BRITISH MEDICAL JOURNAL

LONDON SATURDAY JULY 14 1951

THE SURGERY OF THE SPLEEN*

BY

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[WITH COLOUR PLATE]

The mystery with which Galen shrouded the spleen more than seventeen hundred years ago is not yet fully penetrated, and the surgeon who elects to speak about the organ cannot be over-bold or over-confident. In these circumstances I have chosen for the most part to share with you my own disappointments and successes, and I cannot claim for this lecture that it will be a comprehensive survey of all the diseases in which surgery of the spleen may play some part in treatment.

Nearly two years ago there appeared in the *Lancet* (1949) an editorial headed "Splenectomy and the Haematologist," which made a point with which I am so much in agreement that I quote it at the outset. "How," asked the editorial writer, "do surgeons find their way through this jungle of difficult diagnosis? . . . The answer is that, if they are wise, they do not try. Mortality will depend on how well cases are selected by the haematologist, who must take the responsibility for determining whether the lesion is primary, or secondary to some condition for which operation would not be helpful." For nearly twenty years I have had the privilege and the advantage of co-operating with Professor L. S. P. Davidson and his younger colleagues in the management of many of these cases, and to him and to them I am grateful for guidance and for a grounding in at least elementary haematology.

Possible Arguments against Splenectomy

The spleen is relatively heaviest during the first year of life. Thereafter it is largest and heaviest in the young adult (average 170 g.), but its size decreases with age until its average weight is 128 g. in patients over 60 years of age (McNee, 1931a). Extreme atrophy of the spleen is rare; it is said to occur in sickle-cell anaemia, and I have encountered it in a patient aged 53 during oesophago-gastrectomy for carcinoma, the cause not being determined. The very persistence of the spleen presumes some useful function throughout the span of human life.

When one is considering the applicability of any operation it is wise to decide whether there may be any possible undesirable effects. It seems clear from the examination of patients whose spleens have been removed because of injury (Ek and Rayner, 1950) that there are no permanent alterations in the numbers of red cells, white cells, and blood platelets, and that bleeding and clotting times remain within normal limits. It is still believed by some that splenectomy impairs the process of adjustment to haemorrhage by removing a potential reservoir for blood. Though this may be true in certain animals it is not true in man, whose normal spleen is so small that it could add only a trifling quantity of blood to a depleted circulation. Moreover, McMichael and Sharpey-Schafer (1944) have pointed out that when a person is bled deliberately the fall in pressure in the right auricle varies directly with the amount of blood lost, which would not happen if the volume of circulating blood was being augmented from a blood depot in the spleen.

There is no evidence for the belief that a patient lacking the spleen is more vulnerable to infections, either protozoal or pyogenic. It is true that lymphocytes and perhaps antibodies of lymphocytic origin are produced in the spleen, but the splenic lymph follicles are using the spleen merely as a manufacturing site. They are almost as apart from it as are the lymph nodes in the submandibular salivary glands, and there is plenty of spare lymphatic tissue in the body. During the last war it was shown at a physical development centre that the lack of the spleen did not prevent healthy adult males from reaching the highest degree of endurance to physical exertion (*J. roy. Army med. Cps*, 1945; editorial).

In two conditions—fibrous metaplasia of the bone marrow and osteosclerosis—the spleen enlarges because it has reverted to its function in the foetus of producing the cells of the blood. In these two conditions splenectomy is contraindicated because it would be harmful.

From these reflections one cannot make more accurate deductions than are suggested by the remark of W. J. Mayo (1926), that, "while physiologically the spleen is not very important, pathologically it is extremely important."

Technique of Splenectomy

Many techniques have been described (Pemberton and Kiernan, 1945; Lahey and Norcross, 1948; Cole *et al.*, 1949; Chamberlain, 1950; Welch and Dameshek, 1950), and as many more are to be seen during visits to colleagues.

When the spleen is normal in size or only moderately enlarged it may be removed through a left transrectus vertical incision or through a transverse epigastric incision. I have come to prefer the latter, which gives good access to the biliary apparatus, always required in operations for primary haemolytic anaemia, and allows one

*Lecture delivered to the Harveian Society of London in the Royal College of Surgeons of England on March 15, 1951.

to search for accessory spleens. When the spleen is large and adherent it is not easily removed through abdominal incisions unless it has "polished off" the roughness of perisplenitis (McNee, 1931b). There is no doubt whatever that a large spleen which has formed venous attachments to surrounding structures and which has large vessels in its pedicle is best exposed through a combined thoraco-abdominal incision (Carter, 1947). This passes between the eighth and ninth ribs and at the costal margin is continued as a transverse epigastric incision which ends short of the midline in order to preserve any collateral veins in the round ligament. The diaphragm is split in the line of its fibres almost to the oesophageal opening, care being taken to control any large anastomotic veins under its peritoneum.

