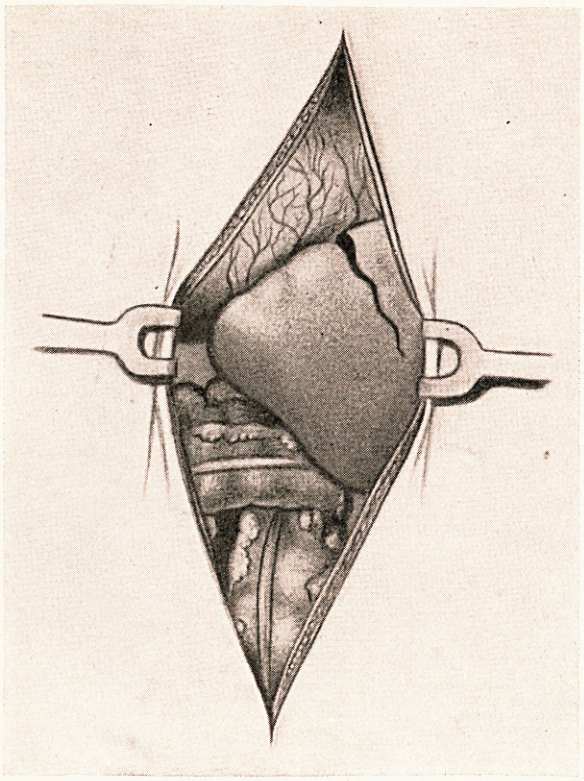

Fig. 1.—The abdomen is opened and the edges of the wound are retracted showing the rupture on the anterior border and the diaphragmatic surface. On account of the operative position of the patient the enlarged spleen has moved more towards the middle line than is usual even for an enlarged spleen. Note how it overlies the stomach.

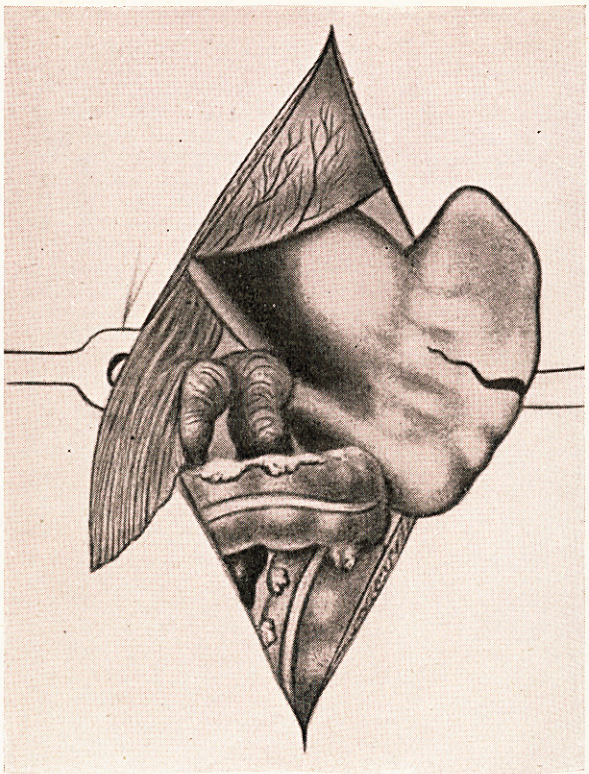

Fig. 2.—The spleen is rotated on its longitudinal axis with its anterior border lying outside the left edge of the operation wound. The rupture on the gastric surface is now well seen. Note the tense and swollen appearance of the pedicle with its peritoneal covering owing to accumulation of blood clots inside.

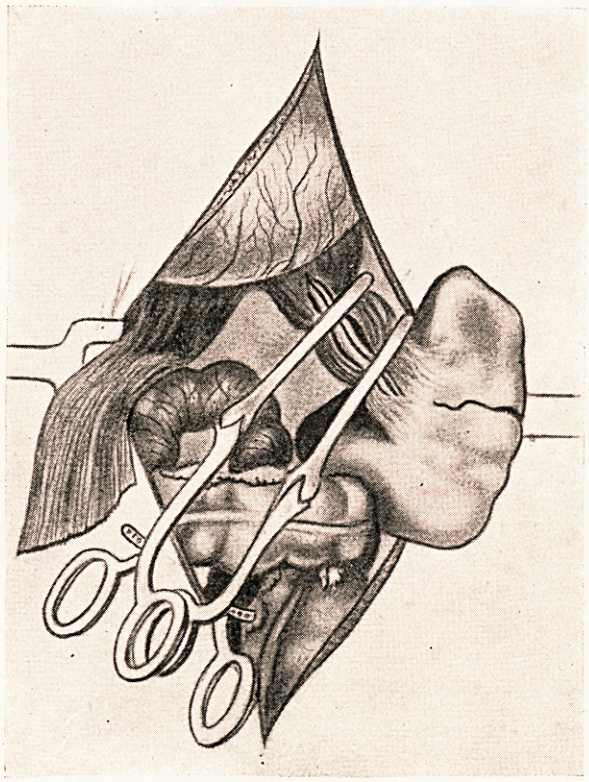

Fig. 3.—The capsule over the pedicle is opened, the clots evacuated and two clamps applied—one close to the spleen and the other immediately to the left of the commencement of the splenic ligaments.

