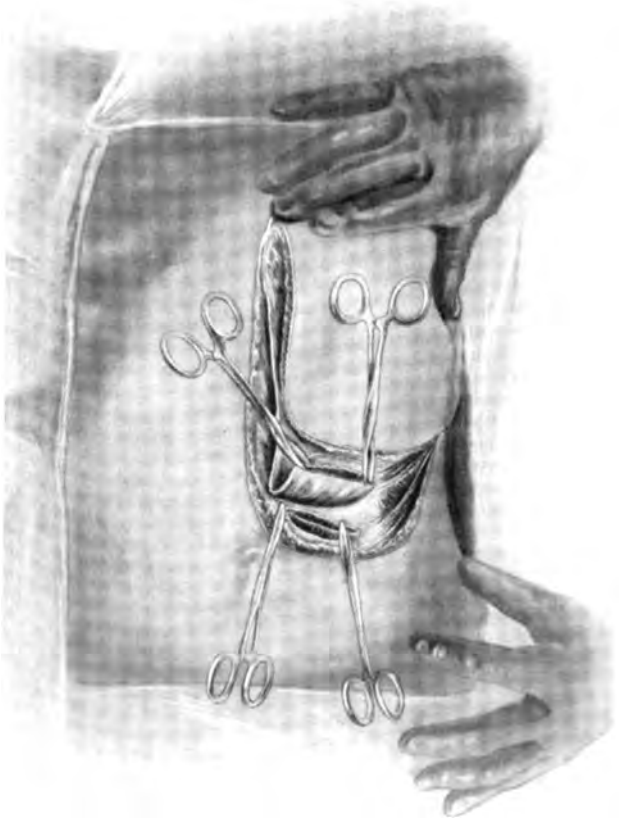
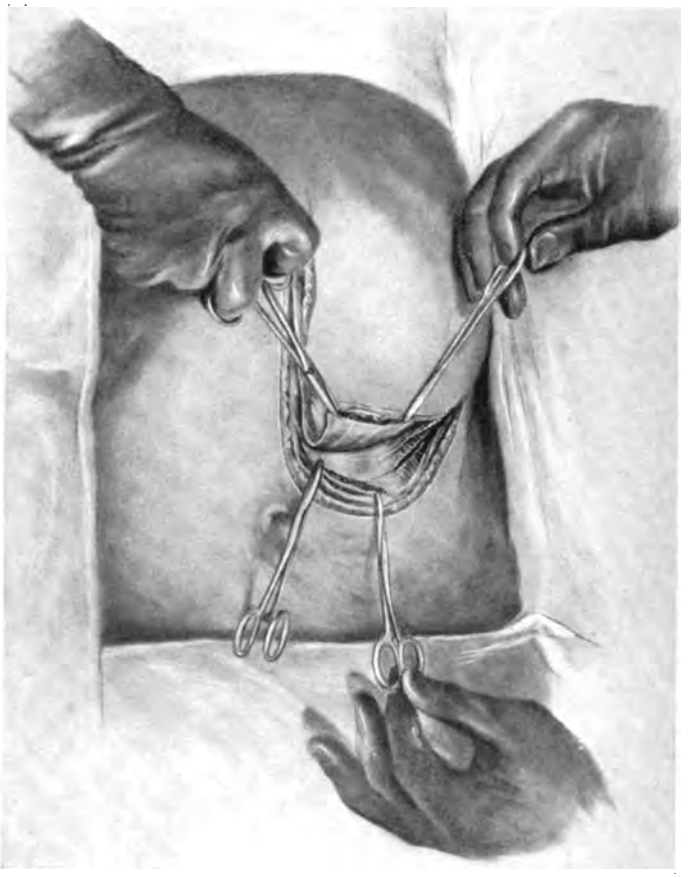


FIG. 4.



STATED MEETING, HELD MAY 5, 1913.

GEORGE G. ROSS, M.D., in the Chair.

ACUTE GASTRIC DILATATION FOLLOWING OPERATION FOR HERNIA.

DR. JOHN SPEESE presented a man, fifty-six years of age, who was operated upon at the Presbyterian Hospital for an incomplete inguinal hernia by the Bassini method. The patient recovered consciousness readily and experienced but slight nausea from the ether. Thirty-four hours later, after drinking a small quantity of milk, he complained of nausea, which gradually became more severe, and was followed by vomiting two hours later. Temporary relief was experienced, and no alarm was felt as the temperature, pulse and leucocytic count were normal and the abdominal muscles relaxed. The vomiting recurred, the fluid was dark in color, odorless and was ejected in small amounts without effort every few minutes. The patient then developed considerable abdominal pain, and on examining the abdomen at this time, the stomach was found greatly dilated, extending to the umbilicus, and partially filled with fluid, as determined by a succussion splash. A stomach tube was passed and several quarts of dark fluid withdrawn, and lavage performed. An abdominal binder was applied and the patient turned on his abdomen, a position he could assume only for short periods.

He was entirely relieved of the pain and vomiting for six hours, when the latter recurred in a less severe form. The same treatment was carried out and permanently relieved the condition. Examination of the stomach twelve hours after the vomiting began, disclosed a normal state of affairs, the dilatation having perfectly subsided. The patient was given salt solution by rectum to relieve his thirst, and small quantities of water by mouth were allowed twelve hours later without causing any disturbance.

Dr. Speese remarked that acute dilatation of the stomach may follow any variety of operation, although it appears to be relatively uncommon as a post-operative complication of hernia. Lyle (*N. Y. Med. Jour.*, 1911, xciv, 932) observed a case follow-

ing the radical cure of a hernia, the operation was performed under local anæsthesia, and a large amount of omentum was resected. The dilatation began one hour after the operation, and in all probability was caused by the removal of the large amount of omentum which profoundly altered the circulation and possibly the nervous mechanism of the stomach, thus causing a temporary muscular paralysis.

The finding, at post-mortem, of constriction of the duodenum by the superior mesenteric artery at the point where it crosses the intestine, has naturally caused this factor to be regarded as of much importance in the etiology of the affection. That the constriction can be caused by an abnormally long mesentery or by ptosis seems unlikely, and is denied by Mathieu (*Arch. d. Mal. d l' App. Dig.*, 1911, v, 409) who regards the gastric dilatation as the primary factor. Distention of the stomach must take place in a downward direction, the intestines are compressed, the superior mesenteric artery is put on a stretch and the duodenum is thus constricted. The primary dilatation is caused by aërophagia, which is the result of the constant swallowing which follows the nausea and pharyngeal irritation induced by ether. This view seems feasible in the case reported as the patient complained of annoying thirst soon after his recovery from the ether and the constant swallowing must have been accompanied by the passage of a considerable quantity of air into the stomach. The dryness and irritation of the pharyngeal mucosa were moreover increased by a chronic nasal obstruction which made mouth breathing a necessity.

Dr. Edward B. Hodge said that recently, in the case of a woman, fifty-seven years of age, who developed pneumonia one week after operation for appendicitis, temperature was normal for two days, at which time she developed acute dilatation of the stomach; this was relieved after several lavages, but the patient finally succumbed to acute nephritis.

Dr. George G. Ross reported a case of dilatation of the stomach which occurred about the seventh day after an operation in an otherwise healthy young woman who had pus tubes. She had done very well until the night of the second day when she drank the contents of an ice-bag (about one quart). The next morning she had acute dilatation from which she promptly died in spite of all treatment; the water from the ice-bag being the supposed cause.

RETROPERITONEAL ABSCESS.

DR. SPEESE reported the history of a child, eight years of age, who was admitted to Dr. Jopson's service at the Presbyterian Hospital, complaining of pain in the right wrist and left thigh. One week before he had been struck over the left hip, and in several hours the leg became stiff. This increased to such an extent that in three days the boy could not walk, and the thigh became very tender on pressure. The wrist also began to pain considerably and was slightly swollen.

September 5, 1912, four days after the accident, when admitted he complained of abdominal distention, pain and swelling over the lower end of the radius. The region about the hip was very tender, there was slight swelling on the outer side of the femoral vessels, and pressure over the trochanter caused pain. The temperature was 103°, W. B. C. 15,000. Exploration over the trochanter by Dr. Speese revealed normal bone and no evidence of pus. The lower end of the radius was exposed and free pus was found under the periosteum which was raised, the cortex of the radius was chiselled and several drops of pus found in the medullary cavity.

September 7, 1912, the pain in the hip region continued, and the abdomen was markedly distended, tender, but not rigid. W. B. C. 21,000.

September 9, 1912, an area of fluctuation was detected in the left groin, below Poupart's ligament and to the inner side of the femoral vessels. A large amount of pus containing blood clot was evacuated, and on exploring the abscess cavity it was found to extend behind the peritoneum for a distance of four or five inches. The abdominal distention disappeared after evacuation of the abscess.

The cavity was drained, and closed slowly as the convalescence was prolonged by the formation of several pyæmic abscesses. The bacteriological examination of the pus showed staphylococcus aureus.

Instances of retroperitoneal abscess of this type are uncommon, and may cause considerable difficulty in diagnosis because of the symptoms suggesting an acute inflammation of the abdominal viscera. The origin of the abscess is not easy to explain, it seems rational to suppose that the primary process consisted in the formation of a hæmatoma, the result of the

injury sustained. Subsequent infection occurred by bacteria derived either from the intestine or from the blood stream and abscess formation resulted. The same sequence has been observed in rupture of the liver or kidney, followed by hemorrhage into the retroperitoneal tissues.

Diffuse inflammation of the retroperitoneal tissues with pus formation may follow suppurative infections of the appendix, gall-bladder or pancreas. Sprengel (*Arch. Klin. Chir.*, 1912, c, 382) in a recent contribution has thoroughly reviewed the subject, and believes that the general peritoneal cavity in some instances may be uninvolved after appendicitis, for example, while a diffuse suppurative process may attack the retroperitoneal space. As a general rule the peritoneum acts as a barrier and prevents rupture of the pus into the abdominal cavity, but œdema of the mesentery and intestines, and a serous or blood-tinged effusion may result from the infection, and prove misleading when the abdomen is opened.

Sprengel refers to a case which resembles the one reported in many respects. The patient, a woman sixty years of age, sustained a fracture of the neck of the femur, and ten days later was seized with an attack of pain, vomiting and abdominal distention. The patient collapsed and death resulted on the eleventh day. The autopsy disclosed a suppurative para-proctitis which was regarded as the primary infection, and from this a fatal peritonitis arose. The rectal mucosa did not show any trace of injury from which the infection might have started.

The origin and formation of many of these infections is not clear. In children the diagnosis is difficult because of the possibility of an acute osteomyelitis of the femur, and because of the abdominal symptoms produced by pressure of the retroperitoneal effusion. That the disease is not so uncommon may be judged from the occurrence of this case, a second one a short time afterward in Dr. Rodman's service, and several of similar nature in Dr. Wharton's wards at the Children's Hospital.

Dr. J. Stewart Rodman recalled the particulars of a case which occurred at the Presbyterian Hospital, in the service of Dr. W. L. Rodman. The patient was a boy of ten who had been run over by a wagon three weeks previous to his admission to the hospital, thus giving a history of trauma more or less remote. The diagnosis was not altogether clear at first, as tenderness and

fixation of the left hip suggested an acute tubercular arthritis. Later on, however, an easily palpable tumor appeared above Poupart's ligament on the left side which was quite tender to touch and accompanied by a septic temperature. The diagnosis of retroperitoneal abscess was now quite clear. This was opened extraperitoneally and the boy afterward made a rapid recovery.

PERFORATION OF ILEUM BY A FOREIGN BODY FOLLOWED BY SYMPTOMS SIMULATING APPENDICITIS.

DR. SPEESE recited the history of a man, aged fifty-three, who was admitted to Dr. Jopson's service at the Presbyterian Hospital with the following history: Two days previously he experienced a sudden and severe pain in the right iliac region, this was accompanied by nausea and vomiting. The pain which continued until his admission to the hospital, gradually became more severe, the nausea persisted but the vomiting ceased; the bowels had not moved for two days. The leucocytic count was 24,500, pulse 92, temperature 99.4°. The abdomen was slightly distended, and on palpation the right rectus muscle was rigid, and pronounced tenderness was present over McBurney's point.

A diagnosis of acute appendicitis was made and the operation which was at once performed by Dr. Speese revealed many recent adhesions about the cæcum, a small amount of turbid fluid and fibrin, but a normal appendix. Believing that an inflamed Meckel's diverticulum might have caused the condition, the ileum was drawn out of the wound and examined. About four inches from the ileo-cæcal junction a foreign body was felt in the lumen of the bowel, one end being firmly embedded in the intestinal wall almost penetrating the serous coat. The body was extracted and was found to consist of a flat piece of bone, 4 x 1 cm., resembling a sequestrum, one end was splintered and as sharp as a needle. The intestinal wound was closed, examination of the surrounding coils of intestine did not reveal any evidence of perforation. The local peritonitis present was regarded as the result of a puncture of the intestine by the foreign body, the opening being small was soon closed by plastic lymph making its site difficult to discover. The abdomen was drained, convalescence was uninterrupted.

The patient when questioned was unable to remember that he had swallowed a piece of bone, but as he used alcohol to

excess, it seemed likely that the accident occurred during one of his frequent debauches, from one of which he was recovering at the time of his admission to the hospital.

Dr. George G. Ross reported a case seen by him which was diagnosed as acute appendicitis; there was tenderness rather higher up than usual. At operation he found the appendix inflamed, but also noticed that on the outer wall of the cæcum there was a great deal more inflammatory lymph than on the inner. In pulling up the cæcum a tooth pick floated up into the wound and was removed. It seemed as if the tooth pick had gone through the ileocæcal valve and had pinned itself on the opposite wall of the cæcum and had ulcerated through.

Dr. Edward B. Hodge said that he saw a patient last summer which resembled Dr. Speese's case very closely: A boy of four years came to the Children's Hospital. It was thought that he had appendicitis; he had the usual symptoms and had had two attacks previously of colicky pain which had been diagnosed as appendicitis by the family physician. Operation revealed some adhesions in the right iliac fossa and some free dirty fluid; no pus. Appendix looked all right. Further search detected a stricture of the ileum about one foot from the ileocæcal valve and a perforation above through which a piece of pickle was protruding. He did not think the pickle made the puncture but the boy had perforation and a very tight stricture. A resection was done and the patient eventually got well.