It is best to begin to isolate a large spleen at its lower pole by dividing the spleno-colic ligament. This gives access to a plane between the spleen and the posterior abdominal wall which is extraordinarily bloodless. A pack is gradually introduced upwards and outwards in this plane to the reflection of the parietal peritoneum on to the convex aspect of the spleen. The greater part of the gastro-splenic omentum is then divided and the splenic artery is ligated. The dissection returns to the postero-lateral aspect of the spleen, which is steadily diminishing in size, and the parietal peritoneum is detached from it by a diathermy needle to expose the pack which has been left in position. The vasa brevia can now be ligated with accuracy and the spleen raised to return as much of its blood as possible to the patient, a process which may be hastened by causing the organ to contract by applying a faradic current to the arterial part of its pedicle. The splenic vein is ligated, or clamped if it is to be used for splenic–renal anastomosis. The operation is completed by suturing together the cut edges of the gastro-splenic omentum and of the parietal peritoneum. It will be appreciated that through this thoraco-abdominal incision the left kidney can be readily exposed for anastomosis of the splenic vein to the renal vein. If the operation is lengthy the left lung must be reinflated from time to time. My own operations have been greatly facilitated by the superb anaesthesia provided by Dr. John Gillies and his junior colleagues.

For a week or ten days after the removal of a large spleen the patient's temperature chart often shows intermittent fever. This is probably due to absorption from the raw area left after the splenectomy, and is not an indication for recourse to antibiotics.

Accessory Spleens

Accessory spleens originate as additional rudiments formed in the dorsal mesogastrium. They tend to disappear with increasing age, for the incidence at necropsy is higher in children (25%, Jolly, 1895) than in adults (11%, Adami and Nichols, 1910); they are more common in conditions in which the spleen is at fault. Failure to remove all accessory spleens at the original operation may be the cause of recurrence of thrombocytopenic purpura or of congenital haemolytic anaemia; examples of this have been collected by Curtis and Movitz (1946).

By far the most common situation for accessory spleens (more than 50%) is at the hilum of the spleen proper, and most of the remainder occur in the neighbourhood of the splenic vessels and behind the body and tail of the pancreas. Less commonly they are found in the great omentum, in the various splenic ligaments, in the mesenteries of the small intestine and colon, where they correspond to the scattered splenic masses present in some fishes, and rarely near the left ovary or the left testicle, situations accounted for by the proximity of the dorsal mesogastrium to the genital ridge. All these sites should be thoroughly explored in the course of splenectomies for idiopathic thrombocytopenic purpura and congenital haemolytic anaemia. An accessory spleen overlooked at a first operation, which reproduces the original disease, is always found at a subsequent operation to be of considerable size and may reach that of a normal spleen. In children an accessory spleen may become twisted on its pedicle and give rise to an acute abdominal emergency (Settle, 1940).

Splenosis is a different condition, in which numerous (up to 300 have been counted) small masses of splenic tissue are found widely scattered over the peritoneum in situations other than those to be expected on embryological grounds. It results from the implantation of fragments of spleen freed by injury, either rupture by violence or incomplete operative removal, and possibly distributed throughout the peritoneal cavity by bleeding from the torn vessels of the organ. This autografting of splenic tissue occurs most readily in young patients (Hamrick and Bush, 1942).

Rupture of the Spleen

One of the odd gaps in my clinical material has been experience of rupture of the spleen. During the eighteen years I have had charge of general surgical wards I have seen only one example; the patient recovered after splenectomy. The incidence of this lesion in all accidents occurring in urban communities has been variously estimated at from 0.1 to 0.2%, and at from 30 to 47% in all closed abdominal injuries (Roettig *et al.*, 1943); this does not take into account rupture resulting from blast injuries (Gordon-Taylor, 1942). In slightly more than half the cases the damage is so extensive and the resultant bleeding is so profuse that the patient dies within an hour.

The remaining lesions fall into two groups: (1) considerable tears, the clinical picture of which includes as its dominating features abdominal rigidity and pain, either generalized or in the left upper quadrant, associated with the features of shock; and (2) small tears and subcapsular haematomas, inevitably followed by delayed rupture (McIndoe, 1932). When the tear is small the haemorrhage may at first be limited by adhesion of the neighbouring viscera; when the bleeding is subcapsular the haematoma slowly increases in size. In both sets of circumstances, after a latent interval which may vary from 48 hours to six months, during which the pulse rate often fails to return to normal and an ache may persist in the left epigastric region, the haematoma bursts its confines as the result of some minor strain. The resulting clinical picture of intraperitoneal haemorrhage may be puzzling, because the original injury may not be considered as an explanation of the later emergency.