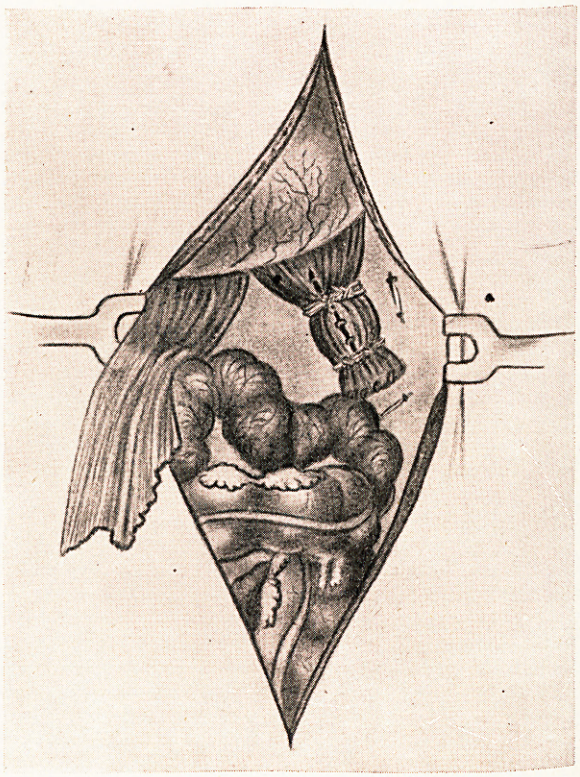

Fig. 4.—The spleen is removed, the stump of the pedicle is peritonized and the divided adhesions are stitched up.

THE SURGERY OF THE RUPTURED SPLEEN

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The normal spleen

THE normal spleen is deeply placed in the abdominal cavity and protected from injury by its position. It is supported on all sides by important viscera among which it is wedged in. It derives its anchorage from folds of peritoneum usually spoken of as its ligaments.

The oblique longitudinal axis of this somewhat wedge-shaped gland corresponds to the 10th rib and its upper and lower limits, which correspond to the inner half of its anterior border and the outer third of its posterior border, lie behind and parallel to the upper border of the 9th and the lower border of the 11th ribs, respectively.

Its apex lies about 1½ inches from the spinous process of the 10th dorsal vertebra and its base, a small concave and triangular area, rests on the splenic flexure of the colon and the phrenico-colic ligament at the 11th rib about 3½ inches away from the middle line looking downwards, inwards and slightly forwards.

The anterior border sharp, notched and somewhat convex separates its superior or diaphragmatic surface from the gastric half of its inferior or visceral surface.

The posterior border rather straight, thick and rounded lies between the superior surface and the renal half of the visceral surface.

The superior surface smooth and convex rests on the diaphragm, which separates it from those portions of the left lung and pleura occupying the interval between the diaphragm and the 9th, 10th and 11th ribs with the intervening intercostal spaces. This surface looks obliquely upwards, backwards and to the left.

The inferior surface is divided into two unequal halves by a longitudinal ridge extending from the apex to the upper angle of the triangular base. It is the shape of this surface that gives the organ its wedge-like appearance. The upper and larger half of this concave surface, which looks upwards, forwards and inwards, supports a portion of the stomach while the lower and smaller half, also concave but looking downwards and medially, rests on the upper half of the anterior surface of the left kidney and occasionally on the left suprarenal gland also.

The hilus of the spleen, a longitudinal and irregular cleft on its gastric surface about 2½ inches long through which the 5 or 6 main branches of the splenic artery pass into the substance of the gland and a similar number of tributaries of the splenic vein pass out, lies about ¼ inch above and parallel to the ridge dividing the gastric from the renal area of the inferior surface.

The pedicle of the spleen composed of its vessels rests partly on the tail of the pancreas.

The surgical anatomy of the spleen, normal and enlarged

The peritoneal investment of the spleen.—The spleen is completely invested by the peritoneum except for an oval area round the hilus where its blood vessels enter its substance. Here the peritoneum leaves the capsule and lies loosely over the vessels and the pancreatic tail.

The spleen therefore may be said to lie as it were in a peritoneal pouch of which the serous covering of the pedicle forms the neck.

From the gastric and renal surfaces of the spleen the peritoneum passes on to the postero-inferior surface of the stomach and the anterior surface of the kidney, respectively. The angular space left between these two diverging folds covering the stomach and the kidney is lined by a portion of the peritoneal diverticulum, which forms the omental bursa.

In this way is the spleen attached to the stomach and the kidney by two separate double folds of peritoneum, known respectively as the gastro-splenic and the lieno-renal ligaments.

As mentioned before, the triangular base of the spleen rests on the peritoneal shelf, the phrenico-colic ligament, a fold of peritoneum reflected from the splenic flexure of the colon to the left lateral abdominal wall at the 11th rib.

The splenic peritoneal ligaments do not interfere with its rhythmical expansion and contraction nor hamper the temporary increase in its size during digestion, nor its permanent enlargement due to disease.

Under normal conditions, it is possible to pass one's hand round the spleen, *i.e.*, between it and the viscera that surround it.

The cone-shaped peritoneal space, having its base at the hilus and the bare area of the spleen immediately surrounding the hilus and its apex at the point where the splenic peritoneum is reflected over the stomach and the kidney, is occupied by the tail of the pancreas, the lienal vessels and their main branches, as described above, and is capable of considerable distension without tearing as seen in rupture of the spleen when it is often filled and distended with clotted blood.

If a coloured fluid is introduced under pressure through a small opening in the peritoneum over the splenic pedicle, the fluid will be seen to pass through the folds of peritoneum forming its ligaments in the direction of (1) the postero-inferior surface of the stomach where it is soon stopped, and (2) the lateral half of the anterior surface of the left kidney. In the latter situation the peritoneum from over the kidney is more easily lifted up and the fluid may descend along the lateral border of the kidney to the retroperitoneal space behind the descending colon.

In the case of a diseased and enlarged spleen the peritoneum over the kidney is on the stretch and is still more easily raised and enables the fluid to pass into the post-colic retroperitoneal space.

In severe injuries to the spleen the hæmorrhage is partly intracapsular and partly extracapsular. The intracapsular hæmorrhage passes in a similar way through the peritoneal sheath covering the pedicle and between the leaves of the splenic ligaments to the retroperitoneal area behind the descending colon.

Except at the hilus where the spleen capsule, which is described later, is invaginated into it along the blood vessels entering it, the peritoneum is inseparably adherent to the capsule. The spleen is kept in place not only by its peritoneal anchorage but also by the stomach and the kidney pressing it against the diaphragm. Thus a moderate enlargement and increase in weight are not sufficient to dislodge it from its position.