Dr. Morris Booth Miller raised the question of perforation from foreign bodies. This matter has never been definitely worked out, *i.e.*, why certain objects cause perforation and others do not. All are familiar with the fact that children often have passed through their intestinal tract extraordinary substances, such as coins, pins, buttons and various materials of similar type. The great majority of these pass through without disturbance, but occasionally serious trauma is inflicted.

Twice he had seen perforation caused by a piece of bone. Several years ago he operated upon a woman who had all the symptoms of acute appendicitis. When opened it was found that she had a perforation of the appendiceal tip due to a piece of chicken bone, which was hardly larger than one-half the size of an ordinary pin. Again, about a year ago he had an experience with a woman, age fifty-five, who had previously under-

gone a hysterectomy. Following this she had had an incisional hernia which was characterized by adhesions. She was suddenly taken ill with pain, local tenderness in the hernial sac and vomiting. At operation an irreducible hernia was found but not obstruction. Within the sac a piece of chicken bone over an inch long had perforated the gut with some local contamination. It was hoped she would get well, but as she was diabetic she died from coma in two or three days. There was no generalized peritonitis.

(1) PERFORATED MECKEL'S DIVERTICULUM, (2) TWO INDEPENDENT SACS IN AN INGUINAL HERNIA, (3) REMOVAL OF PARAFFINE FROM INGUINAL CANAL IN CASE OF INGUINAL HERNIA.

DR. A. BRUCE GILL reported the following cases from the service of Dr. Hodge at the Presbyterian Hospital:

CASE I.—A school-boy, aged thirteen years, admitted to the ward on the evening of March 4, 1913. For a week previous he had been having crampy pains about the umbilicus that occurred for the most part during the afternoon while the patient was in school. On the day of his admission about two o'clock in the afternoon while in school, he was seized with a sudden severe pain about the umbilicus and in the lower right part of the abdomen. He vomited repeatedly and was carried home from school.

On examination at ten o'clock P.M. there was present general abdominal rigidity which was most marked in the lower right quadrant. Pain and tenderness were chiefly in the same quadrant. The patient lay on his left side with the knees drawn up. On preparing the abdomen for operation a small red papule at the umbilicus bled slightly on being scrubbed.

The mother stated later that he had bled from the umbilicus in infancy, and the patient himself had observed a red spot at the umbilicus that would bleed on irritation.

Operation was performed at 10.25 P.M. The abdomen was filled with bloody serum. The appendix was long and kinked, but not diseased. A Meckel's diverticulum was found coming from the ileum about two inches from the ileo-cæcal junction. It was about two inches long and as large as the middle finger. A cord three inches long extended from the end of the diver-

ticulum to the umbilicus. The diverticulum was highly congested and was thickened. Near its base was an annular thickening and constriction. On the distal edge of the dense ring was a small perforation filled with a blood clot.

With the exception of enlarged glands in the transverse mesocolon there was no other evidence of abdominal disease.

The cord attached to the diverticulum was ligated and divided near the umbilicus. The diverticulum was excised and its stump was inverted by a purse-string suture reinforced by Lembert's sutures. The appendix was removed. The abdomen was freely flushed with hot salt solution and a drain was placed in the pelvis.

Recovery was uninterrupted and the patient was discharged on March 21, 1913.

CASE II.—Man, aged fifty-five years. Four years ago he suddenly developed a right inguinal hernia. He was stacking soap-boxes weighing 100 pounds each. While he had the seventh box above his head and was about to place it upon the pile, his right foot trod upon a potato and he slipped suddenly backward. The patient immediately felt a sharp nauseating pain in his right groin, and shortly afterward he noticed a soft swelling in the same region. The hernia has slowly increased in size to the present time. He has latterly worn a truss that did not restrain the hernia but did cause considerable pain. The hernia was reducible.

At operation a hernial opening large enough to admit the forefinger was found internal to the cord and one inch above the external ring. Protruding about two inches through the opening was a pouch of transversalis fascia which contained preperitoneal fat and a thin empty peritoneal sac. The sac was drawn down, ligated, and excised, and its stump was transposed upward and inward and fastened behind the rectus muscle. Upon examination of the cord a second hernial sac was found coming out of the internal ring. It was freed from adherent veins, opened, and found to be empty. The sac was excised and the stump disappeared into the abdomen. The operation was completed according to the Bassini method, but with the addition of one suture that fastened the edge of the rectus fascia to Poupart's ligament near the pubic spine, and a second suture external to the internal ring.

Recovery was uneventful. The patient was seen recently, which is more than a year since the operation. There has been no recurrence of the hernia, although he returned to his former work and has been doing heavy lifting.

CASE III.—Man, aged forty-eight years. He developed a right inguinal hernia seven years ago. Four years ago paraffine was injected at the side of the hernia by some person unknown. For eight months following this the hernia seemed cured, but at the end of that time it recurred and gradually increased in size. Several masses of paraffine could be felt beneath the skin and within the inguinal canal.

At operation one mass of paraffine was found imbedded in the aponeurosis of the external oblique in front of the canal, another lay beneath the skin above the canal, several pieces, including the largest, were within the canal. The vas was firmly adherent to three separate masses of paraffine and was dissected free with difficulty. The aponeurosis in front of the canal was dense and thick, and structures were obscured by the fibrous tissue about the masses of paraffine. The canal was exposed by splitting the external oblique above the canal and not in front of it and dissecting the lower flap downward. The largest piece of paraffine was near the internal ring, and its fibrous capsule was continuous with a mass of dense tissue that appeared to be an old obliterated sac. All the pieces of paraffine were removed.

A thin, wide-mouthed, empty sac was found posterior and internal to the cord and to the old obliterated sac. It was excised and its stump was transplanted upward and inward and fixed behind the rectus. The conjoined tendon and the edge of the rectus were sutured to Poupart's ligament in front of the cord. The upper flap of the aponeurosis of the external oblique was also sutured to Poupart's ligament, and the lower flap overlapped it and was sutured above.

DR. ASTLEY P. C. ASHHURST said that Dr. Gill's case of two sacs in an inguinal hernia reminded him of a similar sac found this winter while operating on a cadaver in the University. There were two sacs in the canal and extending down beyond the external ring; each had a separate opening into the peritoneal cavity at the internal ring. They were excised and sent to Dr. C. Y. White at the Episcopal Hospital for examination. He reported that both sacs were lined with endothelium appar-

ently identical. The existence of adventitious bursæ which are not very rare in the wall of a hernial sac is easily explained, but he knew of no satisfactory explanation for two distinct sacs, unless both are congenital.

As regards Meckel's diverticulum, he had seen three cases. The first case was reported to the Academy some years ago. There was an internal hernia through a rent in the mesentery of the ileum. The patient was a child, aged twelve, and, as he had learned that if in children from thirteen to fifteen years of age, the umbilicus appears abnormal, one should suspect the existence of a Meckel's diverticulum, the opinion was hazarded before operation that this might be the origin of the obstruction.

Only a month later he saw a man of forty-six years with symptoms of intestinal obstruction. He also had an abnormal umbilicus; four spots could be seen where the fetal structures had come through. In this case the diverticulum was found adherent to the umbilicus on the inside and twisted on itself. Unfortunately, the man died, in spite of relief of the obstruction.

Another case of Meckel's diverticulum he saw in a patient with typhoid perforation. The perforation occurred in the ileum and in looking for more perforations he found a slightly inflamed but non-adherent diverticulum higher up. This patient, who was eighteen years of age, had an abnormal umbilicus.

Dr. Addinell Hewson said that recently, in his service at the hospital, a case came in with symptoms suggesting acute obstruction. Operation was performed and it was found that a Meckel's diverticulum was present and located about 2 feet from the ileocæcal valve, was twisted around the intestine and caused complete obstruction. This case was not in a young person, but in a man of mature age who made an uneventful recovery.

SUTURE OF THE HEART.

Dr. Arthur E. Billings reported the case of a man, thirty-one years old, who was brought to the Pennsylvania Hospital, at 2.55 A.M. on May 7, 1911, and was admitted to the service of Dr. Hutchinson, to whom the reporter was greatly indebted for the privilege of operating upon and reporting this case.

The patient's previous history was negative except for marked alcoholism for the last two years. Present condition: While

walking through the east side of Franklin Square he was attacked by a man who stabbed him and fled. He was found by a policeman lying in an alley between Fifth and Sixth and between Race and Vine Streets, from where he was brought to the Pennsylvania Hospital by the patrol wagon. He was unconscious at the time of his admission, presenting the picture of extreme shock with hemorrhage. Temperature was 95°, breathing labored and sighing in character, radial pulse was imperceptible, and skin was clammy and cold. On examination of his thorax, there was a wound about 2 cm. in length in the midclavicular line of the left fifth interspace which was bleeding quite freely with respiratory movements. Percussion and auscultation revealed signs of a marked hæmopneumothorax. The heart sounds were irregular and distant, rapid, and at times almost inaudible.

Operation.—Local preparation consisted of shaving and the application of tincture of iodine to the field of operation. A semilunar incision was made over the left fourth and fifth ribs with the convexity toward the sternum. The fourth and fifth ribs were then severed near the costal border and turned outward about 5 cm. away from the sternal border forming a trap door. The left pleural cavity was filled with blood and the pericardium showed a wound about 1.5 cm. in length and was distended with blood. This opening was increased in length exposing the heart which revealed a wound in the left ventricle between 2 and 3 cm. in length on the external surface and slightly triangular in character, which was bleeding profusely with each systole, and was partially controlled by digital pressure during suturing.

This was closed with five interrupted sutures of No. 1 iodized catgut. The pericardium was closed with a continuous No. 1 iodized catgut suture, without drainage. In the lower lobe of the left lung on its anterior surface, there was a ragged wound about 4 cm. in length which continued to bleed slowly and persistently. The sixth rib was then resected in the left midaxillary line and a small gauze pack was introduced to control bleeding from the lung. The trap door was then closed, the ribs being sutured in place with No. 5 iodized catgut and the skin closed with interrupted silkworm gut sutures without drainage. During the first part of the operation, an anæsthetic was not necessary, ether

being used during the latter part of it. During the operation the patient received twenty-four ounces of normal salt solution, intravenously, and strychnine sulph., grains 1/20 hypodermically. After the operation the reaction was fairly prompt and surprisingly good. Pulse one hour after operation was 88 and temperature 96.2°.

During the following twelve hours, temperature reached 102.2°, soon dropping to normal with pulse ranging from 88 to 120, and the patient rational. Thirty-six hours after operation, patient's temperature began to go up, reaching 101.1°, pulse 120 to 140, with much respiratory distress and increasing cough with gradual circulatory failure, death resulting about forty-eight hours after operation. Cardiac compression is suggested in this case because of the marked increase in bleeding after opening the pericardium and improvement in patient's condition at this time.

Autopsy revealed extensive pleurisy and complete solidification of the already collapsed left lung with slight pericarditis about the line of incision and wound.

SPLENIC ANÆMIA, WITH SPECIAL REFERENCE TO ETIOLOGY AND SURGICAL TREATMENT.

BY J. STEWART RODMAN, M.D.,

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AND

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(Experimental work from the Laboratory for Research, Medico-Chirurgical College.)

SPLENIC anæmia, although recognized as a pathologic entity even in the early years of the nineteenth century, presents to-day many unsolved problems. This condition has been described under various names during the past twenty-five years but at the present time splenic anæmia is the generally accepted term. By it we mean a disease of long duration, generally lasting several years, characterized by an anæmia of the chlorotic type, considerable enlargement of the spleen without known cause, an absence of leucocystosis and a tendency to gastro-intestinal hemorrhage. In the advanced stage, cirrhosis of the liver, ascites and jaundice are also present. Banti, Osler and others have fully described the course of the disease and divide it into three distinct stages.

The first stage lasts two to ten years, with splenomegaly, anæmia, gastro-intestinal hemorrhage and pigmentation of the skin as its prominent symptoms. The second stage lasting only a few months adds to these symptoms kidney insufficiency with scanty high-colored urine. The third and terminal period presents the stage of liver involvement in which appears a cirrhosis of the Lænnec type with its accompanying ascites.

The name, Banti's disease, has been applied incorrectly to all three stages but it is the symptom-complex of the ascitic

stage alone that should bear the name of the man who did so much to clear up its symptomatology and pathology.

During the first stage, there are usually marked fluctuations in the severity of the symptoms. Months may pass in which slight enlargement of the spleen is the only demonstrable lesion. Again during the exacerbations, the spleen may enlarge enormously, with coincident increase in the anæmia and icterus.

Etiology.—The etiology of splenic anæmia has proved baffling. Much confusion has arisen and the main purpose befogged in endless discussion as to whether or not the splenomegaly is primary or secondary. The mere fact that we have first this splenomegaly is important, but not necessarily convincing that it alone is responsible for the anæmia, the cirrhosis and endophlebitis of the splenic and portal veins. Rather would it seem more reasonable to us that the splenomegaly was a link in the chain which resulted from the chronic irritation of some as yet unknown toxin, probably of gastro-intestinal origin. Having once acquired splenomegaly, we believe that it is responsible for the anæmia by reason of an exaggeration of its normal hæmolytic functions. In other words, the spleen finds itself much in the same position as the thyroid, which, as the result of some irritation, increases its activity and thus brings about hyperthyroidism. It is logical to assume this because we know that splenectomy, if done in the first stage, stops the anæmia with an early return of the blood picture to normal. Here again, comparison with the results of partial thyroidectomy are apt, since there too an excessive physiological function is checked. But we do not have to depend on splenectomy alone to feel sure this is so. Banti in his latest paper states that the anæmia is due to increased hæmolysis chiefly, and secondly to an actual increase in the blood destroying units of the spleen.

Banti, Joannovico, Lemarre and Gaucher have shown that splenectomized dogs have an increased resistance to certain hæmolytic poisons (pyridine, etc.). F. Bartazzi has also shown increase in the red blood corpuscle resistance in

splenectomized dogs. Pagliesi and Tuzzati, later Charrin and Moussie, and later still Gaucher, have shown that splenectomized dogs have a considerable lessening of biliary secretion. The logical conclusion is that the spleen has a great influence on hæmolytic and bilogenic processes not only because it diminishes red cell resistance and prepares them for destruction in other organs, but because it destroys them directly and forms thereby the necessary substances for biliary secretion.