A haemangioma of the spleen may rupture into the general peritoneal cavity (Pines and Rabinovitch, 1942). When it is enlarged, and especially when it is soft, the spleen is easily ruptured by relatively slight violence; in infectious mononucleosis even abdominal palpation has caused the accident (Johnson, 1949). The association of rupture of the spleen with pregnancy is probably fortuitous; the complication has been described in first pregnancies and in a sixteenth, and at various stages of

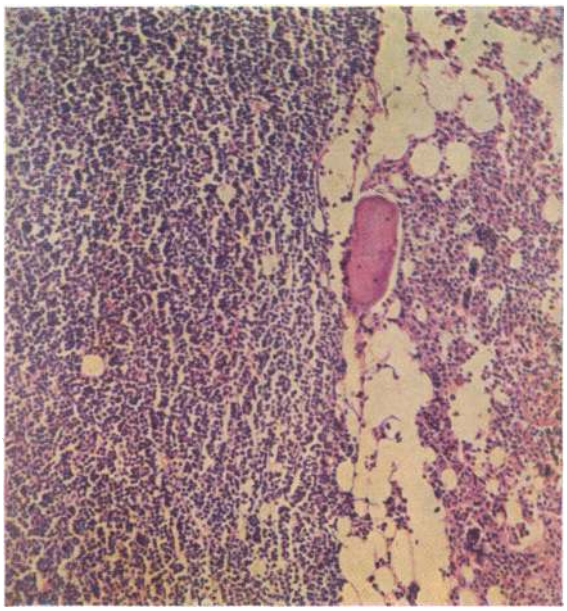

FIG. 1.—Splenectomy for secondary purpura. Case 1. The bone marrow has been almost completely replaced by cells of the lymphocyte series. The small areas of residual marrow show a brisk leuco-erythroblastic response, and megakaryocytes are also numerous. (H. and E. ×95.)

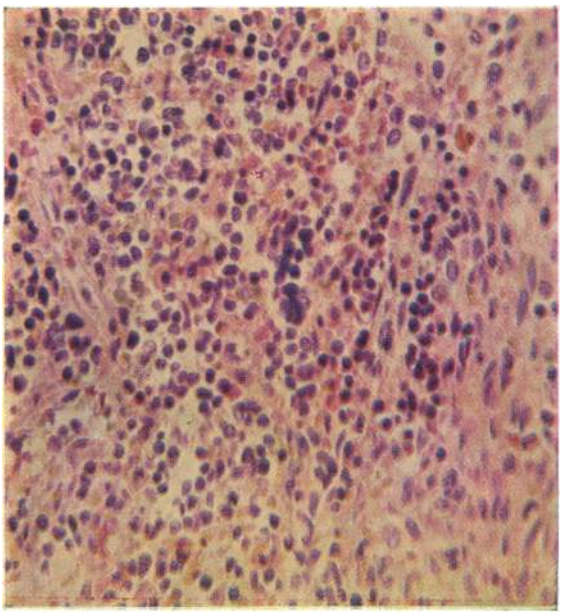

FIG. 2.—Splenectomy for secondary purpura. Case 1. Widespread haemopoiesis in an ovary. (H. and E. ×340.)

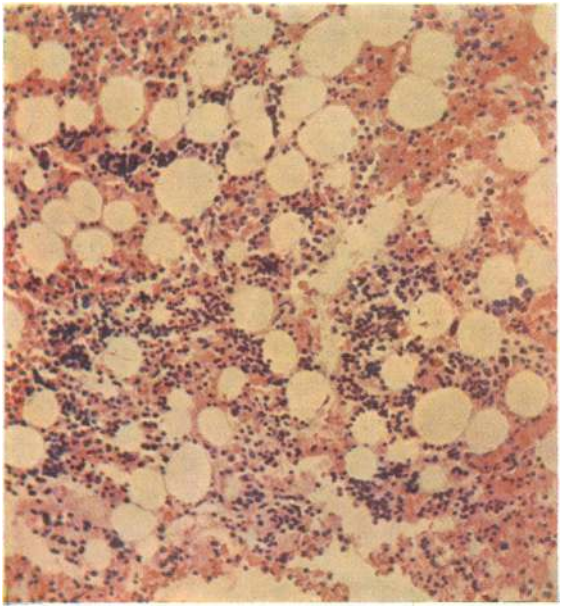

FIG. 3.—Splenectomy for secondary purpura. Case 2. Bone marrow showing irregular haemorrhage, a moderate reaction along both normoblastic and leucoblastic lines, and scanty diffuse infiltration with mature lymphocytes. (H. and E. ×130.)

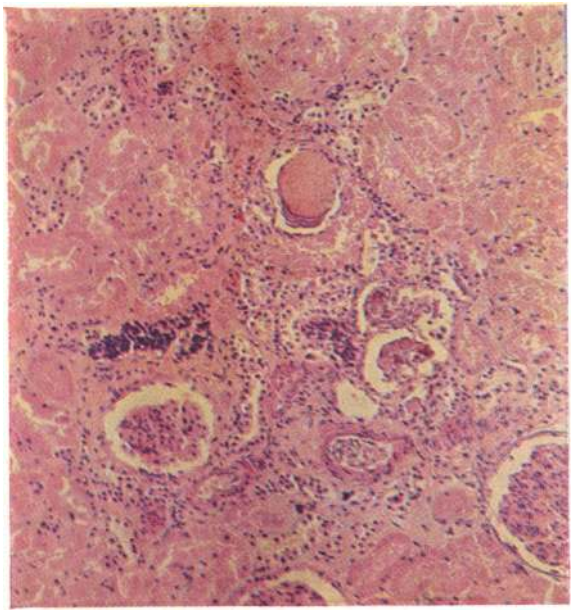

FIG. 4.—Splenectomy for secondary purpura. Case 2. Kidney showing mature and immature lymphoid cells in the vessels. (H. and E. ×100.)

gestation between the third and ninth months (McElin and Mussey, 1950).