When, however, a spleen is considerably enlarged and heavy there is a risk that its weight and the physical exertions of the individual will dislodge it from its position. When this happens it is rotated through a semi-circle on its antero-posterior axis and is suspended by its pedicle with the diaphragmatic surface looking downwards, the anterior border obliquely directed forwards from left to right, and the base directed downwards and to the right. The pedicle may be still more stretched and lengthened, on account of the increasing weight of the spleen; the organ may descend into the pelvis or even wander about in the abdominal cavity in response to the changes of position of the patient.

The rarity of such an occurrence in Bengal, where spleens greatly increased in size are so very common, is due to the gradual enlargement of the gland and to its contracting firm adhesions with the diaphragm as a result of perisplenitis at an early stage.

Ordinarily, however, the diaphragmatic surface and the anterior border, on account of the enlargement, come to the surface by displacing the colon and the

coils of the small intestine and occupy the whole of the left hypochondriac space from its anterior to its posterior wall. The base descends into the left lumbar region and the notched anterior border also follows the base in its descent; here they may be easily palpated and the movements upwards and downwards of the spleen in obedience to the respiratory excursions of the diaphragm appreciated.

The mechanisms of rotation and of rupture of an enlarged spleen

The mechanism of the descent of the enlarged spleen, which occupies the whole of the left hypochondriac region from its anterior to its posterior wall, involves a rotation on its antero-posterior axis to descend into the lumbar region past the constriction at the waist below the 12th rib.

This rotation changes the direction of the longitudinal axis of the spleen from an oblique to a more or less perpendicular position, and thus places its anterior border at right angles to the anterior costal border.

The enlargement of the spleen extends uniformly in all directions and as a result its anterior border often curves over even the anterior surface of the stomach sometimes almost completely separating it from the anterior abdominal wall on the left.

Rupture of the spleen is caused by the anterior costal border being driven into its anterior border. Hence the tear always lies across the spleen and passes between the bundles of the circularly disposed muscle fibres of its capsule. It may extend both ways into the gastric and diaphragmatic surfaces, but usually it extends more on the latter than the former, because of the superficial position of the diaphragmatic surface and its direct contact with the lower ribs anteriorly.

Owing to the enlargement, descent, and rotation of the spleen, its pedicle is not only lengthened and its direction changed from the transverse to one downwards and outwards, but it also loses contact with the tail of the pancreas, which does not accompany it in its descent.

Occasionally, however, when the spleen is not greatly enlarged the pedicle may still maintain its contact with the pancreatic tail and even contract firm adhesions with it difficult to separate.

The splenic vessels

The splenic artery is a branch of the cœliac artery. It passes to the left along the upper border of the pancreas in a remarkably tortuous manner behind the stomach and the omental bursa and reaches the hilus of the spleen between the folds of the lieno-renal ligament. Here it divides into 5 to 6 branches, which enter the spleen substance through the hilus.

The subdivisions of these branches, which are end arteries and are distributed along the transverse axis of the spleen, escape injury in any rupture of the spleen that passes across its anterior border and therefore parallel to them.

The splenic vein has a similar horizontal course from left to right across the abdomen. It is straight and not tortuous, and lies below the artery and behind the posterior surface of the pancreas, where it is subject to considerable pressure when the stomach is full and presses back the pancreas posteriorly.

It joins the superior mesenteric vein at right angles to the latter behind the neck of the pancreas and is therefore placed at a great disadvantage in draining into it specially during periods of active digestion when the superior mesenteric vein is engorged with blood.

At the hilus of the spleen, where it is formed by 5 or 6 tributaries, and in the pedicle, the splenic vein with the splenic artery lies on the tail of the pancreas, which is posterior to them in this situation.

The main tributaries of the splenic vein in the spleen pulp are disposed like the arteries on the transverse axis of the gland.

The short gastric and the left epiploic branches of the splenic artery are given off at the apex of the V-shaped junction of the gastro-splenic and lieno-renal ligaments; they pass between the folds of the former to be distributed to the stomach and do not in any way interfere with an operation for splenectomy.

Structure of the spleen

Besides its serous covering the spleen may be said to consist principally of a fibro-muscular capsule, a supporting framework of reticulated tissue inside the capsule and the spleen pulp made up of blood and various types of cells, which fill in the interstices of the reticulum.

The capsule is strong, and is composed mainly of elastic tissue and unstriped muscle fibres, which are circularly arranged round the longitudinal axis of the spleen and on account of their elasticity are normally capable of considerable expansion without rupture. The capsule is invaginated through the hilus over the blood vessels entering the hilus. Processes of this capsular tissue passing from the inner surface of the capsule and the surface of the blood vessels divide the spleen into small trabeculæ. These trabeculæ are further subdivided into a fine, microscopic reticulum by the reticular cells with processes radiating from their cytoplasm.

The branches of the splenic artery on entering the spleen divide and subdivide. The subdivisions lie in the direction of the transverse axis of the spleen and do not anastomose with each other, but gradually getting thinner and smaller in size end in cones of minute twigs, which lose their arterial walls and

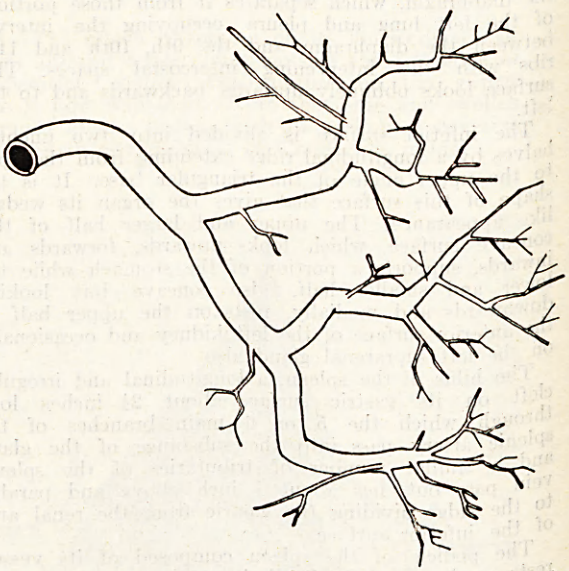

Diagram of a dissected specimen of splenic artery in the Anatomical Museum of the Vienna University showing the transverse and parallel directions of the main branches of the artery, which do not anastomose and which terminate as minute end-arteries.

retain only the endothelial coats. The endothelial coats soon get fenestrated and bulbous and are finally merged in the reticulum of the spleen pulp.