The part that the enlarged spleen plays in the production of the anæmia has given rise to other theories. Harris and Hertzog believe that by reason of an erythrolytic enzyme secreted by the endothelial cells of the hyperplastic spleen there is a destruction of red blood corpuscles, and cite splenectomy with its cure to substantiate them in their position. Barr thinks that an increased blood supply to the spleen, with its consequent fibrosis, causes increased hæmolysis. The congestion is explained on a theory of vasomotor paresis of the splanchnic area from disease of the visceral sympathetic ganglia. Lintvarev believes that the anæmia is due to an increased destruction of the red cells by the erythrophages of the spleen, which cells have been produced in excessive numbers because of certain poisons (exogenous as well as endogenous). Rolleston would explain the anæmia upon an inhibition of blood formation due to the endothelial hyperplasia found in the enlarged spleen. So, while opinions differ as to the actual manner of accomplishment, it seems reasonably certain that the spleen causes the anæmia. Banti's theory of increased hæmolysis brought about by lessened resistance of the red blood corpuscles so that they are destroyed in greater proportion in the liver, lymph nodes and spleen itself, seems to us the most satisfying, since it is based on reliable experimental evidence and clinical findings. In these cases we have icterus without decoloration of the fæces, and we find that in periods of aggravation of the disease a further diminution of the red blood corpuscles and hæmoglobin is coincident with augmentation of icterus and urobilinuria.

The question of the primary intoxication is by no means so definitely settled. Senator was perhaps the first to suggest that the toxin was absorbed from the gastro-intestinal tract. Others have followed accepting this theory, some offering clinical data to substantiate their position. Ostrowski, who states that in ten cases of splenic anæmia in children all had had an antecedent diarrhœa, and Ungar and Neuberg believe that the gastro-intestinal tract must be looked into. It would seem not illogical to assume that the gastro-intestinal tract might furnish the toxin because of the somewhat similar clinical picture that is found in protracted auto-intoxication and in the beginning of splenic anæmia. Banti, however, adhering to the infectious theory, does not believe that the irritant comes from the gastro-intestinal tract, reasoning that the atrophy occurring in the liver from cirrhosis due to auto-intoxication is not produced for years, whereas that occurring in the liver during the third stage of splenic anæmia occurs in a few months. Conflicting ideas also are advanced as to casual relation existing between splenic anæmia and certain diseases, as tuberculosis, syphilis, Kala-Azar, malaria and gout. Banti again thinks that his syndrome, when occurring as the third stage of splenic anæmia at least, is a disease *sui generis*, but does not deny that enlarged spleen, cirrhosis and ascites may be found in the terminal stages of other diseases, as is pointed out by Hultgen, Rolleston and others. Osler thinks that when secondary anæmia and splenomegaly are found in other conditions the differential diagnosis may be made, since (*a*) the cause is usually apparent as malaria, tuberculosis, syphilis, etc., (*b*) the anæmia and splenomegaly usually yield to appropriate treatment, (*c*) the spleen is not so large, (*d*) sequences not so characteristic, (*e*) the blood, while showing secondary anæmia, does not usually show the exaggerated chlorotic type, and (*f*) leucocytosis is more common than leucopænia.

We know that the spleen is not rigidly controlled by nervous influences from the central or sympathetic nervous system, but that its stimulation depends largely upon the

circulation of systemic blood which comes in direct contact with the parenchyma of this organ. Further, that a part of its physiologic function is to sift out from the circulating blood bacteria, toxins and other detritus. These facts offer additional theoretical evidence of the probable toxic nature of the primary irritant.

Certainly one of the most interesting problems to be solved is the connection between endophlebitis of the portal and splenic veins and the enlarged spleen. Is the thrombosis primary or secondary? That some relation exists is reasonably certain, because of the frequency with which thrombosis of the portal and splenic is found at autopsy in cases dying of splenic anæmia. The reports of Dock and Warthin, Oettinger and Fiessinger, Banti, Rolleston and many others amply attest this fact.

It was really to attempt to shed additional light on this phase of the question that led the authors to carry out experiments on animals.[1] Our aim was first to produce thrombosis of the portal and splenic and later to study the splenic changes and blood picture. It seemed easy to ligate the splenic vein and cause a passive congestion of the spleen which would, in a measure, at least reproduce what actually occurred in cases of portal and splenic thrombosis. This we did in seven dogs, and found that in every case the immediate enlargement of the spleen was enormous, but in none of our cases did this increase in splenic dimensions last longer than one month, to be followed by permanent atrophy of the organ. The results coincide with Warthin's earlier ligations of the splenic vein. In another series of three dogs and two rabbits ligation of the splenic veins and injection of aleuronat directly into the splenic pulp were tried. Here again, in every instance, temporary increase in the size of the spleen followed with local necrosis of the splenic pulp at the injection sites. The splenic congestion with its consequent enlargement gave way to permanent atrophy. The aleuronat was used to supply an irritant and was chosen because we wished to produce an inflammation that would stop short of pus formation. Feeling that ligation, with its sudden congestion, did not reproduce the actual pathological condition, we attempted partially to occlude the lumen of the splenic vein. This we did by metal clips and loosely tied ligatures in a series of three dogs. Slight enlargement of the spleen occurred temporarily, to be followed in three weeks by a return to the normal size. Having failed with the splenic we next tried, in a series of five dogs, to produce portal thrombosis by exposing the vein, breaking the intima by compression and partially occluding the lumen

[1] All experimental work on animals done under ether anæsthesia

STATED MEETING, HELD MAY 5, 1913.

GEORGE G. ROSS, M.D., in the Chair.

ACUTE GASTRIC DILATATION FOLLOWING OPERATION FOR HERNIA.

DR. JOHN SPEESE presented a man, fifty-six years of age, who was operated upon at the Presbyterian Hospital for an incomplete inguinal hernia by the Bassini method. The patient recovered consciousness readily and experienced but slight nausea from the ether. Thirty-four hours later, after drinking a small quantity of milk, he complained of nausea, which gradually became more severe, and was followed by vomiting two hours later. Temporary relief was experienced, and no alarm was felt as the temperature, pulse and leucocytic count were normal and the abdominal muscles relaxed. The vomiting recurred, the fluid was dark in color, odorless and was ejected in small amounts without effort every few minutes. The patient then developed considerable abdominal pain, and on examining the abdomen at this time, the stomach was found greatly dilated, extending to the umbilicus, and partially filled with fluid, as determined by a succussion splash. A stomach tube was passed and several quarts of dark fluid withdrawn, and lavage performed. An abdominal binder was applied and the patient turned on his abdomen, a position he could assume only for short periods.

He was entirely relieved of the pain and vomiting for six hours, when the latter recurred in a less severe form. The same treatment was carried out and permanently relieved the condition. Examination of the stomach twelve hours after the vomiting began, disclosed a normal state of affairs, the dilatation having perfectly subsided. The patient was given salt solution by rectum to relieve his thirst, and small quantities of water by mouth were allowed twelve hours later without causing any disturbance.

Dr. Speese remarked that acute dilatation of the stomach may follow any variety of operation, although it appears to be relatively uncommon as a post-operative complication of hernia. Lyle (*N. Y. Med. Jour.*, 1911, xciv, 932) observed a case follow-

ing the radical cure of a hernia, the operation was performed under local anæsthesia, and a large amount of omentum was resected. The dilatation began one hour after the operation, and in all probability was caused by the removal of the large amount of omentum which profoundly altered the circulation and possibly the nervous mechanism of the stomach, thus causing a temporary muscular paralysis.

The finding, at post-mortem, of constriction of the duodenum by the superior mesenteric artery at the point where it crosses the intestine, has naturally caused this factor to be regarded as of much importance in the etiology of the affection. That the constriction can be caused by an abnormally long mesentery or by ptosis seems unlikely, and is denied by Mathieu (*Arch. d. Mal. d l' App. Dig.*, 1911, v, 409) who regards the gastric dilatation as the primary factor. Distention of the stomach must take place in a downward direction, the intestines are compressed, the superior mesenteric artery is put on a stretch and the duodenum is thus constricted. The primary dilatation is caused by aërophagia, which is the result of the constant swallowing which follows the nausea and pharyngeal irritation induced by ether. This view seems feasible in the case reported as the patient complained of annoying thirst soon after his recovery from the ether and the constant swallowing must have been accompanied by the passage of a considerable quantity of air into the stomach. The dryness and irritation of the pharyngeal mucosa were moreover increased by a chronic nasal obstruction which made mouth breathing a necessity.

Dr. Edward B. Hodge said that recently, in the case of a woman, fifty-seven years of age, who developed pneumonia one week after operation for appendicitis, temperature was normal for two days, at which time she developed acute dilatation of the stomach; this was relieved after several lavages, but the patient finally succumbed to acute nephritis.

Dr. George G. Ross reported a case of dilatation of the stomach which occurred about the seventh day after an operation in an otherwise healthy young woman who had pus tubes. She had done very well until the night of the second day when she drank the contents of an ice-bag (about one quart). The next morning she had acute dilatation from which she promptly died in spite of all treatment; the water from the ice-bag being the supposed cause.

RETROPERITONEAL ABSCESS.

DR. SPEESE reported the history of a child, eight years of age, who was admitted to Dr. Jopson's service at the Presbyterian Hospital, complaining of pain in the right wrist and left thigh. One week before he had been struck over the left hip, and in several hours the leg became stiff. This increased to such an extent that in three days the boy could not walk, and the thigh became very tender on pressure. The wrist also began to pain considerably and was slightly swollen.

September 5, 1912, four days after the accident, when admitted he complained of abdominal distention, pain and swelling over the lower end of the radius. The region about the hip was very tender, there was slight swelling on the outer side of the femoral vessels, and pressure over the trochanter caused pain. The temperature was 103°, W. B. C. 15,000. Exploration over the trochanter by Dr. Speese revealed normal bone and no evidence of pus. The lower end of the radius was exposed and free pus was found under the periosteum which was raised, the cortex of the radius was chiselled and several drops of pus found in the medullary cavity.

September 7, 1912, the pain in the hip region continued, and the abdomen was markedly distended, tender, but not rigid. W. B. C. 21,000.

September 9, 1912, an area of fluctuation was detected in the left groin, below Poupart's ligament and to the inner side of the femoral vessels. A large amount of pus containing blood clot was evacuated, and on exploring the abscess cavity it was found to extend behind the peritoneum for a distance of four or five inches. The abdominal distention disappeared after evacuation of the abscess.

The cavity was drained, and closed slowly as the convalescence was prolonged by the formation of several pyæmic abscesses. The bacteriological examination of the pus showed staphylococcus aureus.

Instances of retroperitoneal abscess of this type are uncommon, and may cause considerable difficulty in diagnosis because of the symptoms suggesting an acute inflammation of the abdominal viscera. The origin of the abscess is not easy to explain, it seems rational to suppose that the primary process consisted in the formation of a hæmatoma, the result of the

injury sustained. Subsequent infection occurred by bacteria derived either from the intestine or from the blood stream and abscess formation resulted. The same sequence has been observed in rupture of the liver or kidney, followed by hemorrhage into the retroperitoneal tissues.

Diffuse inflammation of the retroperitoneal tissues with pus formation may follow suppurative infections of the appendix, gall-bladder or pancreas. Sprengel (*Arch. Klin. Chir.*, 1912, c, 382) in a recent contribution has thoroughly reviewed the subject, and believes that the general peritoneal cavity in some instances may be uninvolved after appendicitis, for example, while a diffuse suppurative process may attack the retroperitoneal space. As a general rule the peritoneum acts as a barrier and prevents rupture of the pus into the abdominal cavity, but œdema of the mesentery and intestines, and a serous or blood-tinged effusion may result from the infection, and prove misleading when the abdomen is opened.

Sprengel refers to a case which resembles the one reported in many respects. The patient, a woman sixty years of age, sustained a fracture of the neck of the femur, and ten days later was seized with an attack of pain, vomiting and abdominal distention. The patient collapsed and death resulted on the eleventh day. The autopsy disclosed a suppurative para-proctitis which was regarded as the primary infection, and from this a fatal peritonitis arose. The rectal mucosa did not show any trace of injury from which the infection might have started.

The origin and formation of many of these infections is not clear. In children the diagnosis is difficult because of the possibility of an acute osteomyelitis of the femur, and because of the abdominal symptoms produced by pressure of the retroperitoneal effusion. That the disease is not so uncommon may be judged from the occurrence of this case, a second one a short time afterward in Dr. Rodman's service, and several of similar nature in Dr. Wharton's wards at the Children's Hospital.

Dr. J. Stewart Rodman recalled the particulars of a case which occurred at the Presbyterian Hospital, in the service of Dr. W. L. Rodman. The patient was a boy of ten who had been run over by a wagon three weeks previous to his admission to the hospital, thus giving a history of trauma more or less remote. The diagnosis was not altogether clear at first, as tenderness and

fixation of the left hip suggested an acute tubercular arthritis. Later on, however, an easily palpable tumor appeared above Poupart's ligament on the left side which was quite tender to touch and accompanied by a septic temperature. The diagnosis of retroperitoneal abscess was now quite clear. This was opened extraperitoneally and the boy afterward made a rapid recovery.

PERFORATION OF ILEUM BY A FOREIGN BODY FOLLOWED BY SYMPTOMS SIMULATING APPENDICITIS.

DR. SPEESE recited the history of a man, aged fifty-three, who was admitted to Dr. Jopson's service at the Presbyterian Hospital with the following history: Two days previously he experienced a sudden and severe pain in the right iliac region, this was accompanied by nausea and vomiting. The pain which continued until his admission to the hospital, gradually became more severe, the nausea persisted but the vomiting ceased; the bowels had not moved for two days. The leucocytic count was 24,500, pulse 92, temperature 99.4°. The abdomen was slightly distended, and on palpation the right rectus muscle was rigid, and pronounced tenderness was present over McBurney's point.

A diagnosis of acute appendicitis was made and the operation which was at once performed by Dr. Speese revealed many recent adhesions about the cæcum, a small amount of turbid fluid and fibrin, but a normal appendix. Believing that an inflamed Meckel's diverticulum might have caused the condition, the ileum was drawn out of the wound and examined. About four inches from the ileo-cæcal junction a foreign body was felt in the lumen of the bowel, one end being firmly embedded in the intestinal wall almost penetrating the serous coat. The body was extracted and was found to consist of a flat piece of bone, 4 x 1 cm., resembling a sequestrum, one end was splintered and as sharp as a needle. The intestinal wound was closed, examination of the surrounding coils of intestine did not reveal any evidence of perforation. The local peritonitis present was regarded as the result of a puncture of the intestine by the foreign body, the opening being small was soon closed by plastic lymph making its site difficult to discover. The abdomen was drained, convalescence was uninterrupted.