When rupture of the spleen is diagnosed or discovered at laparotomy splenectomy is the only operative procedure to be considered. Its success depends on its early performance and on the rapidity with which transfusion of blood can be begun and continued until the blood volume has been restored. Every fragment must be removed in order to avoid the autogenous on-grafting of splenic tissue which ends as splenosis (see above).

Idiopathic Thrombocytopenic Purpura

It is essential to exclude from operation patients exhibiting a haemorrhagic tendency secondary to other conditions such as the effects of ingestion of a drug, severe infections, leukaemia, and aplasia of the bone marrow. The most important points are an accurate history, a complete physical and haematological examination, and examination of a specimen of bone marrow. As Pemberton and Kiernan (1945) insist, splenectomy for " purpura " is not likely to be successful if brisk regeneration cannot be detected in the marrow and if immature white cells are abnormally numerous ; and Schwartz and Kaplan (1950) state that the prognosis in *idiopathic* thrombocytopenic purpura is better, and the operative mortality of splenectomy lower, when the marrow contains increased numbers of eosinophils.

As a preliminary to deciding if splenectomy is indicated it is possible to classify patients suffering from idiopathic thrombocytopenic purpura into certain broad groups. Thus the disease may first manifest itself in an acute form, which causes anxiety at any age because of continuing external haemorrhage which must be corrected by transfusion, and in the older age groups because of the possibility of intracerebral bleeding. It is a useful clinical guide that children usually recover from a first acute attack without operation, although about one child in four will continue to suffer from the disease in a chronic and less dangerous form which may require splenectomy " in the cold " at a later date. I know of no more anxious condition than acute thrombocytopenic purpura in an adult, and I cannot rid myself of the memory of a young woman, one of my early cases, whom I watched too long ; she did not recover consciousness after splenectomy, and the necropsy disclosed extensive cerebellar haemorrhage with medullary compression. With this tragedy in mind, years later, I carried out an emergency splenectomy on a young woman suffering from idiopathic thrombocytopenic purpura whose clinical examination had disclosed an extensor response in one great toe, only to find on reviewing the case after two years that she suffered also from disseminated sclerosis.

When idiopathic thrombocytopenic purpura is chronic from the first or when an acute attack merges into a chronic condition there is no possibility of a spontaneous cure, although there may be periods of remission. During these periods, which may be of considerable length, the bleeding time may be normal, although the clot still does not retract completely, and the capillary resistance may be normal or nearly normal. Indeed, when the remission is ending, the bleeding time increases before the capillary fragility increases. In such chronic cases I believe that splenectomy should be advised, and I do not consider it necessary to wait for a remission before performing the operation. In my series of 19 cases there has been one post-operative death, to which I have referred.

Robson (1949a) has shown that the clinical result of the operation is as a rule satisfactory (16 out of 19 cases, in the majority of which splenectomy was performed in my unit) (Table I). Of the remaining three cases in his series, in one (Case 20) mild manifestations persisted, in the second (Case 14) there was a relapse after seven years of freedom from symptoms (possibly as a consequence of the growth and activity of an accessory spleen), while the third (Case 28) did not derive any benefit from splenectomy. It is of interest that in my experience (Cases 21 and 22) the platelet count may ultimately fall to a low level after splenectomy without any recurrence of bleeding ; this has not been Wintrobe's (1946) experience. As Robson remarks, such a finding suggests that in this disease thrombocytopenia is not the only factor in the production of the tendency to bleed ; his present view is that a product elaborated in the spleen interposes a barrier in the form of some abnormality of capillary endothelium which prevents the delivery into the blood stream of platelets derived from normal megakaryocytes (Robson, 1949b).

The combined experience seems to be that about 70% of patients suffering from idiopathic thrombocytopenic purpura are cured clinically and haematologically by splenectomy. In an additional 10 to 15% the tendency to bleed is less marked or the episodes of bleeding are less frequent ; in this group such haematological abnormalities as a low platelet count may reappear after a period of three to four weeks' absence. Recurrence of the disease after intervals measured in years is probably due to enlargement of an accessory spleen overlooked at the original operation.