The splenic vein commences in the same way as the artery ends, that is, as pencils of minute tributaries, which have a fenestrated appearance as they are formed from endothelial cells, which seem to be derived from modified connective tissue corpuscles. These fenestrated endothelial tubes soon acquire a covering and are formed into small veins, which by coalescence form larger and larger ones and finally leave the spleen at the hilus as the 5 or 6 main tributaries, which anastomose to form the splenic vein immediately on their exit from the spleen.

Although the main tributaries of the vein inside the spleen lie along its transverse axis like the arterial branches, they, unlike the latter, anastomose with each other to a great extent.

The coats of the arteries are surrounded by lymphoid tissue, which at places is concentrated into definite nodules.

The nodules are known as the Malpighian corpuscles and are visible as small whitish specks to the naked eye.

In the meshes of the reticulum the arteries empty their contents.

Functions of the spleen

The normal functions of the spleen are stated to be (1) storage of iron, (2) beginning the formation of bilirubin from hæmoglobin, (3) filtration of bacteria and protozoal parasites, (4) manufacture of antibodies and lymphocytes, and (5) reduction or destruction of erythroblasts.

But it is with the structural peculiarities of the spleen, its rhythmical expansion and contraction, and the mode of circulation of blood in it that we are mainly concerned.

Circulation in the spleen

Blood entering the spleen through its arteries first passes into the splenic reticulum through the inter-endothelial open spaces in the bulbous ends of the terminal branches of the arteries and is finally poured into the boggy mass of the spleen pulp by the abrupt opening of these branches in its midst.

Thus there is a sudden considerable fall of blood pressure and slowing of the circulation in the spleen, a condition for which there is no parallel in any other organ or tissue of the human body.

The increased density and viscosity of the blood, due to its admixture with numerous cells of various types present in the spleen pulp with its reticular framework, cause a further retardation of circulation through the spleen.

This abnormal stagnation of blood would be impossible to overcome by the ordinary mechanism of circulation if it were not for the elastic and contractile character of the spleen capsule, its trabeculæ, and the splenic rhythm, which help to overcome the inertia and maintain a constant, but greatly retarded, venous flow through the spleen.

Circulation during digestion

During periods of active digestion the spleen is enlarged in size on account of an increase in the flow of arterial blood and a further decrease in the rate of venous blood flow. In the earlier stages of digestive activity a greatly increased volume of arterial blood is distributed in the digestive tracts and glands. In this distribution the spleen has its share. But while the arterial flow is increased the rate of venous flow in the spleen is not only not increased but, as the process of digestion proceeds, is further retarded by congestion in the portal system on account of the superior mesenteric vein bringing back to the portal vein a much larger volume of venous blood than usual.

The splenic vein joining as it does the superior mesenteric at right angles to the latter and lying behind the pancreas and the stomach is placed at a great disadvantage in emptying its contents owing not only to the congestion referred to above but also on account of (1) the direction of its venous blood current at right angles to that of the portal vein, and (2) the pressure exerted on it by the now active pancreas and the full stomach.

The splenic rhythm now fails to overcome the obstruction sufficiently to maintain the ordinary rate of venous outflow from the spleen and enable it to retain its normal size. We have therefore an increase in its size and a temporary venous congestion physiological in character with a rise of blood pressure inside it above its normal.

Its sluggish circulation, the greater density and viscosity of the blood in its pulp and the presence in this blood of the products of the cells destroyed or disintegrated in it render the loss of blood a very slow process in rupture of the spleen.

To the same factors may also be attributed a quicker coagulation than normal of the effused blood within the spleen capsule, round the spleen, and in the retroperitoneal space with which its capsule communicates.

Pathology of the rupture of an enlarged spleen

By an enlarged spleen is here meant a spleen which is suffering from a chronic enlargement due to a chronic inflammatory process, the result of such pathological conditions as malaria and kala-azar.

The enlarged spleen while retaining its shape may attain many times its normal size; there is a proportionate increase in weight. A spleen removed by me 5 to 6 hours subsequent to its rupture weighed seven pounds after removal.

Its relations to other viscera remain unchanged, though it may push the costal arch forward and outward on the left side, and present its base and anterior border several inches below the arch.

While the size of the splenic artery and its branches, which enter the hilus, are but moderately increased there is a compensatory dilatation of the vein proportionate to the increase in size of the spleen and it may attain as much as four times or more its normal size.

The splenic dullness, which normally can be outlined by percussion posteriorly, is now well marked over a large area anteriorly also.

These changes in size and weight of the spleen, distressing as they are to the patient on account of the pressure exerted by them chiefly on the stomach, lungs, and the heart, would add to the difficulties of its removal, were it not for the adhesions with the diaphragm and the neighbouring viscera as a result of perisplenitis. Thus it may be found adherent to the stomach, the colon, and to the diaphragm; the kidney, however, always escapes.

The extent of such adhesions varies considerably and so does the difficulty of removal of the spleen. The capsule and the trabeculæ are also hypertrophied in response to the enlargement.

It is difficult to imagine therefore that an enlarged spleen may rupture spontaneously. I never found a case of rupture where careful inquiry failed to elicit a definite history of injury.

The frequency of rupture of an enlarged spleen, I believe, is due chiefly to its accessibility as compared to the normal spleen. The enlarged spleen, lying next to the parietes and immediately behind the lower ribs with its anterior border and base frequently descending beyond the left anterior costal border at right angles to it, is exposed to trauma.

This view is supported by the fact that the seat of rupture is always at its anterior border, which is most accessible to trauma. The further fact that most often the injury to the spleen pulp is deep and extensive and out of all proportion to the tear in the capsule proves that the hypertrophied capsule even under such abnormal conditions is resistant to violence.