The patient when questioned was unable to remember that he had swallowed a piece of bone, but as he used alcohol to

excess, it seemed likely that the accident occurred during one of his frequent debauches, from one of which he was recovering at the time of his admission to the hospital.

Dr. George G. Ross reported a case seen by him which was diagnosed as acute appendicitis; there was tenderness rather higher up than usual. At operation he found the appendix inflamed, but also noticed that on the outer wall of the cæcum there was a great deal more inflammatory lymph than on the inner. In pulling up the cæcum a tooth pick floated up into the wound and was removed. It seemed as if the tooth pick had gone through the ileocæcal valve and had pinned itself on the opposite wall of the cæcum and had ulcerated through.

Dr. Edward B. Hodge said that he saw a patient last summer which resembled Dr. Speese's case very closely: A boy of four years came to the Children's Hospital. It was thought that he had appendicitis; he had the usual symptoms and had had two attacks previously of colicky pain which had been diagnosed as appendicitis by the family physician. Operation revealed some adhesions in the right iliac fossa and some free dirty fluid; no pus. Appendix looked all right. Further search detected a stricture of the ileum about one foot from the ileocæcal valve and a perforation above through which a piece of pickle was protruding. He did not think the pickle made the puncture but the boy had perforation and a very tight stricture. A resection was done and the patient eventually got well.

Dr. Morris Booth Miller raised the question of perforation from foreign bodies. This matter has never been definitely worked out, *i.e.*, why certain objects cause perforation and others do not. All are familiar with the fact that children often have passed through their intestinal tract extraordinary substances, such as coins, pins, buttons and various materials of similar type. The great majority of these pass through without disturbance, but occasionally serious trauma is inflicted.

Twice he had seen perforation caused by a piece of bone. Several years ago he operated upon a woman who had all the symptoms of acute appendicitis. When opened it was found that she had a perforation of the appendiceal tip due to a piece of chicken bone, which was hardly larger than one-half the size of an ordinary pin. Again, about a year ago he had an experience with a woman, age fifty-five, who had previously under-

gone a hysterectomy. Following this she had had an incisional hernia which was characterized by adhesions. She was suddenly taken ill with pain, local tenderness in the hernial sac and vomiting. At operation an irreducible hernia was found but not obstruction. Within the sac a piece of chicken bone over an inch long had perforated the gut with some local contamination. It was hoped she would get well, but as she was diabetic she died from coma in two or three days. There was no generalized peritonitis.

(1) PERFORATED MECKEL'S DIVERTICULUM, (2) TWO INDEPENDENT SACS IN AN INGUINAL HERNIA, (3) REMOVAL OF PARAFFINE FROM INGUINAL CANAL IN CASE OF INGUINAL HERNIA.

DR. A. BRUCE GILL reported the following cases from the service of Dr. Hodge at the Presbyterian Hospital:

CASE I.—A school-boy, aged thirteen years, admitted to the ward on the evening of March 4, 1913. For a week previous he had been having crampy pains about the umbilicus that occurred for the most part during the afternoon while the patient was in school. On the day of his admission about two o'clock in the afternoon while in school, he was seized with a sudden severe pain about the umbilicus and in the lower right part of the abdomen. He vomited repeatedly and was carried home from school.

On examination at ten o'clock P.M. there was present general abdominal rigidity which was most marked in the lower right quadrant. Pain and tenderness were chiefly in the same quadrant. The patient lay on his left side with the knees drawn up. On preparing the abdomen for operation a small red papule at the umbilicus bled slightly on being scrubbed.

The mother stated later that he had bled from the umbilicus in infancy, and the patient himself had observed a red spot at the umbilicus that would bleed on irritation.

Operation was performed at 10.25 P.M. The abdomen was filled with bloody serum. The appendix was long and kinked, but not diseased. A Meckel's diverticulum was found coming from the ileum about two inches from the ileo-cæcal junction. It was about two inches long and as large as the middle finger. A cord three inches long extended from the end of the diver-

ticulum to the umbilicus. The diverticulum was highly congested and was thickened. Near its base was an annular thickening and constriction. On the distal edge of the dense ring was a small perforation filled with a blood clot.

With the exception of enlarged glands in the transverse mesocolon there was no other evidence of abdominal disease.

The cord attached to the diverticulum was ligated and divided near the umbilicus. The diverticulum was excised and its stump was inverted by a purse-string suture reinforced by Lembert's sutures. The appendix was removed. The abdomen was freely flushed with hot salt solution and a drain was placed in the pelvis.

Recovery was uninterrupted and the patient was discharged on March 21, 1913.

CASE II.—Man, aged fifty-five years. Four years ago he suddenly developed a right inguinal hernia. He was stacking soap-boxes weighing 100 pounds each. While he had the seventh box above his head and was about to place it upon the pile, his right foot trod upon a potato and he slipped suddenly backward. The patient immediately felt a sharp nauseating pain in his right groin, and shortly afterward he noticed a soft swelling in the same region. The hernia has slowly increased in size to the present time. He has latterly worn a truss that did not restrain the hernia but did cause considerable pain. The hernia was reducible.

At operation a hernial opening large enough to admit the forefinger was found internal to the cord and one inch above the external ring. Protruding about two inches through the opening was a pouch of transversalis fascia which contained preperitoneal fat and a thin empty peritoneal sac. The sac was drawn down, ligated, and excised, and its stump was transposed upward and inward and fastened behind the rectus muscle. Upon examination of the cord a second hernial sac was found coming out of the internal ring. It was freed from adherent veins, opened, and found to be empty. The sac was excised and the stump disappeared into the abdomen. The operation was completed according to the Bassini method, but with the addition of one suture that fastened the edge of the rectus fascia to Poupart's ligament near the pubic spine, and a second suture external to the internal ring.

Recovery was uneventful. The patient was seen recently, which is more than a year since the operation. There has been no recurrence of the hernia, although he returned to his former work and has been doing heavy lifting.

CASE III.—Man, aged forty-eight years. He developed a right inguinal hernia seven years ago. Four years ago paraffine was injected at the side of the hernia by some person unknown. For eight months following this the hernia seemed cured, but at the end of that time it recurred and gradually increased in size. Several masses of paraffine could be felt beneath the skin and within the inguinal canal.

At operation one mass of paraffine was found imbedded in the aponeurosis of the external oblique in front of the canal, another lay beneath the skin above the canal, several pieces, including the largest, were within the canal. The vas was firmly adherent to three separate masses of paraffine and was dissected free with difficulty. The aponeurosis in front of the canal was dense and thick, and structures were obscured by the fibrous tissue about the masses of paraffine. The canal was exposed by splitting the external oblique above the canal and not in front of it and dissecting the lower flap downward. The largest piece of paraffine was near the internal ring, and its fibrous capsule was continuous with a mass of dense tissue that appeared to be an old obliterated sac. All the pieces of paraffine were removed.

A thin, wide-mouthed, empty sac was found posterior and internal to the cord and to the old obliterated sac. It was excised and its stump was transplanted upward and inward and fixed behind the rectus. The conjoined tendon and the edge of the rectus were sutured to Poupart's ligament in front of the cord. The upper flap of the aponeurosis of the external oblique was also sutured to Poupart's ligament, and the lower flap overlapped it and was sutured above.

DR. ASTLEY P. C. ASHHURST said that Dr. Gill's case of two sacs in an inguinal hernia reminded him of a similar sac found this winter while operating on a cadaver in the University. There were two sacs in the canal and extending down beyond the external ring; each had a separate opening into the peritoneal cavity at the internal ring. They were excised and sent to Dr. C. Y. White at the Episcopal Hospital for examination. He reported that both sacs were lined with endothelium appar-

ently identical. The existence of adventitious bursæ which are not very rare in the wall of a hernial sac is easily explained, but he knew of no satisfactory explanation for two distinct sacs, unless both are congenital.

As regards Meckel's diverticulum, he had seen three cases. The first case was reported to the Academy some years ago. There was an internal hernia through a rent in the mesentery of the ileum. The patient was a child, aged twelve, and, as he had learned that if in children from thirteen to fifteen years of age, the umbilicus appears abnormal, one should suspect the existence of a Meckel's diverticulum, the opinion was hazarded before operation that this might be the origin of the obstruction.

Only a month later he saw a man of forty-six years with symptoms of intestinal obstruction. He also had an abnormal umbilicus; four spots could be seen where the fetal structures had come through. In this case the diverticulum was found adherent to the umbilicus on the inside and twisted on itself. Unfortunately, the man died, in spite of relief of the obstruction.

Another case of Meckel's diverticulum he saw in a patient with typhoid perforation. The perforation occurred in the ileum and in looking for more perforations he found a slightly inflamed but non-adherent diverticulum higher up. This patient, who was eighteen years of age, had an abnormal umbilicus.

Dr. Addinell Hewson said that recently, in his service at the hospital, a case came in with symptoms suggesting acute obstruction. Operation was performed and it was found that a Meckel's diverticulum was present and located about 2 feet from the ileocæcal valve, was twisted around the intestine and caused complete obstruction. This case was not in a young person, but in a man of mature age who made an uneventful recovery.

SUTURE OF THE HEART.

Dr. Arthur E. Billings reported the case of a man, thirty-one years old, who was brought to the Pennsylvania Hospital, at 2.55 A.M. on May 7, 1911, and was admitted to the service of Dr. Hutchinson, to whom the reporter was greatly indebted for the privilege of operating upon and reporting this case.

The patient's previous history was negative except for marked alcoholism for the last two years. Present condition: While

walking through the east side of Franklin Square he was attacked by a man who stabbed him and fled. He was found by a policeman lying in an alley between Fifth and Sixth and between Race and Vine Streets, from where he was brought to the Pennsylvania Hospital by the patrol wagon. He was unconscious at the time of his admission, presenting the picture of extreme shock with hemorrhage. Temperature was 95°, breathing labored and sighing in character, radial pulse was imperceptible, and skin was clammy and cold. On examination of his thorax, there was a wound about 2 cm. in length in the midclavicular line of the left fifth interspace which was bleeding quite freely with respiratory movements. Percussion and auscultation revealed signs of a marked hæmopneumothorax. The heart sounds were irregular and distant, rapid, and at times almost inaudible.

Operation.—Local preparation consisted of shaving and the application of tincture of iodine to the field of operation. A semilunar incision was made over the left fourth and fifth ribs with the convexity toward the sternum. The fourth and fifth ribs were then severed near the costal border and turned outward about 5 cm. away from the sternal border forming a trap door. The left pleural cavity was filled with blood and the pericardium showed a wound about 1.5 cm. in length and was distended with blood. This opening was increased in length exposing the heart which revealed a wound in the left ventricle between 2 and 3 cm. in length on the external surface and slightly triangular in character, which was bleeding profusely with each systole, and was partially controlled by digital pressure during suturing.

This was closed with five interrupted sutures of No. 1 iodized catgut. The pericardium was closed with a continuous No. 1 iodized catgut suture, without drainage. In the lower lobe of the left lung on its anterior surface, there was a ragged wound about 4 cm. in length which continued to bleed slowly and persistently. The sixth rib was then resected in the left midaxillary line and a small gauze pack was introduced to control bleeding from the lung. The trap door was then closed, the ribs being sutured in place with No. 5 iodized catgut and the skin closed with interrupted silkworm gut sutures without drainage. During the first part of the operation, an anæsthetic was not necessary, ether

being used during the latter part of it. During the operation the patient received twenty-four ounces of normal salt solution, intravenously, and strychnine sulph., grains 1/20 hypodermically. After the operation the reaction was fairly prompt and surprisingly good. Pulse one hour after operation was 88 and temperature 96.2°.

During the following twelve hours, temperature reached 102.2°, soon dropping to normal with pulse ranging from 88 to 120, and the patient rational. Thirty-six hours after operation, patient's temperature began to go up, reaching 101.1°, pulse 120 to 140, with much respiratory distress and increasing cough with gradual circulatory failure, death resulting about forty-eight hours after operation. Cardiac compression is suggested in this case because of the marked increase in bleeding after opening the pericardium and improvement in patient's condition at this time.

Autopsy revealed extensive pleurisy and complete solidification of the already collapsed left lung with slight pericarditis about the line of incision and wound.

SPLENIC ANÆMIA, WITH SPECIAL REFERENCE TO ETIOLOGY AND SURGICAL TREATMENT.

BY J. STEWART RODMAN, M.D.,

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AND

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(Experimental work from the Laboratory for Research, Medico-Chirurgical College.)

SPLENIC anæmia, although recognized as a pathologic entity even in the early years of the nineteenth century, presents to-day many unsolved problems. This condition has been described under various names during the past twenty-five years but at the present time splenic anæmia is the generally accepted term. By it we mean a disease of long duration, generally lasting several years, characterized by an anæmia of the chlorotic type, considerable enlargement of the spleen without known cause, an absence of leucocystosis and a tendency to gastro-intestinal hemorrhage. In the advanced stage, cirrhosis of the liver, ascites and jaundice are also present. Banti, Osler and others have fully described the course of the disease and divide it into three distinct stages.

The first stage lasts two to ten years, with splenomegaly, anæmia, gastro-intestinal hemorrhage and pigmentation of the skin as its prominent symptoms. The second stage lasting only a few months adds to these symptoms kidney insufficiency with scanty high-colored urine. The third and terminal period presents the stage of liver involvement in which appears a cirrhosis of the Lænnec type with its accompanying ascites.

The name, Banti's disease, has been applied incorrectly to all three stages but it is the symptom-complex of the ascitic

stage alone that should bear the name of the man who did so much to clear up its symptomatology and pathology.

During the first stage, there are usually marked fluctuations in the severity of the symptoms. Months may pass in which slight enlargement of the spleen is the only demonstrable lesion. Again during the exacerbations, the spleen may enlarge enormously, with coincident increase in the anæmia and icterus.

Etiology.—The etiology of splenic anæmia has proved baffling. Much confusion has arisen and the main purpose befogged in endless discussion as to whether or not the splenomegaly is primary or secondary. The mere fact that we have first this splenomegaly is important, but not necessarily convincing that it alone is responsible for the anæmia, the cirrhosis and endophlebitis of the splenic and portal veins. Rather would it seem more reasonable to us that the splenomegaly was a link in the chain which resulted from the chronic irritation of some as yet unknown toxin, probably of gastro-intestinal origin. Having once acquired splenomegaly, we believe that it is responsible for the anæmia by reason of an exaggeration of its normal hæmolytic functions. In other words, the spleen finds itself much in the same position as the thyroid, which, as the result of some irritation, increases its activity and thus brings about hyperthyroidism. It is logical to assume this because we know that splenectomy, if done in the first stage, stops the anæmia with an early return of the blood picture to normal. Here again, comparison with the results of partial thyroidectomy are apt, since there too an excessive physiological function is checked. But we do not have to depend on splenectomy alone to feel sure this is so. Banti in his latest paper states that the anæmia is due to increased hæmolysis chiefly, and secondly to an actual increase in the blood destroying units of the spleen.