TABLE I.—*Cases of Idiopathic Thrombocytopenic Purpura Treated by Splenectomy. (Reproduced by kind permission of Dr. H. N. Robson)*

Case No.:	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
Sex:	F	F	F	F	M	F	F	F	F	F	M	F	F	F	F	F	F	M	F
Age at onset ..	21	25	23	50	7	5	14	30	5	47	30	24	8	54	19	23	14	6	32
Episodes in history ..	3	4	10	1	10	8	4	2	20	1	3	20	5	1	20	20	6	3	1
Platelets (thous.) ..	20	20	35	40	70	20	30	20	10	15	70	50	40	5	25	35	20	15	20
Bleeding-time (mins.)	18	10	22	18	16	7	20	20	10	20	10	4	8	45	45	8	10	12	14
Capillary fragility ..	+	+	+	+	+	+	+	+	+	+	+	N	+	+	+	+	+	+	+
	Splenectomy																		
Years since operation	17	10	8	6	4	3	3	3	2	1	2	2	2	1	1	2	1	1	½
Clinical condition ..	R	G	G	G	G	G	O	G	G	G	G	G	G	G	N.I.	G	G	G	G
Platelets (thous.) ..	40	340	301	125	140	240	45	30	22	410	190	290	305	60	20	55	200	280	376
Bleeding-time (mins.)	8	3	6	3	8	3	9	2	3	2	4	3	5	10	18	3	2	2	2
Capillary fragility ..	+	N	+	+	N	N	+	+	+	N	+	N	N	+	+	+	N	N	N

R = Relapsed after 7 years. Many bleeding episodes since. O = Occasional, slight epistaxis. N.I. = No improvement after operation—bleeding continues. G = Clinical condition good—no bleeding episodes since operation.

Thrombocytopenic Purpura in Pregnancy

A review of this subject by McElin and Mussey (1950) shows that both the maternal death rate (from post-partum haemorrhage) and the foetal and neonatal death rates (from bleeding) are high. Robson and Walker (1951) have collected the recorded cases of congenital and neonatal thrombocytopenic purpura. Although many of these cases are badly documented, they fall into two groups: (1) *infants whose mothers had purpuric manifestations*: whether the maternal purpura was idiopathic or secondary (and due to rheumatic fever, severe pyogenic infections, syphilis, or the action of drugs such as quinine), the tendency might be transmitted to the offspring (which was most likely to die if the mother had had active purpura during her pregnancy) or the infant might be normal; and (2) *thrombocytopenic infants born of mothers said to be normal*: more prolonged observation has shown that some of the mothers in this group were in a period of remission from the disease, but such an odd circumstance has been recorded as the appearance of purpura in one infant of twins.

I have operated on a mother suffering from idiopathic thrombocytopenic purpura who two weeks previously had given birth to a child who for the first three weeks of its life exhibited purpura and thrombocytopenia (Robson and Walker's Case 1). This patient, and one other who had undergone splenectomy for thrombocytopenic purpura in my unit, subsequently have had uncomplicated pregnancies and have been delivered of normal infants. When a pregnant woman is found to be suffering from idiopathic thrombocytopenic purpura it seems to be the consensus of opinion that it is best for the mother and unborn child if splenectomy is performed as soon as the diagnosis is certain.

Occasionally a case of thrombocytopenic purpura is encountered in which the bleeding-time is normal. An extreme example of this condition has been described by Suchecki and Glass (1945) in which splenectomy was completely curative, and these authors recall that in such cases (as described by White, 1944) treatment may be directed to the bleeding organ alone with disastrous results, as indeed has been recorded in typical cases of idiopathic thrombocytopenic purpura (Dameshek and Rheingold, 1949). It has been pointed out by Wintrobe *et al.* (1937) that in the hereditary type of purpura the platelet count may be normal although the bleeding-time is prolonged; in cases of this type splenectomy is contra-indicated.

Splenectomy Mistakenly Undertaken in Symptomatic Purpura

There were three cases in this group, and as it is important to avoid such mistakes I shall describe them in detail.

The first patient, a woman aged 26, was admitted with a history of spontaneous bruising of four weeks' duration. The spleen was enlarged to 6 cm. below the costal margin. Haematological examination showed: haemoglobin, 60%; red cells, 2,600,000; white cells, 9,800; platelets, 50,000; bleeding-time, nine and a half minutes; films showed an occasional primitive white cell. The patient died six hours after splenectomy. Microscopical examination of the spleen showed chronic non-specific lymphadenitis and megakaryocytic hyperplasia. At necropsy the only macroscopic finding proved to be replacement of the marrow of a femur by grey "tumour" tissue.

The microscopical report was kindly provided by Dr. Robertson Ogilvie. "Microscopical examination reveals that this is an example of the rather rare medullary type of aleukaemic lymphatic leukaemia. It shows several interesting features: (1) tumour-like replacement of marrow without leukaemic infiltration of the other organs examined (Plate, Fig. 1); (2) widespread extramedullary haemopoiesis—for example, in liver and ovary (Fig. 2); and (3) an apparent concentration of megakaryocytic formation in lymph nodes and an apparent lack of evidence of excessive lymphocytic production by lymph nodes. Such widespread extramedullary haemopoiesis suggests that splenectomy may have removed one of the important centres of blood formation."