Next to accessibility, its semi-solid consistence held in by its capsule renders it more liable to rupture than the hollow viscera in its neighbourhood.

Thus it is rather the exception than the rule to find any serious injury to any other neighbouring viscus to be associated with the rupture of an enlarged spleen.

A study of the nature of the injury which caused rupture in 19 cases of enlarged spleen that came under my care showed direct violence in some form or other to have been responsible for them all.

The nature of the injury in the order of frequency was as follows :—

1. Knocked down by a motor car.
2. Knocked down by a bullock cart.

3. Kicked by a man.
4. Kicked by a horse.
5. Blow with the fist over the spleen.
6. Fall from a tree in the prone position.
7. Fall of a load on the abdomen.
8. Fall of a heavy load on the back which doubled up the patient driving the lower ribs into the spleen.

The history of each patient and the nature of the violence sustained by him, which in every case acted directly over the enlarged spleen lying partly in the left hypochondriac and partly in the left lumbar regions, showed that the rupture was caused by the anterior halves of the lower ribs or the left anterior costal border having been driven into the anterior border of the spleen.

The lower ribs (8th, 9th and 10th), owing to their articulation posteriorly with the bodies and transverse processes of the vertebræ to which they are connected by powerful ligaments and their attachment anteriorly by single facets and thin ligamentous bands to each other's cartilaginous ends, have only a slight, rotatory movement at the neck in their longitudinal axes, but enjoy considerable freedom of movement and elasticity anteriorly.

Thus when they are driven backwards by force against the anterior border of the spleen across which they lie one or more tears in the capsule are produced, some trabecular bands are torn and the spleen pulp is more or less crushed or contused.

The rupture of the capsule may extend on either side of the anterior border to the gastric and the diaphragmatic surfaces. Rarely is there more than one line of rupture and, although the depth to which it apparently encroaches into the spleen substance varies, the intracapsular laceration of the pulp all round the tear is usually considerable. This laceration or contusion of the pulp is caused by the impact on the spleen of the blunt agent of the violence, which drives in the rib or ribs before it.

I have seen only one example of intracapsular laceration of the pulp without an external tear in the capsule. There was only an indentation on the anterior border but considerable retroperitoneal hæmorrhage.

The abdomen was opened because of the symptoms of rupture. No intraperitoneal hæmorrhage was found but an examination of the spleen showed intracapsular clots over the gastric surface in the region of the hilus. While the spleen was being removed extensive extravasation of blood in the retroperitoneal space behind the descending colon was discovered and evacuated.

The stomach, the colon, and the ileum, which are in close anatomical relationship to the spleen, are very rarely injured. The explanation of this immunity lies in the facts that they are all hollow, elastic and compressible viscera, which lie protected under cover of the enlarged spleen, which displaces them from the surface, itself encroaching on the surface areas previously occupied by them.

Equally immune is the spleen pedicle. Except in very severe and rare crushing injuries of the thorax and the abdomen when other vital organs besides the spleen, such as the liver and the lungs, are also seriously injured leading to almost instantaneous death, the pedicle always escapes injury owing no doubt to its deep situation and the protection afforded it by the enlarged concave gastric surface and the overhanging anterior border.

Hæmorrhage in rupture of an enlarged spleen

Hæmorrhage after rupture of an enlarged spleen is very slow for the following reasons :—

1. The low pressure of blood in the spleen pulp.
2. Reflex and spasmodic contraction of the capsule after the injury narrowing the openings of the trabecular meshes.
3. Early intracapsular coagulation of the extravasated blood.
4. The escape from injury of the main branches of the artery and the vein, which enter the spleen at the hilus, and lie transversely across the spleen pulp and therefore parallel to the direction of the rupture and not across it.
5. The injury to the blood vessels is confined mainly to the capillaries.
6. Suspension of the splenic rhythm.

The course taken by intracapsular hæmorrhage and the mechanism of this extravasation has already been described.

Intraperitoneal hæmorrhage first collects round the spleen in the left hypochondriac and lumbar regions and thence trickles down into the pelvic cavity and may overflow into the right paracolic gutter if not early arrested by operation.

The extravasated blood soon coagulates in lumps of various sizes, which are freely intermixed with fluid blood.

Symptoms

The symptoms of a ruptured spleen which are first to appear are shock, pain, tenderness and well-marked hyperæsthesia over the left hypochondriac region.

The clinical signs of the catastrophe are slow and gradual in their onset. Thus the cardinal symptoms of hæmorrhage, such as shifting dullness in the abdomen, pallor, changes in the rate and tension of the pulse, fall of blood pressure, shallow and rapid breathing, develop to an appreciable degree such a long time after the onset of hæmorrhage that if the surgeon waits for them for the confirmation of his preliminary diagnosis the auspicious moment for effective surgical interference will have passed.

The hesitation in coming to a decision is chiefly due to a not-unnatural expectation to find the more serious and later symptoms in the early stage of the injury. Such expectation in the inexperienced is bound to be disappointed.

Had I not followed the golden rule in emergency abdominal surgery of opening the abdomen when in doubt, even in the very first case of rupture of the spleen that I came across, several lives would have been lost, others jeopardized, and experience gained at a high cost.

Shock, although constant, varies in degree but is very seldom acute and alarming, as is usually stated in textbooks. Patients have sometimes walked into the hospital with slight assistance from friends within a short time after the injury complaining only of pain and tenderness over the left hypochondrium.

Pain, tenderness and hyperæsthesia, however, are well marked and should be the deciding factors for immediate surgical interference. Associated with them there is most often to be found an enlarged and palpable spleen. Great care and gentleness should be exercised in palpation to discover it and the patient should not be urged to increased respiratory efforts, if there is any difficulty in appreciating it on account of the presence of the reflex muscular rigidity over the left hypochondrium.

This parietal motor reflex, the spasm of the fibromuscular capsule of the spleen and the diminished respiratory excursions are helpful in retarding or even in arresting the venous oozing.