Banti, Joannovico, Lemarre and Gaucher have shown that splenectomized dogs have an increased resistance to certain hæmolytic poisons (pyridine, etc.). F. Bartazzi has also shown increase in the red blood corpuscle resistance in

splenectomized dogs. Pagliesi and Tuzzati, later Charrin and Moussie, and later still Gaucher, have shown that splenectomized dogs have a considerable lessening of biliary secretion. The logical conclusion is that the spleen has a great influence on hæmolytic and bilogenic processes not only because it diminishes red cell resistance and prepares them for destruction in other organs, but because it destroys them directly and forms thereby the necessary substances for biliary secretion.

The part that the enlarged spleen plays in the production of the anæmia has given rise to other theories. Harris and Hertzog believe that by reason of an erythrolytic enzyme secreted by the endothelial cells of the hyperplastic spleen there is a destruction of red blood corpuscles, and cite splenectomy with its cure to substantiate them in their position. Barr thinks that an increased blood supply to the spleen, with its consequent fibrosis, causes increased hæmolysis. The congestion is explained on a theory of vasomotor paresis of the splanchnic area from disease of the visceral sympathetic ganglia. Lintvarev believes that the anæmia is due to an increased destruction of the red cells by the erythrophages of the spleen, which cells have been produced in excessive numbers because of certain poisons (exogenous as well as endogenous). Rolleston would explain the anæmia upon an inhibition of blood formation due to the endothelial hyperplasia found in the enlarged spleen. So, while opinions differ as to the actual manner of accomplishment, it seems reasonably certain that the spleen causes the anæmia. Banti's theory of increased hæmolysis brought about by lessened resistance of the red blood corpuscles so that they are destroyed in greater proportion in the liver, lymph nodes and spleen itself, seems to us the most satisfying, since it is based on reliable experimental evidence and clinical findings. In these cases we have icterus without decoloration of the fæces, and we find that in periods of aggravation of the disease a further diminution of the red blood corpuscles and hæmoglobin is coincident with augmentation of icterus and urobilinuria.

The question of the primary intoxication is by no means so definitely settled. Senator was perhaps the first to suggest that the toxin was absorbed from the gastro-intestinal tract. Others have followed accepting this theory, some offering clinical data to substantiate their position. Ostrowski, who states that in ten cases of splenic anæmia in children all had had an antecedent diarrhœa, and Ungar and Neuberg believe that the gastro-intestinal tract must be looked into. It would seem not illogical to assume that the gastro-intestinal tract might furnish the toxin because of the somewhat similar clinical picture that is found in protracted auto-intoxication and in the beginning of splenic anæmia. Banti, however, adhering to the infectious theory, does not believe that the irritant comes from the gastro-intestinal tract, reasoning that the atrophy occurring in the liver from cirrhosis due to auto-intoxication is not produced for years, whereas that occurring in the liver during the third stage of splenic anæmia occurs in a few months. Conflicting ideas also are advanced as to casual relation existing between splenic anæmia and certain diseases, as tuberculosis, syphilis, Kala-Azar, malaria and gout. Banti again thinks that his syndrome, when occurring as the third stage of splenic anæmia at least, is a disease *sui generis*, but does not deny that enlarged spleen, cirrhosis and ascites may be found in the terminal stages of other diseases, as is pointed out by Hultgen, Rolleston and others. Osler thinks that when secondary anæmia and splenomegaly are found in other conditions the differential diagnosis may be made, since (*a*) the cause is usually apparent as malaria, tuberculosis, syphilis, etc., (*b*) the anæmia and splenomegaly usually yield to appropriate treatment, (*c*) the spleen is not so large, (*d*) sequences not so characteristic, (*e*) the blood, while showing secondary anæmia, does not usually show the exaggerated chlorotic type, and (*f*) leucocytosis is more common than leucopænia.

We know that the spleen is not rigidly controlled by nervous influences from the central or sympathetic nervous system, but that its stimulation depends largely upon the

circulation of systemic blood which comes in direct contact with the parenchyma of this organ. Further, that a part of its physiologic function is to sift out from the circulating blood bacteria, toxins and other detritus. These facts offer additional theoretical evidence of the probable toxic nature of the primary irritant.

Certainly one of the most interesting problems to be solved is the connection between endophlebitis of the portal and splenic veins and the enlarged spleen. Is the thrombosis primary or secondary? That some relation exists is reasonably certain, because of the frequency with which thrombosis of the portal and splenic is found at autopsy in cases dying of splenic anæmia. The reports of Dock and Warthin, Oettinger and Fiessinger, Banti, Rolleston and many others amply attest this fact.

It was really to attempt to shed additional light on this phase of the question that led the authors to carry out experiments on animals.[1] Our aim was first to produce thrombosis of the portal and splenic and later to study the splenic changes and blood picture. It seemed easy to ligate the splenic vein and cause a passive congestion of the spleen which would, in a measure, at least reproduce what actually occurred in cases of portal and splenic thrombosis. This we did in seven dogs, and found that in every case the immediate enlargement of the spleen was enormous, but in none of our cases did this increase in splenic dimensions last longer than one month, to be followed by permanent atrophy of the organ. The results coincide with Warthin's earlier ligations of the splenic vein. In another series of three dogs and two rabbits ligation of the splenic veins and injection of aleuronat directly into the splenic pulp were tried. Here again, in every instance, temporary increase in the size of the spleen followed with local necrosis of the splenic pulp at the injection sites. The splenic congestion with its consequent enlargement gave way to permanent atrophy. The aleuronat was used to supply an irritant and was chosen because we wished to produce an inflammation that would stop short of pus formation. Feeling that ligation, with its sudden congestion, did not reproduce the actual pathological condition, we attempted partially to occlude the lumen of the splenic vein. This we did by metal clips and loosely tied ligatures in a series of three dogs. Slight enlargement of the spleen occurred temporarily, to be followed in three weeks by a return to the normal size. Having failed with the splenic we next tried, in a series of five dogs, to produce portal thrombosis by exposing the vein, breaking the intima by compression and partially occluding the lumen

[1] All experimental work on animals done under ether anæsthesia

by means of a silver wire passed around the vein, so as to constrict, but not totally obstruct it. In no case were we able to produce by this means portal thrombosis, although slight temporary enlargements of the spleen were again noticed to measurements.

The pathological studies of the removed spleen following ligation showed passive congestion in the early cases, that is, those removed not later than one month after operation, but no real hyperplasia of the splenic substance. In the later cases, those removed after one month or longer had elapsed, there was atrophy of the splenic pulp and an increase in connective tissue. The blood pictures during the period of splenic enlargement showed no characteristic change beyond a leucocytosis following operation, which returned to the normal after an average period of two weeks had elapsed.

Other experimental work has been done; thus Solowieff produced a fibroid condition of the liver by producing occlusion of the portal branches in dogs. Osler cites a case of his own, however, in which there was obliteration of the portal but no true cirrhosis, although the liver was atrophied and had undergone fatty degeneration to some extent. This case showed enormous enlargement of the spleen with the thrombosis of some of the splenic veins and with that of Cruveilhier, shows that obliteration of the portal can take place in man without cirrhosis of the liver. Banti reports the results of an extensive bacteriological study. Believing that the disease is bacterial in origin, he cultured the peripheral blood, splenic substance, blood of splenic vein and bone marrow in fatal cases without result. Aërobic and anaërobic cultures were both negative. In addition, guinea pigs, rabbits, dogs and rats were injected with the blood, splenic juice, liver and bone marrow without result. Fragments of spleen, liver and bone marrow have been directly injected under skin and into the peritoneal cavity still without infection. Finally Banti grafted pieces of the diseased spleen into the normal spleen of dogs, also without result. He states that similar bacteriological studies by others have always yielded negative findings. Although these experiments have been thoroughly carried out the parasitic origin of the disease has by no means been disproved. The cellular hyperplasia of the spleen in this condition is suggestive of an irritation due to bacterial or at least protozoal growth. Again, in the diseases most closely simulating splenic anæmia, such as tuberculosis and syphilis of the spleen, malaria, etc, specific organisms have been isolated.

Pathology.—Many observers have carefully studied the pathology of splenic anæmia. In the main they agree as to the macroscopic and microscopic picture of the disease. In brief, the essential pathology is first an anæmia of the chlorotic type, the microscope showing marked aniso- and pœcilocytosis, polychromatophilia and often basophilic degeneration of the red cells, with the occasional finding of

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normoblasts and myelocytes. Secondly, an enlargement of the spleen which weighs on an average 61 ounces. It retains its normal shape, is dense and firm and under the microscope shows a general fibrosis, especially of the malpighian bodies, which are sclerosed and atrophied and often show hyaline degeneration. In addition, the blood spaces show marked hyperplasia of the endothelial cells and many large phagocytic cells, spoken of by Lintvarev as macrophages. In the third stage the liver shows a varying degree of atrophic interlobular cirrhosis, closely corresponding to that seen in chronic alcoholism. In addition, in a large majority of cases, there is reported an obliterating thrombophlebitis in various stages of degeneration of the splenic and portal veins, which often show almost complete obliteration of the lumen.

Treatment.—In the treatment of splenic anæmia, as in its etiology, a great diversity of opinions is met with. Medicine and surgery have had their advocates and opponents. With the rapid advance of surgical technic in the past few decades, the operative treatment now offers the greatest chance for permanent recovery.

In the medical treatment, iron and arsenic stand preeminent. They are of undoubted value in combating the anæmia and in improving the general condition, but at the best they are only temporary aids and in no way counteract the causal factor. Up to the present time literature shows no cases of permanent cure by medical treatment alone.

As our knowledge of the etiology of this condition advances, the medical treatment will of a certainty become more valuable, especially in the early stages. If we can but discover the primary intoxicant and attack this before the spleen is permanently damaged, our therapeutic agents will become powerful factors in what may be called the prophylactic treatment of splenic anæmia. At the present time, the early eradication of any chronic source of intoxication, especially of the alimentary tract, must be of general if not of specific benefit to the patient. As an example, a case of one of the authors may be cited.

A man, forty-six years of age, suffering from mild anæmia of two years' duration, with its usual symptoms and with a spleen reaching to the level of the umbilicus, was examined early in the year. His past history was negative with the exception of occasional attacks of gastro-enteritis and chronic nasal catarrh. An examination showed chronic frontal sinus trouble and marked gingivitis with symptoms of gastric and intestinal stasis. After several months of treatment for these conditions there has been a decided improvement as to the blood and spleen, and marked betterment of the patient's subjective symptoms. The case has been under observation for too short a time to definitely decide whether the improvement is to be more than temporary.

The intravenous injection of salvarsan has been used for this condition and its results parallel those of internal medication, *i.e.*, improvement in symptoms without permanent recovery.

Treatment by X-ray has been tried at various times. Lucatello reports a marked improvement in general condition in the blood and in the spleen for a time, but the relief from this, as from medicine, is only temporary.

In taking up the surgical treatment it may be wise to review the topography of the spleen. We are dealing with an organ of the upper abdomen, lying well within the vault of the diaphragm and overlaid anteriorly by the fundus of the stomach and posteriorly by the ribs. It is securely held in place by its ligaments and in the disease under discussion is often additionally fastened to the surrounding organs and parietes by firm adhesions. Its blood-vessels are large and extremely thin-walled, making ligation a serious matter. The splenic pulp is brittle and tears and bleeds easily under rough handling.

With these points in mind, the difficulties of operative procedures can readily be estimated. Proper exposure is the first essential. Mayo, Meyer, Treves and others have suggested various methods of approach, all of which have many points of merit. We would like to call special attention to a modification of the incision described by Dr. Edward Martin

for exposure of the liver and which, in the dissecting room at least, has given us the best access to the upper left quadrant of the abdomen.

The incision used is a reversed " J " (see Figs. 1–5). The vertical portion starts one cm. to the left of the middle line, at the tip of the ensiform cartilage and is continued downward to the level of the umbilicus. It is then carried outward across the rectus and outward and upward toward the tip of the seventh rib. The rectus muscle is cut transversely, preferably at the linea transversa. The external oblique and the transversalis muscles are separated in the direction of their fibres; the internal oblique fibres are cut across. The flap can then be turned up and out giving free access to the splenic area.

We consider that this incision not only gives us the best operative exposure but also greatly lessens the dangers of post-operative hernia. The blood supply of the flap is not interfered with on account of the free anastomosis with the vessels of the chest. The nerves supplying this area, coming as they do from the lower intercostals, run parallel with the transverse portion of the incision and therefore are not cut across. The transverse incision of the rectus has already proven satisfactory and the splitting of two of the three layers of the lateral wall adds greatly to the post-operative strength.

Having obtained the proper exposure, the greatest remaining difficulty lies in the efficient ligation of the vascular pedicle. Mayo and Sutherland both strongly urge the use of a temporary rubber covered clamp on this pedicle before ligation and the thorough testing of the ligatures before the complete removal of the clamp.

Extirpation of the spleen has been performed for various conditions by many operators and the consensus of opinion is that while the operation is a serious one with high mortality, it is entirely justifiable under certain conditions. The statistics for splenectomy from any cause compiled by Bessel Hagen in 1900, and by Johnson in 1908, show a mortality of 36.3 per cent. in a series of 353 cases and of 18.5 per cent. in a series of 355.

FIG. 1.