This was a mistake for which I was responsible. Unfortunately a specimen of sternal marrow was not obtained before operation, and the significance of the primitive white cells found in the blood films was not appreciated. The mere fact that the spleen was palpable should have imposed caution; the heaviest spleen known to me in the literature of idiopathic thrombocytopenic purpura weighed 240 g., only about 30% heavier than the *average* weight of the organ in an adult.

The second patient, one of my earliest cases, was a woman aged 52, who gave a history of purpuric lesions of two months' duration. For three weeks she was given transfusions at intervals, but she continued to show purpuric lesions and bled from the gums, the gastro-intestinal tract, and finally the uterus. The spleen was not enlarged. Repeated blood films showed no abnormal white cells, the platelets were never higher than 50,000, and the bleeding-time always exceeded 30 minutes. Splenectomy was undertaken as a last resort, but the patient died within 24 hours. Necropsy did not immediately reveal the cause of death, but this was clear from the report on the microscopical examination of the tissues, for which I am indebted to Dr. Robertson Ogilvie. "*Bone Marrow:* The outstanding change is marked irregular haemorrhage (Fig. 3). Otherwise it shows a moderate reaction along both normoblastic and leucoblastic lines, mainly the former, and also a scanty, diffuse infiltration with mature lymphocytes. *Liver:* Some of the portal tracts are enlarged owing to a considerable infiltration with lymphoid cells, mature and immature. Similar cells are present in moderate numbers in the blood of the sinusoids. *Kidney:* Many of the vessels contain mature and immature lymphoid cells, often in conspicuous numbers (Fig. 4). *Comment:* The changes in the tissues, and especially the presence of excessive and immature lymphoid cells in the vessels and the increase of lymphoid tissue in the portal tracts of the liver, indicate that the case has been one of lymphatic leukaemia."

This was an ill-advised operation, but there is comfort in an observation made by Wintrobe (1946): "In a few instances the final answer may have to be given at necropsy, when the characteristic histological changes of leukaemia are found."

The third patient, a grossly obese woman aged 55, had been under observation for three months as a case of thrombocytopenic purpura, when she was admitted to the Royal Infirmary of Edinburgh suffering from a subarachnoid haemorrhage. The spleen was not palpable. The platelets numbered 20,000, the bleeding-time was eight minutes. It now transpired that for one month before the onset of her purpuric signs she had been taking a preparation containing phenacetin. The interval since the drug had been stopped was long enough for me to accept the responsibility for splenectomy. There was no improvement in her condition, and her platelet count did not rise. Later she died in a medical ward as the result of massive intracerebral haemorrhage.

Comment.—Splenectomy is usually effective in the treatment of idiopathic thrombocytopenic purpura. Other causes of purpuric manifestations must first be excluded, in particular by the examination of a specimen of bone marrow.

Haemolytic Anaemia

The surgeon whose opinion is asked on the management of a case of haemolytic anaemia can approach the problem with the knowledge that his haematological colleague will have made every attempt to exclude the secondary forms of the disease in which normal erythrocytes are haemolysed by some known extraneous agent. This may occur in both pyogenic and protozoal infections; as the result of the absorption or ingestion of a considerable number of chemical compounds; and in carcinomatosis and certain of the reticuloses.

There remains the primary group of haemolytic anaemias, and it is customary to divide these into *congenital* and *acquired* types. The work of Loutit and Mollison (1946) has shed much light on the differences between these two forms. They believe that in the *congenital form* the process of formation of red blood corpuscles is congenitally defective in that they are abnormally sensitive to the normal process of species-specific haemolysis constantly going on in the blood. They consider that the first step in the breaking down of these abnormal red cells is their conversion to spherocytes in the blood stream, that lysolecithin is the agent responsible, and that in the backwaters of the spleen pulp there is more time for this lysin to act. Treatment by splenectomy is successful because it removes the conditions most favouring haemolysis although the red blood corpuscles remain abnormal.

In the *acquired type* the red blood corpuscles are not abnormal. When transfused into a normal recipient they survive for the normal length of time. The haemolysis is the result of the presence in the patient's blood of abnormal known or unknown lytic agents, destructive alike to the patient's red blood corpuscles and to those of any normally compatible transfusion donor. Destruction of the patient's erythrocytes or those of a donor may also result from a significant reduction in titre of that fraction of the patient's serum which acts as non-specific inhibitor of lysin.

Congenital Haemolytic Anaemia

This is recognized by its familial incidence and by the association of splenomegaly with anaemia, spherocytosis, increased fragility of red cells, and jaundice. It is usually discovered in early life, but may be so mild as to escape detection until late adult life. Its severity varies, but tends to be fairly constant in any one family. The course of the disease is punctuated by crises—the first of which may lead to its detection—characterized by increased haemolysis with fever, abdominal pain, and increased jaundice. Sometimes several members of a family are affected by a crisis about the same time; the literature has been collected and a remarkable series recorded by Horne *et al.* (1945). In well over half the cases small pigment stones form in the biliary passages and may provide the nuclei of larger "mixed" calculi.