A patient brought to hospital 3 to 4 hours after the injury, by a doctor who had made a quick diagnosis and rendered first aid by bandaging the lower part of the thorax and the whole of the abdomen with a *dhotie,* showed only slight hæmorrhage on opening the abdomen although the rupture was about 2½ inches in length and about ¼ inch deep.

Where the spleen is not greatly enlarged and descends but slightly below the costal margin it may not be palpable 2 or 3 hours after the injury on

account of its decrease in size following loss of blood and the spasmodic contraction of its capsule.

Pallor by itself is an inconclusive sign as most of the patients subject to pathological spleen suffer from a varying degree of anæmia; but when it is associated with the other cardinal symptoms of hæmorrhage it is of some significance in diagnosis.

The initial fall of blood pressure due to shock is not considerable and is recovered from soon after the trauma.

Definite and appreciable softness and quickness of the pulse, except in rare instances, can only be felt from 6 to 7 hours after the injury. Thus patients brought in early to hospital often have an almost normal pulse, temperature and blood pressure.

Air hunger and restlessness are the last signs to appear and indicate the final stages of the tragedy.

Shifting dullness is not early appreciable and, although it can be perceived at a later stage of the hæmorrhage, the movements necessary to elicit it may prove injurious to the patient. The time of its onset varies in individual cases in accordance with the amount of bleeding.

An aseptic general peritonitis manifested by well-marked but not excessive rigidity of the whole of the abdominal wall and obstinate constipation may develop 24 hours after the injury if the patient is still alive. Even at this stage pain is chiefly localized at the left hypochondrium and is only slightly diffuse.

This symptom was well seen in a boy of eight, who sustained a rupture of the spleen as the result of a fall from a tree in the prone position. The hæmorrhage was slow and the symptoms though definite were not alarming at any stage and the boy's mother, a widow, at first refused permission to an operation on her only child. General abdominal rigidity without much pain was definitely perceived after 24 hours. The rigidity and the obstinate constipation persisted until the abdomen was opened 72 hours after the injury and the effused blood, which was distributed all over the peritoneal cavity with a lot of clots in the pelvis, was quickly aspirated and sponged out and the spleen removed.

The child's bowels moved with a small soap-water enema 8 hours after the operation and thereafter naturally and normally. The recovery was uneventful.

There was a small shallow tear about half an inch long and about an eighth of an inch deep over the anterior border of the spleen. Any clot, which might have sealed the rupture, was not visible when the spleen was examined after clearing out the peritoneal cavity. It was about twice its normal size.

A consideration of the symptoms as described above leaves no doubt as to the need for immediate surgical interference.

Acute pain, tenderness and hyperæsthesia over the left hypochondrium immediately following an injury of that region in a patient suffering from enlarged spleen or in a patient with a history of malaria or kala-azar form sufficient justification for an exploratory laparotomy. We should not await the advent of the late symptoms, if we wish to save the patient's life.

In 18 out of 19 cases that came under my care the diagnosis was made on the early symptoms and splenectomy performed immediately. The patients all came to hospital within 4 to 6 hours after the injury, survived the operation, and had uneventful recoveries.

In the one case in which death followed the operation, the patient was a boy of 12 whom I saw 18 hours after the injury and after practically all the fatal symptoms had developed. Even his life possibly could have been saved if an immediate blood transfusion had been possible.

Treatment

In every abdominal catastrophe first aid and immediate and careful removal of the patient to hospital are of prime importance, and, if carried out by the attending physician, profoundly modify the prognosis of the case.

In suspected rupture of the spleen, the abdomen should be carefully bandaged over a pad with the knee and hip joints in a flexed position. The bandage should be efficiently and tightly put on. Commencing from the xiphoid process and lower part of the chest, it should extend down to the symphysis pubis. This not only limits respiratory excursions and diminishes pain due to movement to a minimum, but also helps to arrest the hæmorrhage by increased pressure on the spleen, which is already contracted as a result of the trauma. The effused blood is held in round the spleen in the left hypochondrium and coagulation is accelerated.

The patient should be placed on his back with the body in a slanting position, the head being on a lower level than the feet, *i.e.*, in a moderate degree of Trendelenburg position.

The question of administration of morphia is a difficult one to decide. If the attending doctor is a surgeon or experienced in surgical diagnosis and is sure of his finding, he will be justified in giving a hypodermic injection of a ¼ grain of morphia, especially if the patient has to be carried over a long distance to hospital and some time must necessarily elapse before he can reach it.

If, however, the patient can be quickly taken to hospital, it is best not to give any morphia as it masks the symptoms of pain, tenderness, cutaneous hyperæsthesia and the motor reflex.

When possible, slow subcutaneous infiltration of normal saline in the chest or the axilla should be given at once. This does not raise the blood pressure even if pushed to the extent of 2 pints, but on the contrary helps in the recovery from shock and counteracts the effects of loss of blood from hæmorrhage. This is seldom done, but on the rare occasions when it was, I found the patient a much better operative risk, because of the practically normal condition in which he was brought to hospital, in spite of the severe injury to his spleen.

If this has not been done, as is usually the case, no time should be lost in administering saline immediately the patient arrives in hospital.

Morphia should be administered as soon as the diagnosis is made. I have always given ½ grain of morphia hypodermically without atropin in all my cases without any ill-effects. The patient, unless he is an opium-eater, goes to sleep in a few minutes.

Anæsthesia

I have found one per cent novocaine infiltration anæsthesia in successive layers of tissues as the operation proceeds, with the preliminary dose of morphia administered hypodermically 15 to 30 minutes before the operation, quite sufficient.

In nervous patients, who insisted on a general anæsthetic, a few whiffs of ether in addition were all that I found necessary.

Novocaine infiltration should extend for 3 inches laterally on each side of the line of incision along its whole length. I have often injected 250 to 300 c.cm. of the fluid with highly satisfactory relaxation and no subsequent harmful reaction of any kind.

However, spinal anæsthesia administered at a suitable level by an experienced anæsthetist I consider to be the best.

Position of the patient on the operation table

The best access is obtained by placing the patient in a reversed Trendelenburg position at an angle of 45 degrees with the table and with a similar inclination to the right.