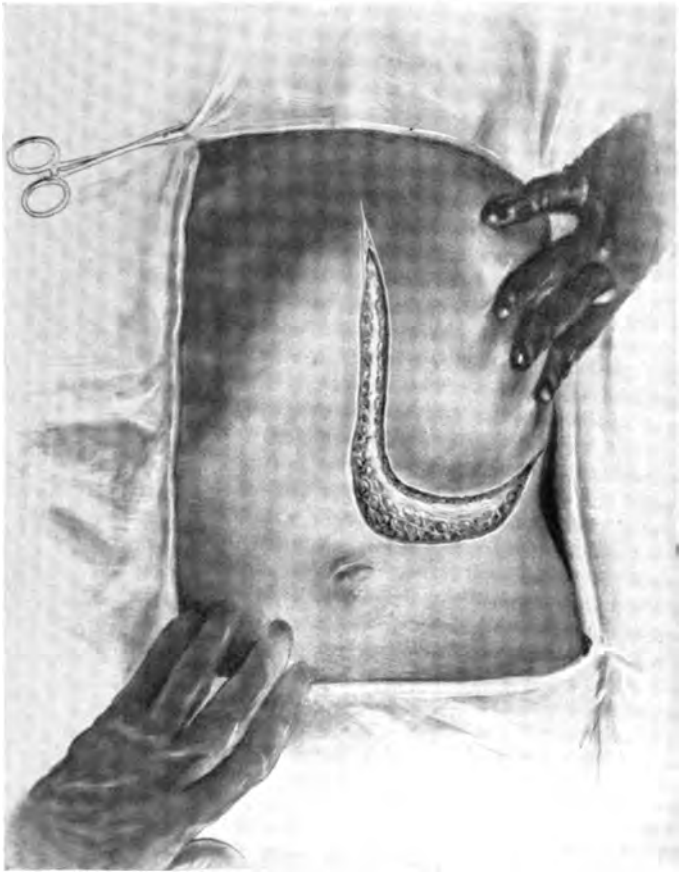


FIG. 2.

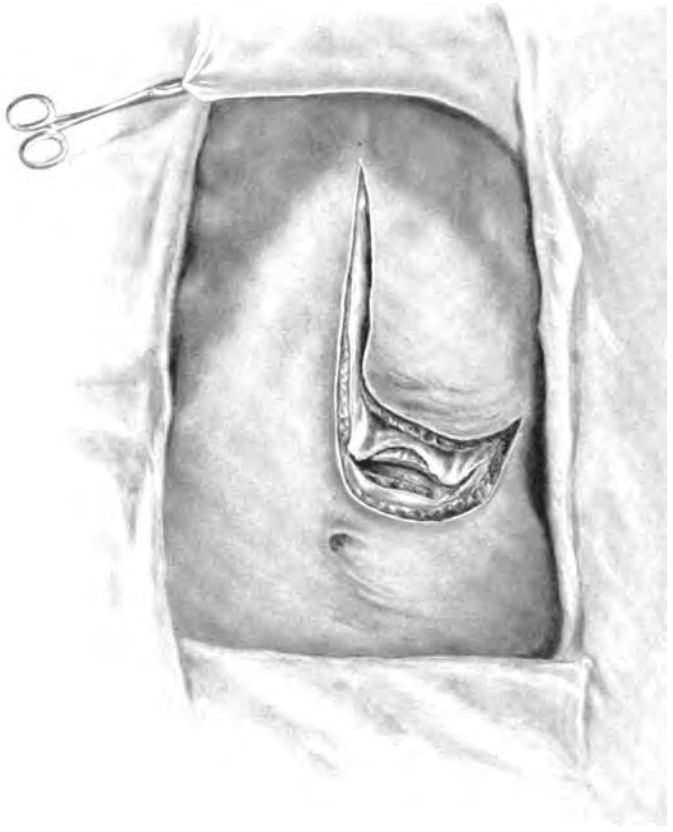


FIG. 3.

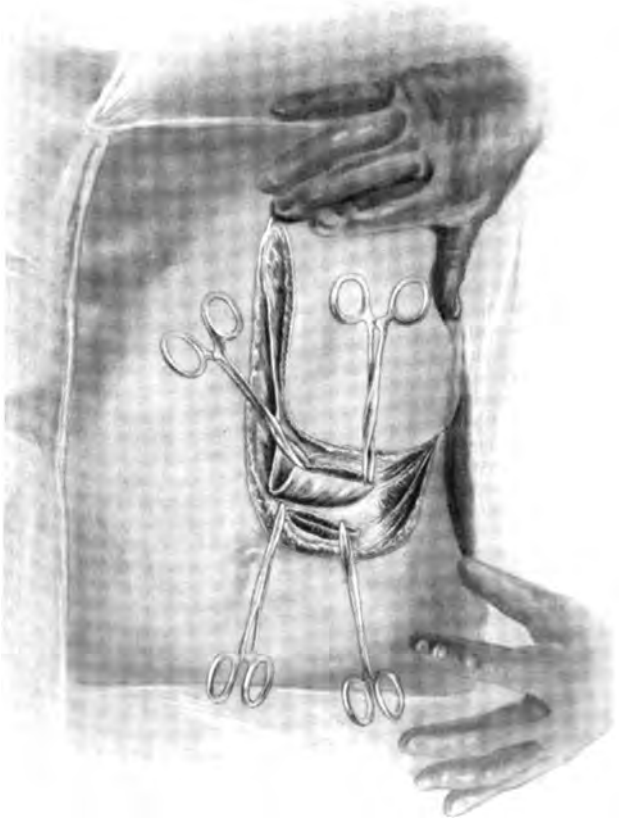


FIG. 4.

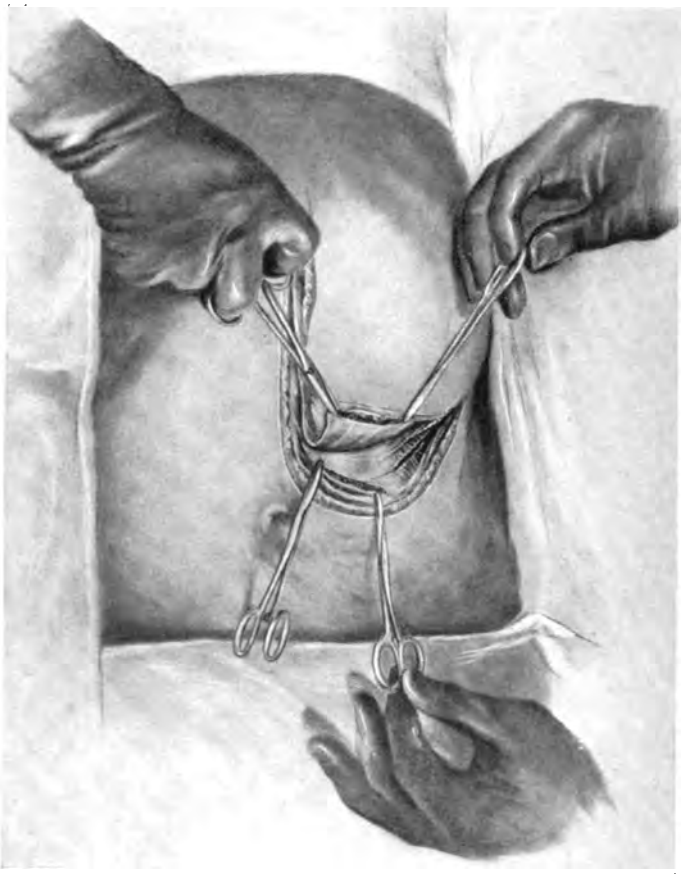
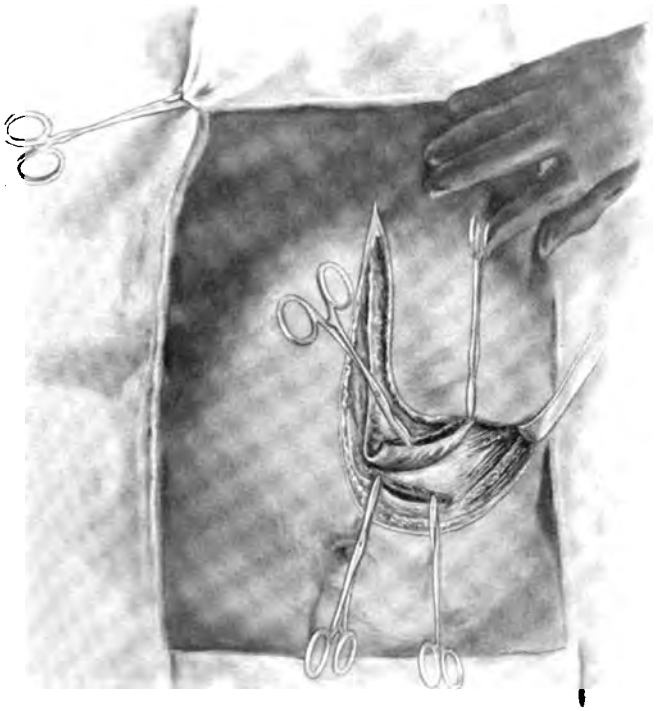


FIG. 5.



In splenic anæmia, splenectomy during the first and second stages gives far better results than any other form of treatment. The production of toxins which affect the blood, liver and other organs is stopped and nature is given a chance to repair the damage already done to the rest of the system. The anæmia rapidly decreases. The resistance of the red blood cells is increased to above normal. The icterus and gastro-intestinal symptoms disappear and weight and strength are regained. The effects of the removal of the spleen in this condition resemble closely those following thyroidectomy for exophthalmic goitre. In both, the secondary and presumably the major, but not the primary source of intoxication, is removed, and the resultant marked improvement in the patient's health, while perhaps not so rapid, is as sure and as permanent from splenectomy as from thyroidectomy.

In cases reported up to the year 1908 splenectomy in the first or second stage gives a mortality of 17 per cent. This figure is based on the reports of Simmonds and Torrance, who collected in all thirty-five cases, with only six deaths. From 1908 to 1912 the results have been even more favorable. Forty-seven cases have been reported by various authors in which splenectomy has been performed before the appearance of Banti's symptom-complex. Of these cases only 5 or 10 ⅞ per cent. have died. It is impossible to state with absolute confidence that splenectomy gives a permanent cure, but at least it can be said that the majority of these cases have been followed for 1 to 9 years after operation and a few have been under observation for 12 years and in no case has there been a return of the previous symptoms.

A case reported by Banti may be cited as typical of the great improvement brought about by early splenectomy. The patient, fifty years of age, with a history of typical symptoms lasting over a period of fourteen years, was operated upon in February, 1903. In October, 1902, the blood count showed Hb. 60 per cent.; R.B.C. 3,760,000; W.B.C. 8,180. In February, 1903, just before operation, the count was Hb. 25 per cent.; R.B.C. 1,615,000; W.B.C. 7,060. One week after operation Hb. was 35 per cent.; R.B.C. 2,565,000; W.B.C. 26,000. Ten months later Hb. and R.B.C. had reached normal and W.B.C. were down to 11,000. The counts taken two, five and nine years later were normal in every respect. In 1911, when the last count was taken there had been no return of symptoms.

In the third stage however, we find a very different story. Here the results of a chronic poisoning have asserted themselves. The vital organs are showing signs of permanent degeneration. The liver has become cirrhotic and we are dealing not with an anæmia alone, but with a degenerative process affecting many organs. Nothing we can do to the spleen will restore the liver cells already destroyed, or prevent the symptoms arising from such destruction. All that we can hope for is to prevent further degeneration. For this reason splenectomy in the third stage has given much poorer results than in the early stages, although a few isolated cases have been reported showing complete recovery in early third stage following splenectomy. In 1908 Simmonds reported six cases following splenectomy, with four deaths, and Torrance five cases with four deaths, giving an average mortality of 72.7 per cent. In the past four years sixteen cases are reported with 9 deaths, giving a mortality of 56 ¼ per cent.

It is perhaps unfair to place too great reliance on the statistics just given. In a disease as rare as the one under discussion, it is extremely unusual to find more than one or two cases reported by a single author. Only the very largest clinics can report a sufficient number of cases to make the statistics reliable. The majority of the single cases reported show a favorable result and it is only reasonable to suppose that many terminating unfavorably have not found their way into literature. However, in the past two years, two articles reporting a series of cases have been published and on this account should perhaps deserve special mention. Mayo, in 1910, reports ten cases of splenectomy for enlarged spleen. Seven of these were undoubtedly splenic anæmia, five being in the early stage, and two in the terminal stage; of these, one resulted fatally and was a case showing advanced cirrhosis of the liver and ascites. The other article by Urbino comes from Burci's Clinic, in Florence, and reports seven cases in the early stages with one death, and two cases in the terminal stage, both of which died.

In the past few years the combination of splenectomy and Talma's operation has been suggested and successfully accom-

plished. It undoubtedly holds out more chances for permanent relief and would seem to be the most rational procedure in the ascitic stage. The attempts are yet too few to state the results with any accuracy.

Complications.—The convalescence following splenectomy varies in no way from that following any operation on the upper abdomen except in two points: First, the danger of gastro-intestinal hemorrhage and second, the lowered resistance to any secondary infection. Hemorrhage into the stomach or upper intestines is the most common as well as the most severe complication. It may occur immediately after operation or at any time during the first two weeks. The loss of blood may be rapid, or as is usually the case, the leakage may be slow, persistent oozing from the mucous membrane of the stomach and upper intestines. It is the most common cause of death in the first two weeks. The frequency of its occurrence cannot be readily estimated. Urbino lays emphasis upon it and noticed it in five out of seven cases. Mayo mentions it as a cause of death in the only fatal case in his series. The treatment consists in absolute rest of the upper gastro-intestinal tract; injections of saline, vasoconstricting drugs, blood serum, or even direct blood transfusion. A case of Dr. Donald Guthrie's, operated upon during the past year, may be cited as typical of this condition.

This case, a young adult male, was seen by Dr. Guthrie in November, 1911. The examination at this time showed a case of splenic anæmia of three years' duration, in the second stage. The blood picture gave a hæmoglobin of 46 per cent.; R.B.C. 4,230,000; W.B.C. 5,600. The lower border of the spleen extended into the pelvis. Splenectomy was performed. During the first five days after operation the recovery was uneventful. At this time he began to pass small quantities of blood by the bowel. This lasted two days and was controlled by injections of normal blood serum. Patient then had no trouble for one week and was able to be up and about in a wheeled chair. At that time he again had attacks of bloody diarrhœa and in spite of saline and blood serum injections the hemorrhage recurred and he died three days later. A post-mortem was made. The vascular pedicle of the spleen was found to be in good condition. In the lower half of the small intestine the mucous membrane showed innumerable small hemorrhagic areas. The mucous membrane of the stomach and of the upper small intestine showed congestion but no hemorrhage.

The danger of secondary infection can perhaps be classed as a post-operative complication. There is undoubtedly a lower resistance to any of the pathogenic organisms, which lasts for months after the removal of the spleen. This fact has been amply proven both by the clinical findings and by experimental work on splenectomized animals.