In these cases splenectomy should be advised, and urged if there is a history of severe crises in other members of the family, and if there is persistent leucopenia. It is impossible to foretell the likelihood of biliary complications (including acute cholecystitis), or the occurrence of severe haemolytic crises; in a child, if possible, operation should be deferred until the age of 10 is reached. When the disease attracts attention on account either of acute biliary obstruction or of acute cholecystitis, this alone should be dealt with at the first operation. As soon thereafter as possible the spleen should be removed lest further calculus formation occurs.

When the presenting symptoms are not biliary, although stones are known to be present or when stones are discovered at operation, splenectomy should be performed, the common bile duct opened, washed out, and drained, and the gall-bladder removed. Sometimes pigment stones can be demonstrated by cholangiography in the smaller intrahepatic biliary passages, and the "T" tube should never be removed until cholangiography has shown that all ducts are free from calculi. On one occasion when I neglected to ensure this I had to reopen the common duct a year later to remove a "family" of mixed stones. If the patient is seen during a crisis severe enough to require transfusion of blood, I believe that this should be limited to the minimum amount necessary, because splenectomy is followed by a rapid increase in blood count. Multiple transfusions may introduce complicated haemolytic reactions.

When a woman suffering from congenital haemolytic anaemia becomes pregnant, usually there is not any increase in the anaemia, but if this does occur towards the end of pregnancy transfusion of blood is the correct treatment, not splenectomy.

In my unit splenectomy has been performed in 11 cases of congenital haemolytic anaemia without operative mortality; the operation is easy because of the absence of vascular adhesions. Ten patients have remained well for periods up to 10 years. The remaining patient failed to benefit from the operation, although the clinical features were typical.

Acquired Haemolytic Anaemia

I have performed splenectomy in 15 cases of haemolytic anaemia which appeared to be of the acquired type, with two post-operative deaths. In one of these an acute process progressed unchanged; in the other, of a more chronic type, oliguria with failure of the peripheral circulation supervened on the fourth day in spite of steady improvement in the haematological picture. Many of the patients who survived were very ill at the time of operation (one had a haemoglobin value of 20%), so that in such cases splenectomy is not attended by a prohibitive risk.

When a surgeon is asked for his opinion of the value of splenectomy in these cases there are certain broad principles which may help him to a decision. First, it is the general experience that the patients who are most likely to respond to operation favourably are those whose haematological picture resembles that of congenital haemolytic anaemia in the presence of spherocytosis and increased capillary fragility (Davis, 1943), although the absence of these findings does not preclude a successful outcome. Secondly, one has to balance against the risk of operation the likelihood of spontaneous recovery after an interval during which life is maintained by transfusions of blood. In my opinion the operative risk is small, and therefore I believe that the responsibility for splenectomy should be accepted if the patient fails to improve after two or three adequate transfusions; this policy is to be preferred to splenectomy as a last resort after multiple transfusions have failed, because these introduce the danger of complicated haemolytic reactions in addition to that of the primary cause of the condition.

Of the 13 patients who survived operation two are untraced and two died of the disease within a year. The

remaining nine are alive after a lapse of from one to seven years, seven in good clinical condition, and two (after 12 and 84 months) in only fair health. Thus there seem grounds for maintaining that a patient suffering from acute haemolytic anaemia should not be allowed to die without the possible benefit of splenectomy performed as soon as transfusion fails. Combined experience shows that the operation will be effective in rather more than 50% of cases.

An interesting case of haemolytic anaemia of the acquired type has been recorded by West-Watson and Young (1938). "Eventually, after four months in hospital, during which splenectomy, three transfusions, and liver therapy had failed to arrest the haemolysis, it was decided to explore again in search of a spleniculus. This was done, but none was found; the gall-bladder and the pelvic tumour, which proved to be an ovarian teratoma, were removed. No transfusion was given. Unexpectedly the haemolysis ceased immediately, the signs of toxaemia disappeared, the temperature settled, and the blood began to return to normal, complete recovery ensuing." The teratoma did not contain any splenic tissue. Other cases have been recorded of recovery from haemolytic anaemia after the removal of an ovarian tumour (Singer and Dameshek, 1941; Jones and Tillman, 1945).

Comment.—Splenectomy should be advised in cases of congenital haemolytic anaemia. In cases of acquired haemolytic anaemia for which no extraneous agent is responsible, splenectomy should be considered early, before multiple transfusions introduce complicating factors.

Splenic Neutropenia

This condition was originally called "splenopathic leucopenia" (Frank, 1925), and it was known that the granulopenia might be associated with a reduction in number both of the other cells in the blood and of the platelets. A primary deficiency in the number of granulocytes due to enlargement of the spleen is rare; but an attempt has been made to segregate such cases in a separate group (Wiseman and Doan, 1942), and an interesting case in which the agranulocytosis was cyclical has been recorded (Fullerton and Duguid, 1949).

I have twice performed splenectomy for neutropenia presumed to be of splenic origin. In one case this tentative diagnosis proved to be incorrect, the presenting haematological features being the initial signs of leukaemia.