This position drags the heavy, enlarged spleen downwards and towards the middle line and removes the intestines away from the site of the operation. Sand-bags should be so placed under the left loin as to arch the hypochondrium forwards.

The operation

The only incision used by me is a straight left paramedian incision about half an inch from the middle line, extending from as high a point on the left side of the subcostal angle as possible to 3 inches below the umbilicus.

With the position of the patient as described and complete relaxation of the parietal muscles on either side of the incision due to novocaine infiltration, very good access is obtained with suitable retractors.

As one dissects down to the peritoneal cavity nothing abnormal is generally noticeable, except occasionally the rupture of a few muscle fibres, until the peritoneum is reached when its tell-tale purple discoloration is at once seen.

On opening the abdomen there is a rush of blood whose venous character is at once apparent. Usually the pelvic cavity is found full of clots mixed with a little fluid blood. In the left paracolic gutter the situation is somewhat similar, but the spleen is generally drowned in fluid blood and only a few clots are seen near it.

The retroperitoneal spaces communicating with the hilus as well as the hilus itself are most often packed with coagulated venous blood.

Unless there is a considerable effusion of blood within a short time due to an extensive rupture deep in character or unless the patient has been made to move from side to side to elicit shifting dullness, as is unfortunately often done, the right side of the abdomen is found free from blood if it is opened within 3 to 4 hours after an injury.

In none of these cases did I find any injury to any other viscera associated with the rupture of the spleen.

All blood and clots are quickly removed by aspiration and with the help of large, warm sponges by an assistant.

In the meantime the surgeon should feel for the hilus, which is easily found, and press on the pedicle with the left hand, while with the right hand he searches for the rupture on the anterior border, as it generally extends both ways, *i.e.*, towards the gastric and diaphragmatic surfaces from this border. The tear can easily be distinguished from the notches by its rough surface and its extent, although it lies parallel to them.

As soon as visibility is secured by the removal of blood and clots the pedicle should be handed over to the assistant to control while the surgeon continues his examination of the spleen and its surroundings.

Adhesions, of varying degrees, to the stomach, diaphragm and colon are always found, and prevent not only the rotation and evisceration of the spleen, but also render the use of clamps on the pedicle difficult or even impossible.

While the assistant stands to the left of the surgeon, both being on the right side of the patient, exercising what pressure he can on the pedicle, the surgeon divides the adhesions between clamps. When they are all cut through and the spleen is at last free, the assistant goes to the left of the patient and firmly grasps the spleen with a towel. The spleen is now rotated backwards by the assistant on its long axis to curve its pedicle forwards and two large curved clamps are easily applied on the generally abnormally long pedicle with the right hand of the operator, while with the left hand he firmly grasps it at its farthest right-hand extremity.

One clamp is applied flush with the hilus and the other an inch or so away from it to the right. As I have already said an enlarged spleen with its more or less elongated pedicle easily permits this.

I have never brought the spleen out of the abdomen. It is often too large and heavy to be safely manipulated out without the risk of serious damage to the pedicle.

Generally the pedicle is so broad and thick, owing to the enlargement of the veins and the stretching of the peritoneum over it by adhesions and due also to a collection of clots inside the peritoneal sheath of the pedicle, that it is expedient to use two pairs of forceps instead of one at each of the two points indicated, one being applied from above and the other from below.

A preliminary incision of the sheath over the hilus and the pedicle anteriorly, to expel the clots and separate the pancreatic tail from the pedicle when necessary and possible, can be easily effected and is helpful in the efficient application of the clamps.

The tail of the pancreas lies behind the lower border of the pedicle and is easy of access. If it is firmly adherent to the pedicle, its sheath and the spleen, the right-side clamp or pair of clamps should include in its grasp both the pedicle and the tail of the pancreas.

The spleen is removed by cutting through the pedicle to the left of the pair of forceps nearest to the hilus, but the incision has sometimes to be made through the spleen substance.

The pedicle is tied with two stout chromic gut ligatures, transfixed and applied one between the clamps and the other in the immediate proximity to and to the right of the clamp placed nearest to the middle line of the body, and the clamps are taken away. The cut end of the pedicle is covered by peritoneum, as well as the circumstances allow.

The divided perisplenic adhesions on the stomach, the diaphragm and the colon are now tied with catgut, or, if long, as they often are, their cut edges are brought together quickly by continuous sutures, and the clamps removed.

When of necessity the tail of the pancreas has to be clamped and later on tied, no ill-effects are produced. I think the only danger of tearing it or cutting into it, is in trying to separate the intimate adhesions by blunt dissections or by excising them with a knife or a pair of scissors. The adhesions of the tail are generally very close and firm.

I never wasted time in attempting to tie the blood vessels separately.

It was my usual practice to leave a pint of sterile normal saline solution in the pelvic cavity before closing the abdomen, which was done in the usual manner in layers without drainage.

Some particulars of the cases operated upon

There were 19 cases in the series with one death. The ages of the patients were from 8 to 42; they were all males.

The average time taken to perform the operation was 25 minutes.

Every one of the patients gave a history of suffering for a long time either from malaria or kala-azar.

In 11 out of the 19 cases there were retroperitoneal hæmorrhages.

In 16 cases the operation was performed 4 to 8 hours, in two cases more than 18 hours (of these one died), and in one case about 72 hours after the injury. In this last instance the patient survived the operation and made a good recovery.

Blood transfusion was carried out in only two cases, both of whom survived the operation. I consider blood transfusion unnecessary if the patient is operated on early.

The convalescence in those cases that survived the operation was rapid and uneventful. The removal of the diseased spleen seemed to bring about a rapid recovery.

The cases were not followed up, but two of the patients, who came to see me over a year after the operation were in perfect health and stated that they were enjoying much better health after the operation than before it.

Unfortunately systematic examinations of the blood prior and subsequent to the operations were not carried out and hence no statement as to blood changes can be made.

The following is a summary of the special features in the anatomy, pathology, symptoms and treatment of the rupture of an enlarged spleen.