A case operated on by Dr. C. H. Frazier, in the Hospital of the University of Pennsylvania, during the past winter, is a good example of this condition. A colored girl, aged eighteen years, with a splenic anæmia in the first stage was operated on and the spleen removed. Her convalescence was uneventful until the tenth day when she began to run a temperature and developed signs of pneumonia and pleural effusion at the left base. This was followed three days later by an empyema which was tapped and a few ounces of sterile turbid fluid removed. The chest condition gradually improved. Thirty days after the operation she began to develop signs of gastro-intestinal disturbance with vomiting, distention, etc. Three days later dulness in the flanks developed. The abdomen was opened in the suprapubic region and a large pocket of thick, green, foul-smelling pus evacuated. The upper abdomen seemed to be walled off from this pocket. Six weeks later the patient's general condition had markedly improved, although there was still slight drainage from the abdominal wound.

Conclusions.—1. Splenic anæmia is a disease entity characterized by a definite symptomatology and pathological picture, and the so-called "Banti's disease" is its terminal stage.

2. In all probability the primary cause of splenic anæmia is a toxæmia, the origin of which is to be sought for outside the spleen itself, possibly in the gastro-intestinal tract, but the exact nature of which is as yet unknown. This toxin probably acts primarily or secondarily on splenic cells, causing an hypertrophy and increased activity.

3. This increased splenic activity is responsible for the anæmia probably through a decreased resistance of the red blood corpuscles. The icteric pigmentation is also due to an increased hæmolysis.

4. Although the thrombophlebitis of the splenic and portal veins is a frequent finding clinically, we believe, from our own experimental work and that of others, that it is not an essential factor in the etiology. If the theory of a primary toxin

is correct, we suggest that the blood coming from the spleen to the liver should be doubly toxic in that it contains not only the primary toxin, but that also elaborated by the splenic substance. To our minds it is the action of this doubly toxic blood that causes the endophlebitis of the splenic and portal veins with its consequent thrombosis.

5. In all probability these same toxic factors play the important rôle in the production of liver cirrhosis, although the mechanical factor of congestion of the portal system may be an additional cause.

6. Up to the present time treatment other than surgical has yielded only temporary benefit.

7. Splenectomy in the first and second stages offers us our only chance of permanent cure. The mortality is 12.5 per cent.

8. Splenectomy in the third stage will arrest the further development of the disease but will not cause a retrogression of the liver cirrhosis. In a few isolated cases of early cirrhosis permanent cure has followed removal of the spleen. In the past five years mortality following splenectomy when done in the third stage has been 56 ¼ per cent. The combination of splenectomy and Talma's operation should be the procedure of choice in this stage.

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DR. ASTLEY P. C. ASHHURST said that the incision in the abdominal wall described by Dr. Willard is very similar to that on the right side which has been employed for many years by Czerny for operations on the gall-bladder and bile-ducts. It has now been adopted by Kocher as his "normal incision" for difficult cases. The extension of the incision outward beyond the semilunar line corresponds to the incision of Kausch and certainly presents many advantages. He had one occasion himself, four years ago, to explore the cardiac orifice of the stomach and the spleen in a case of gunshot wound. The injury involved both the thorax and abdomen. He made first an incision in the line of the eighth left intercostal space, splitting the abdominal wall in a direction parallel to the nerves. Then the diaphragm was incised sufficiently to expose freely, after retraction of the eighth rib upward and the ninth rib downward, the region above the fundus of the stomach, including the spleen, the left lobe of the liver and the pancreas. Later the lung was exposed. He secured ample exposure by this incision, the abdominal portion of which corresponds to Kausch's, and to the horizontal limb of the incision advocated by Dr. Rodman and Dr. Willard; but unfortunately the patient did not recover.

DR. GEORGE P. MÜLLER reported the following case which he believed to be one of splenic anæmia. The history is as follows: A man, aged thirty, was awakened on the morning of August 26, 1912, by a feeling of nausea without pain. He then vomited a pint of blood and some hours later vomited again a lesser amount. Between the two vomitings he was treated by rest, ice, morphine and calcium lactate. He was sent to the University Hospital by his physician, Dr. Janvier, at about 7 P.M. The patient was a healthy baby until about two years of age, when he had some vague stomach or bowel trouble, accompanied by considerable pain and diarrhœa. This lasted for 3 or 4 weeks and he then recovered quite completely. During boyhood and up to the age of 18 years of age he was apparently well, although always of slim build and having a very white skin. Shortly after this time he had an attack of severe abdominal cramps with profuse diarrhœa after eating freely of oysters, ice cream and peaches. A few days later he had a similar attack and from this time on he has never been entirely well or free from pain and abdominal discomfort. The slightest indiscretion in diet brings on an acute

attack of indigestion with abdominal pain and diarrhœa. About a year ago he had a severe attack of abdominal pain and diarrhœa associated with bladder trouble, jaundice, high fever and chills. He was in bed a week or ten days and became reduced in weight and strength. Has had no attack since then but has frequently had sick headaches occasionally associated with vomiting and also with pain in lower abdomen and with diarrhœa. After the attack 4 or 5 years ago he had a small hemorrhage from the bowel, but the diagnosis at that time was never definitely made, the condition being counted as a chronic enteritis. Patient is well built, rather fat, active mentally, and occupying a good clerical position. Upon examination shortly after admission the abdomen was flat without rigidity, masses or tenderness. The liver was not enlarged but the spleen seemed slightly so and was palpable one inch below the costal margin. Blood count made at the time revealed: Hæmoglobin, 67 per cent.; red blood corpuscles, 3,730,000, and a leucocytosis of 12,700, of which 81 per cent. were polynuclear. After his admission to the hospital he continued the line of treatment which Dr. Janvier had begun and in addition gave 20 c.c. of horse serum. At 10 P.M. he vomited 16 oz. of blood and, accordingly, it was thought that his stomach should be explored. Incision in right rectus close to the median line and extending from the ensiform to the umbilicus. Upon opening the abdomen the pale intestines and stomach presented nothing noteworthy and upon exposing and drawing the stomach into the wound, no evidence of ulcer could be detected, there being no points of thickening, scars, adhesions, no milk-white patches. The lymph nodes in the greater and lesser curvature were not enlarged. The liver was normal in appearance with thin, sharp edges. The gall-bladder was slightly distended but not abnormal. The spleen was large, measuring about 8 by 5 inches in size, quite firm to the touch and smooth. It was closely approximated to the fundus of the stomach. A small vertical incision was made in the stomach and considerable blood, both fluid and clots, evacuated. A small proctoscope was introduced and the stomach searched from one end to the other for ulcers, erosions or other evidence to show the origin of the hemorrhage. Absolutely nothing was found. In places the mucous membrane appeared congested but nowhere was there evidence of active bleeding. The wound in the stomach

was closed with catgut and linen and the abdominal one with catgut and silkworm gut. Patient stood the operation fairly well, but was given 750 c.c. of normal saline solution intravenously at the close of operation.

Patient recovered fairly well from operation without much shock. Temperature gradually declined and reached normal on the sixth day after operation. Thirty-six hours after operation a blood count revealed hæmoglobin, 40 per cent., and red blood corpuscles, 2,910,000. The fat in the wound showed signs of infection and four or five of the stitches were removed and several ounces of pus evacuated, after which the condition of the wound remained uneventful. At the end of the first week the patient began to pass considerable dark, foul-smelling material from the bowels, the passage of which was controlled by the administration of opium and resorcin. On September 3, seven days after operation, the blood count revealed a hæmoglobin of 24 and red blood corpuscles of 4,470,000. It was evident that the black material coming from the bowel was due to intestinal bleeding and was not a residual of the stomach hemorrhage. More horse serum and certain other drugs were given, after a consultation with Drs. Stengel and Klaer, but made no impression on the patient who became delirious and died on September 9, thirteen days after the operation. No autopsy was allowed but the reporter had always believed this patient was suffering from splenic anæmia, caused by some intestinal toxæmia.

He was especially anxious to report this case because of the error in diagnosis which was made, that it was a case of hæmatemesis from gastric ulcer, based on the history of gastric distress followed by the vomiting of blood. At the time of operation a full history was not obtained but the diagnosis seemed clearly evident, especially in view of the fact that the the patient was practically a total abstainer from alcohol and there was no evidence of syphilis or other disease of the liver. It was the intention to perform a splenectomy when the patient's condition permitted and the family were so informed at the close of operation, but the occasion never arose.

REPORT OF 105 CASES OF STRANGULATED HERNIA.

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THE 105 cases of strangulated inguinal, femoral, and umbilical hernia herewith reported were operated upon at the Episcopal Hospital within the past six years. Covering as it does quite a large number of cases, and the work of several years, an analysis of these cases, I thought, might prove of interest to the Academy.

It is strange that in the past two decades of progressive surgery, in an era unparalleled by the great strides made in the art of diagnosis, operative technic, and after-treatment, to say nothing of the help obtained by educating the public to surgical conditions, that little has been done, either directly or indirectly, to lower the death rate of this condition.

The onward march of surgery has been from the pelvis upward, until that great divide, the diaphragm, was reached. This obstruction has caused a momentary halt in the advancement of visceral surgery, although a few have crossed over and invaded the thoracic cavity. During this momentary lull in visceral surgery some few have turned back to the old things that were long considered a dead issue. We have but to recall to mind the magnificent work of Lane and Murphy on bones and joints to realize that this momentary pause has been a blessing in disguise. Let us hope that others will turn back to the "dead issue" and that strangulated hernia will be considered.

The mortality of strangulated hernia remains to-day what it was ten or even twenty years ago. It is, unrelieved, an acute progressive abdominal condition, and the mortality will

STATISTICS.

Inguinal Hernia

Right			Left		
Male	34	Rec.	Male	6	Rec.
Female	3		Female	1	
Male	10	Died	Male	2	Died
Female	3		Female	1	

Recovered 44, died 16, not operated (died) 3, total.....63.

Femoral Hernia

Right			Left		
Male	0	Rec.	Male	1	Rec.
Female	12		Female	5	
Male	1	Died	Male	0	Died
Female	4		Female	2	

Recovered 18, died 7, not operated (died) 2, total.....27.

Umbilical Hernia

Right			Left		
Male	3	Rec.	Male	1	Died
Female	8		Female	8	

Recovered 11, died 9, not operated (died) 1, total.....21.

Ages of Patients.

Age.	Inguinal.		Femoral.		Umbilical.		Mortality.
	Rec.	Died.	Rec.	Died.	Rec.	Died.	
1–10	5	1	..	..	..	..	16 per cent.
10–20	4	1	1	..	..	..	16 per cent.
20–30	8	0	4	..	..	..	0 per cent.
30–40	8	3	3	2	3	1	30 per cent.
40–50	7	3	4	2	3	3	36 per cent.
50–60	3	4	3	1	4	3	44 per cent.
60–70	8	2	1	1	1	2	33 per cent.
70–80	1	2	2	1	..	..	50 per cent.
Total..	44	16	18	7	11	9	

Number of Hours Strangulated.

Hours.	Inguinal.		Femoral.		Umbilical.		Mortality.
	Rec.	Died.	Rec.	Died.	Rec.	Died.	
1–12	21	1	7	0	4	1	5 per cent.
12–24	17	7	8	2	5	3	28 per cent.
24–48	3	2	2	2	2	1	41 per cent.
48–	3	6	1	3	0	4	76 per cent.
Total..	44	16	18	7	11	9	

Mortality

Inguinal	Per cent.	Femoral	Per cent.	Umbilical	Per cent.
Right inguinal, male,	22	Right femoral, male,	100		
Left inguinal, male,	24	Left femoral, male,	0		
Right inguinal, female,	50	Right femoral, female,	25		
Left inguinal, female,	50	Left femoral, female,	28		
Inguinal mortality:		Femoral mortality:		Umbilical mortality:	
Male	23	Male	50	Male	25
Female	50	Female	26	Female	50
Total inguinal mortality: Male and female	26	Total femoral mortality: Male and female	28	Total umbilical mortality: Male and female	45

Total Mortality

Male	24 per cent.
Female	38 per cent.
Male and female	30 per cent.

continue to remain unchanged until the profession and public, as in appendicitis, and as we are trying to do in cancer, become better educated to the fact that an early operation and the avoidance of taxis in hernias are the two most important factors in lessening the mortality.

Unfortunately, we cannot in strangulated hernia as in appendicitis with peritonitis, "dry dock our patient for repairs," and hope for an abeyance of the symptoms, and after localization of the inflammatory process, a successful operation and ultimate recovery of the patient.

Of the 105 cases of strangulated hernia, 58 were males and 47 were females. Sixty were inguinal, 25 femoral, and 20 umbilical. During this period of six years 848 cases of hernia of the above types were operated upon at the Episcopal Hospital. Three of this number were strangulated. Six cases of strangulated hernia were admitted to the hospital in a moribund condition and no operation was attempted.

The great majority of these cases of strangulated hernia gave the usual history of having had the hernia for years and of wearing an ill-fitting truss. The symptoms varied. Among the most constant were sharp pains at the hernial orifice, colicky pains, nausea and vomiting. So constant were the last three symptoms mentioned, paroxysmal pain, nausea and vomiting, that when not reported on the histories, I believed

their absence due to an error on the part of the history taker in failing to record them. In almost every instance taxis had been tried by the patient and also by the attending physician.

The ages of the patients ranged from six weeks, the youngest, to eighty years, the oldest; and the duration of the hernia, non-strangulated, from two weeks to sixty years.

The condition of the patients on admission to the hospital varied, depending almost entirely upon the degree and length of time the hernia had been strangulated. The cases admitted in shock, with a subnormal temperature, fast pulse, etc., showed the highest mortality.

The diagnosis is usually easy. Only in one or two instances were the patients referred to us with an incorrect diagnosis, and these were either in small femoral hernias, or in inguinal hernias in which the attending physician recognizing a surgical condition had failed to make a thorough examination. In two instances the condition was mistaken for an appendicitis with diffuse peritonitis.

In any patient giving a history of a hernia coming down and not being able to reduce it, or an irreducible hernia becoming larger and more tense, and if either condition is followed by pain at the hernial orifice, colicky pain, nausea, and vomiting, a strangulated hernia should immediately be thought of. Speculations as to whether the hernia is irreducible, obstructed, inflamed or strangulated, depending on the passage of flatus, percussion sound, or the presence or absence of peristalsis in the hernial sac are misleading and the delay in waiting for such symptoms jeopardizes the life of the patient. The redness of the skin and the exquisite tenderness of the hernia, I believe, is often due to local applications, and too prolonged or vigorous taxis.