1. An enlarged spleen is most exposed to trauma on account of its superficial position lying as it does next to the parietes in the left hypochondriac and lumbar regions. In fact it forms the most prominent abdominal landmark of a person suffering from chronic malaria or kala-azar.

2. It fills in these spaces from the anterior to the posterior wall and in its descent rotates round its antero-posterior axis to get past the constriction of the waist at and below the 12th rib, and lies in its new position with its anterior border across the left anterior costal border.

3. The constancy of the situation of the rupture across the anterior border is due to its being the anterior-most part of the spleen lying across the lower ribs, which are driven into it by direct violence.

4. The contusion or laceration of the pulp is generally extensive and out of all proportion to the superficial extent of the rupture and is caused by the impact of the blunt instrument of the force, which follows the ribs.

5. The extreme rarity of any injury to any other viscera being associated with the rupture of an enlarged spleen.

6. The usual slow progress of the hæmorrhage.

7. The frequency of retroperitoneal hæmorrhage and the channel through which it takes place.

8. The slight degree of initial shock.

9. The development of the serious symptoms 6 to 12 hours after the injury is sustained.

10. The importance and benefit of first aid, which greatly improves the prognosis.

11. The importance of diagnosis on the initial symptoms.

12. The need of immediate operation, and its success in saving the patient's life, if performed early.

13. The need of quickness in the operative procedure.

Medical News

FIFTEENTH INTERNATIONAL PHYSIOLOGICAL CONGRESS, LENINGRAD-MOSCOW, 9TH TO 17TH AUGUST, 1935

ACCORDING to the decision of the Fourteenth International Physiological Congress in Rome, the Fifteenth International Congress will be held in the U. S. S. R. The Congress will take place in Leningrad and Moscow, 9th to 17th August, 1935.

The Committee will provide board, lodging, etc., for all the members of the Congress and dependent members of their families. Arrangements will be made for trips and visits to several parts of the country at reduced rates. In order to avoid any delay with visas, and to procure the best conditions of life and work for the members of the Congress, all the physiologists intending to take part in the Congress are requested to enlist as soon as possible.

The membership fee, Rbls. 10 = 131 Francs 34 Centimes Gold, should be forwarded to the following address: The State Bank of the U. S. S. R., Moscow, Current Account No. 7005 of the Committee of the Fifteenth International Physiological Congress. The cheque can be drawn in any currency according to the exchange rate of the day. Name and address of the sender should be given to the bank.

Detailed information as to the programme of the Congress, life conditions, trips, etc., will be issued in the near future.

Please address mail concerning the Congress: Leningrad, Main P. O., Box 13.

THE COMMITTEE OF
THE CONGRESS.

THE FACULTY OF TROPICAL MEDICINE AND HYGIENE, BENGAL

THE following students are declared to have passed the D.T.M. Examination, session 1934-35.

Passed

1. Amar Jit Singh, M.B., B.S. (Punjab), Assistant Bacteriologist, Clinical Laboratory, Patiala State—Awarded the 'Chuni Lal Bose' Gold Medal, 1935.
2. Jyotirmoy Banerjee, M.B. (Cal.), D.P.H. (Cal.), Private Practitioner.
3. Hans Raj Bhambi (Rai Sahib), L.M.P. (U. P.), L.T.M. (Bengal), Sub-Assistant Surgeon, Government of Punjab.
4. Provash Chandra Bhattacharyya, M.B. (Cal.), D.P.H. (Cal.), Private Practitioner.
5. Pundarikaksha Prasad Bhattacharyya, M.SC. (Cal.), M.B. (Cal.), D.P.H. (Cal.), Private Practitioner.
6. Swadesh Kumar Bose, M.B. (Cal.), Private Practitioner.
7. Devendra Prasad Das, B.SC. (Cal.), M.B. (Cal.), Civil Assistant Surgeon, Government of Bihar and Orissa.
8. Ghanasyam Das, M.B. (Cal.), Medical Officer, Palasbari Charitable Dispensary, Assam.
9. Nagendra Nath De, M.B. (Cal.), Bacteriologist, Calcutta Corporation.
10. Rohini Kumar De, L.M.F. (Bengal), Assistant Medical Officer, Talap Tea Estate, Assam.
11. Laxmikantrao Ramrao Deshpande, L.M. & S. (Osmania Medical College), Hyderabad, Deccan, L.C.P. & S. (Bombay), Assistant Pathologist, Osmania Hospital, Hyderabad, Deccan.
12. Henry Victor Francis, Diploma of Medical College, Calcutta, I.M.D., Assistant Surgeon, Government of India.
13. Joseph Francis Freeman, L.C.P. & S. (Bombay), I.M.D., Assistant Surgeon, Government of India.
14. Chuni Lal Ganguli, M.B. (Cal.), D.P.H. (Cal.), Private Practitioner.
15. Govindan Sambasivan, M.B., B.S. (Madras), Private Practitioner.
16. Tarak Jiban Gupta, M.B. (Cal.), Private Practitioner.
17. Kalyan Singh Makhni, L.S.M.F. (Punjab), L.T.M. (Bengal), Sub-Assistant Surgeon, Government of Punjab.
18. Gopal Narain Khanna, M.B., B.S. (Bombay), Private Practitioner.
19. Gangigunte Krishnaiah, L.M.P. (Mysore), Sub-Assistant Surgeon, Mysore Medical Service.
20. Ujagar Singh Madnawat, L.S.M.F. (U. P.), I.M.D., Sub-Assistant Surgeon, Government of India.
21. Md. Mustafa, M.B. (Cal.), Private Practitioner.
22. Mohammad Belayet Hossain, L.M.F. (Bengal), Medical Officer, District Board, Pabna.
23. Mohammad Jalal-ud-Din, M.P.L. (Punjab), L.T.M. (Bengal), Sub-Assistant Surgeon, Government of Punjab.
24. Nawab Ali, M.B. (Cal.), Assistant Surgeon, Government of Bengal.
25. Krishnan Lal Pathak, M.B., B.S. (Punjab), Private Practitioner.