One often wonders, after operating on a case and seeing the hernial sac full of blood, and the bruised and injured condition of the intestine, if it would not have been better, certainly since the perfection of hernial surgery, if our medical schools would cease to teach the practice of taxis except in a few limited instances. Taxis, like all good things, is often

overdone. It no doubt saves many from being operated upon, and in instances when surgical aid is unobtainable, is frequently a life-saving device. It, nevertheless, causes the death of many. Certainly, in a large percentage of the cases in this series, can the mortality be attributed to the delay caused by taxis.

Simple taxis, or taxis aided by an anæsthetic, morphine injection, local application, hot bath, elevation of the foot of the bed, etc., often prolongs for hours or days the question of operative interference. So important was the question of taxis, and so dangerous its use, that the old surgeons tried to place a limit of time, varying from a few minutes to two hours, during which it was safe to persist in its use. Coley does not think it wise to continue taxis for more than five minutes. The same author believes that taxis should never be attempted in those cases " in which the symptoms of strangulation have been exceedingly severe from the start," in those " in which strangulation has occurred in previously irreducible hernias," and in cases " in which 24 hours have elapsed after strangulation."

The preparation of the patient for operation should be the same as for the usual emergency case. If the patient is vomiting, and especially if the vomitus is fecal in character, the stomach should be washed out. It is believed that the washing out of the stomach of these cases lessens the chance of an aspiration pneumonia, especially if ether be the anæsthetic.

The anæsthetic used depends largely upon the operator, the condition of the patient, and the technic to be employed. In the above cases, ether, chloroform, local and spinal anæsthesia were used.

Ether, I believe, in the majority of instances, is the anæsthetic of choice, and if used, should be preceded, in the cases with fecal vomiting, by washing out the stomach.

Chloroform has a very limited use, but occasionally can be used advantageously in cases with kidney or lung complications.

Local anæsthesia is of advantage in cases complicated by

cardiorenal and lung conditions. The increased length of time required to perform the operation under this anæsthesia, and the mental shock often accompanying it, are its chief disadvantages.

Spinal anæsthesia has a very limited use. I believe it to be a dangerous anæsthetic, even in the hands of the most expert, and certainly should be used cautiously in the aged and in cases of long duration.

Unless absolutely contra-indicated ether is certainly the anæsthetic of choice, and from the deaths recorded in this series, is not complicated by pneumonia or uræmia any more frequently than any of the other anæsthetics mentioned.

The operation performed must relieve the constriction, and if the gut is not gangrenous, return it to the abdominal cavity. If the gut is not viable, and that often is a very hard question to decide, one of the following procedures must be adopted.

If it is a border line condition and you are unable to decide whether the gut is gangrenous or not, it can be left in the wound for 12 to 24 hours until all doubt has passed.

If the gut shows a small gangrenous area we have been quite successful by invaginating this spot. If a large gangrenous area is encountered this portion of the gut must be resected and either a lateral or end-to-end anastomosis performed, the latter preferred, or an artificial anus can be established. Our statistics favor the resection method. In either instance the cases are most unfavorable and the mortality is very high.

One of the most remarkable recoveries in this series was in a woman, 50 years of age, with a hernia of the femoral variety involving the small intestine, which had been strangulated for four days. An artificial anus was established, which later healed spontaneously.

In strangulated hernias of the so-called Richter type, or partial enterocele, either femoral or inguinal, the mortality is high. This is probably due to the fact that these hernias are small, are often overlooked, and consequently come to the

surgeon late. In this type, on three occasions, the hernia reduced itself while the patient was being anæsthetized. In the first case, a herniorrhaphy was performed without exploring the gut; the patient died in two days of a diffuse peritonitis and the autopsy showed a perforated gangrenous area the size of a penny. In the other two cases, at the time of operation, the gut was caught and pulled down into the wound, thus ascertaining the true condition of affairs.

The hernias that reduce themselves while the patient is taking an anæsthetic, especially if the strangulation is of several hours' duration, and the gastro-intestinal symptoms have been marked, should be operated upon, as the true condition of the gut should be ascertained. One of the umbilical hernias in this series reduced itself while the patient was being anæsthetized, no operation was performed. The patient died a day or so later and the autopsy showed a volvulus of the ascending colon.

In the majority of instances of strangulated hernia there is little doubt as to the viability or not of the intestine. In some cases, however, the decision would tax the judgment of the most expert. The best test for the viability of the intestine is the application of compresses wrung out in hot saline solution, although help can be obtained by noting the loss or not of the glossy appearance, whether the gut is dull and granular, whether its color is red, purple, black, grayish or mottled, and also if the gut is firm and elastic or soft, flabby and collapsible.

Kocher lays great stress on the pulsation of the vessels of the mesentery and bowel.

If resection is to be done the excision should go wide of the gangrenous area, especially is it important to do so to the proximal side and get beyond the distended and water-logged portion into healthy bowel.

In all cases operated upon the ideal operation is a herniotomy and herniorrhaphy. Unfortunately, this is not possible in some, as the physical condition of the patient may only warrant the relief of the constriction. In large scrotal and

umbilical hernias, on account of the many and dense adhesions, it is often best to simply relieve the constriction. Especially is this true if the patient's condition does not warrant a prolonged operation.

In this series 32 cases died. The cause of death was as follows: Peritonitis, 11; uræmia, 3; pneumonia, 4; shock, 5; myocarditis, 2; delirium tremens, 2; apoplexy, 2; acute dilatation of heart, 1; volvulus, 1; sarcomatosis, 1. The five cases that died of shock were desperate ones and really should be classed under deaths due to peritonitis.

The prognosis depends almost entirely upon the degree and length of time the hernia has been strangulated and the resisting power of the patient.

The type of hernia, the sex and the age seems to have some bearing on the mortality. The death rate is lowest in children and highest in the aged. The mortality is higher in femoral than in inguinal and is highest in the umbilical.

The reduction of the mortality of strangulated hernia, as stated earlier in this paper, depends upon the education of the public and shall I say, profession, to the importance of early operation and the avoidance of prolonged and vigorous taxis.

The prevention of strangulated hernia is likewise an educational one. By educating the public to the danger of a simple hernia becoming strangulated, and the importance of having the hernia operated upon, or if operative treatment be refused, the wearing, under the supervision of a physician, a properly fitting truss will reduce the number of this almost inexcusable condition.

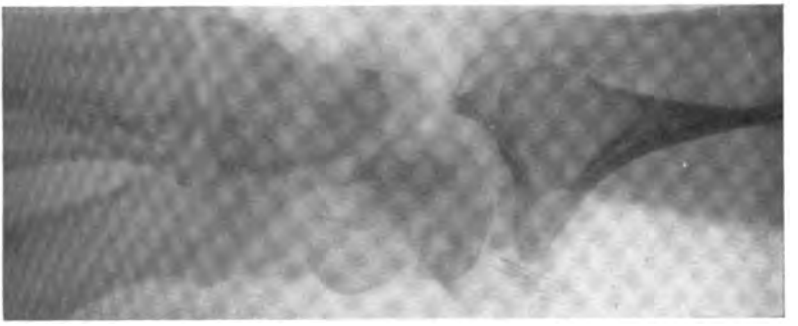
In closing, I wish to make a plea that all simple hernias in children, and especially those of the scrotal type, be operated upon. I believe it is wrong to order a truss for these cases, when we know the percentage of cures is very small. If a radical cure be performed in these cases, we have taken a great step toward the prevention of strangulated hernia.

FRACTURE OF CARPAL SCAPHOID AND CUNEIFORM WITH LUXATION OF SEMILUNAR; BLOODLESS REDUCTION; FUNCTIONAL RECOVERY.

DR. P. G. SKILLERN, JR., reported the history of a man, aged twenty-seven, who fell upon his hyperextended left hand. Examination shows swelling around wrist-joint with tenderness in snuff-box and in front of carpus, and also a bony prominence in wrist beneath flexor tendons with shortening from tip of radial styloid to base of first metacarpal. Diagnosis of fracture of scaphoid with luxation of semilunar. Skiagram (Fig. 3) shows separation and displacement of scaphoid tuberosity between tip of radial styloid and trapezium; luxation of semilunar, and fracture of cuneiform. Lateral view (Fig. 1) shows displacement of semilunar into palm with rotation about its transverse axis, so that its concavity faces dorsally, and also a hollow on dorsum between posterior articular edge of radius and os magnum. The patient was immediately anæsthetized with nitrous oxid and manual reduction effected by the method described by Codman and Chase (ANNALS OF SURGERY, 1905, xli, 321, 863). Dressed on Bond splint. Skiagram (Fig. 2) now showed complete reduction of semilunar and (Fig. 4) improvement in position of fragments of scaphoid and cuneiform. After reduction the patient complained no longer of pain. Immobilization was maintained four weeks *without massage and without passive motion.* It must be remembered that this bone is but feebly capable of osteogenesis and is constantly bathed by the synovial fluid, the amount of which would only be increased by massage and passive motion, thus adding fuel to the fire. There being union at the end of four weeks, careful massage and passive motion rapidly restored complete function to the wrist-joint.

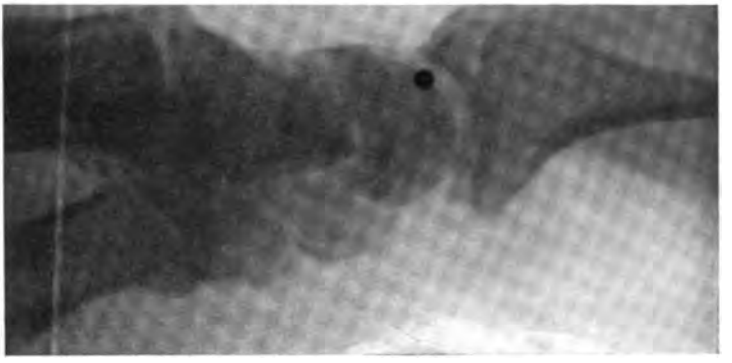
A similar case has been described by Corner (*Trans. Clin. Soc.*, London, 1905, xxxviii, p. 224) but the semilunar was fractured, in addition to being luxated, and the cuneiform showed fracture of the portion nearest the radius which damage, as Corner states, was obviously done by the lower end of the radius being forcibly driven on to the carpus. This was shown by close proximity of ulna to cuneiform. This injury was of eighteen month's duration, and had not been reduced. The patient complained that his hand was weak and occasionally painful, and that it had lost some of its power of movement.

FIG. 1.



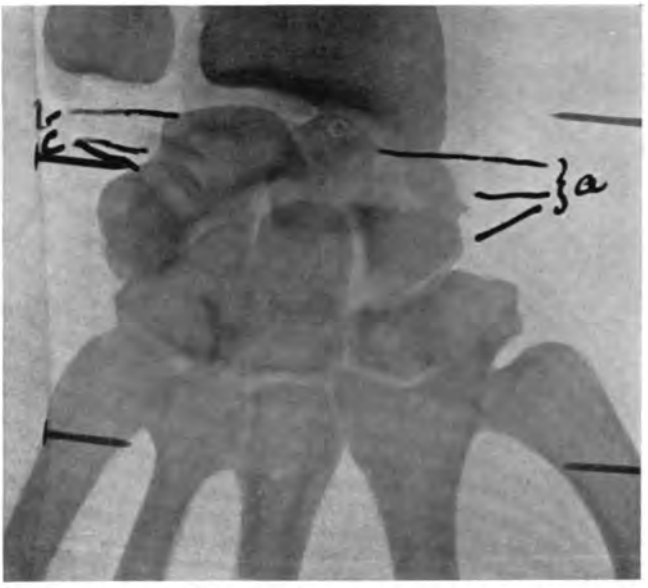
Lateral view before reduction. Note displacement of semilunar anteriorly beneath os magnum; also, shortening of carpus by approximation of metacarpus to radius and ulna.

FIG. 2.



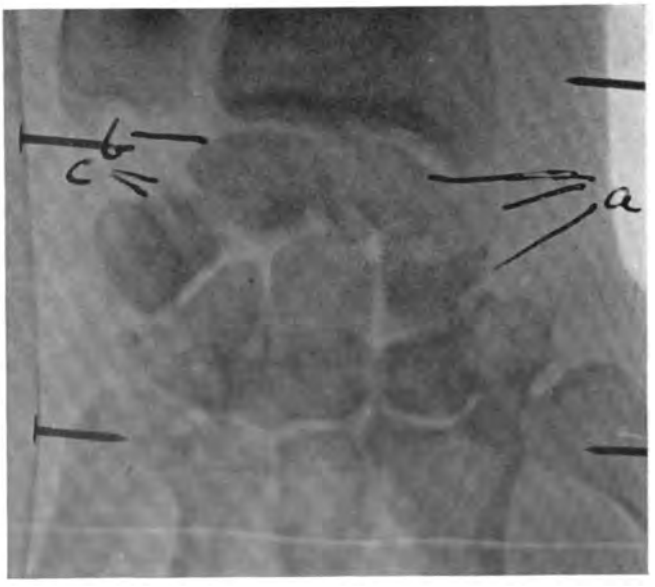
Lateral view after reduction. Note restoration of scaphoid to its normal position with consequent lengthening of carpus.

FIG. 3.



Anteroposterior view before reduction. Note *a*, tripartite fragmentation of scaphoid; *b*, displacement of semilunar; and *c*, fracture of cuneiform.

FIG. 4.



Anteroposterior view after reduction. Note *a*, improvement in position of scaphoid fragments; *b*, restoration of semilunar, and *c*, of fragment of cuneiform.

The results of neglect or of improper management of this injury is further illustrated by an old case of undiagnosed and therefore unreduced fracture of scaphoid with luxation of semilunar recently described by the author (*Jour. A. M. A.*, 1913, lx, p. 1536). This patient had injured his right wrist seven months previously in a street accident, and had been treated in another hospital for two weeks as a "bad sprain," skiagram taken at the time being declared negative. Since then the wrist was crippled, flexion and extension being very limited, and patient had pain in the joint on the slightest motion. Owing to the long lapse of time (seven months) since the injury, but one method of treatment was indicated, removal of the semilunar with both fragments of scaphoid. This was done, with relief of pain and increase of motion.

Fracture of the scaphoid with or without luxation of the semilunar is not a rare injury, and readily lends itself to clinical diagnosis by localized "wincing" tenderness in the snuff-box with, in case the semilunar be luxated, a distinct bony prominence in front of the carpus beneath the flexor tendons. This injury should be suspected with the same facility with which Colles' fracture is suspected and the same careful anatomical reduction should be made in order to avoid crippling of the wrist of a wage-earner and subsequent liability for damages which in such an instance seems glaringly justifiable.