A woman aged 48 had suffered intermittently for two years from septic lesions of the skin, when her blood was found to show agranulocytosis. The haematological features were: haemoglobin, 92%; red cells, 4,290,000 (reticulocytes 1%); white cells, 1,600 (neutrophils 14%, lymphocytes 69%, eosinophils 1%, mononuclears 6%); platelets, 104,000; sternal marrow, sheets of lymphocytes—no polymorphs or their precursors.

The standard methods of medical treatment failed to produce any increase in the white count, and, as splenic neutropenia was thought to be a possible diagnosis, after protracted discussion I somewhat reluctantly agreed to perform splenectomy. The pathologist reported that the microscopical appearance of the spleen was consistent with a diagnosis of "Banti's disease." After operation there was improvement for three months, the total white count remaining about 6,100, with polymorphs 34–35%, lymphocytes 20–30%, monocytes 10%, and eosinophils 25%. The platelet count rose to 450,000. During the subsequent two years the blood count has tended towards its pre-operative values, purpuric spots have appeared on the legs, and ulceration of the mouth has been troublesome; however, the patient feels well and is at work. The latest count showed: haemoglobin, 76%; white cells, 2,400 (polymorphs 41%, lymphocytes 56%, monocytes 1%, eosinophils 2%). Thus splenectomy has not altered the course of the disease, which has declared itself as lymphatic leukaemia of a chronic form.

The second patient, a man aged 55, had a remarkable clinical history. Before right middle and lower lobectomy for bronchial carcinoma by my colleague Mr. Andrew Logan in May, 1950, his white count was 10,000 and his spleen was not enlarged. Six days after operation his white count was found to be 2,400 and his spleen was easily palpable. The white count remained persistently low (1,600 to 2,600), and there was moderate thrombocytopenia (120,000). I removed his spleen three months after the pneumonectomy, and within five hours the white count had risen to 6,800 and the platelets to 272,000. This improvement in the blood picture has been maintained for seven months, in spite of an attack of hepatitis as a complication of a plasma infusion given after the pneumonectomy. Up to the present he has gained 35 lb. (15.9 kg.) in weight; the white count at the last determination was 12,200, the platelets 465,000, and the red cells 5,690,000.

The precipitating factor in this second case remains obscure. At laparotomy no hepatic or other metastases were detected, and the patient's steady general improvement is evidence against the presence of metastases elsewhere.

Congestive Splenomegaly

In a considerable group of cases in which splenomegaly is present the clinical features include, singly or in combination, anaemia, ascites, and bleeding from the gastro-intestinal tract. These conditions—for there is certainly no single aetiological factor concerned—were formerly labelled "Banti's disease." A more modern label, "hepatolienal fibrosis," is not entirely satisfactory because sometimes the liver is macroscopically and microscopically normal, and splenomegaly is absent in about 20% of cases of even advanced hepatic cirrhosis. The label "congestive splenomegaly" is not completely descriptive, because in a small number of cases obstruction to the portal circulation cannot be demonstrated

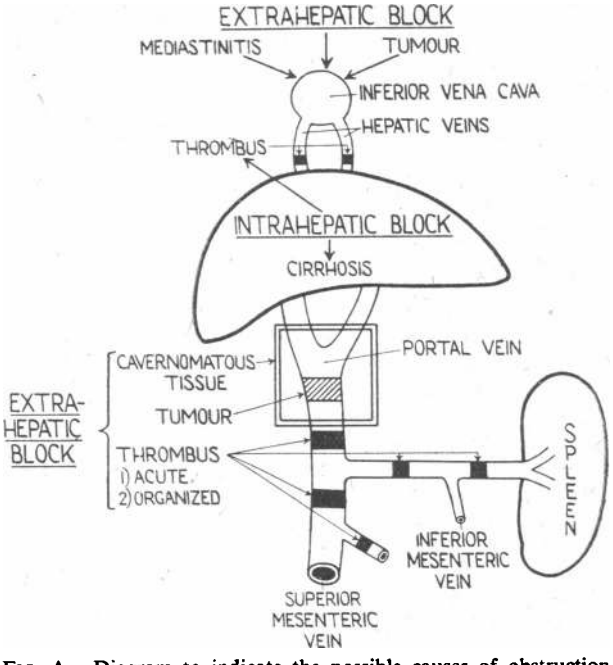

FIG. A.—Diagram to indicate the possible causes of obstruction to the flow of blood in the portal system.

even at necropsy. In the majority the pressure is increased in the portal vein and in its tributaries, including the splenic vein. I do not intend to analyse in detail the various factors which may lead to portal hypertension ; they are sufficiently indicated for my purpose in Fig. A, where they are separated into *intrahepatic* and *extrahepatic* according to the views expressed in the classic paper of Whipple (1945). There are as yet no completely reliable methods for diagnosing the site of the block, even on the operating table. It has been indicated that there may possibly exist a condition of " idiopathic " portal hypertension without demonstrable anatomical block ; in another group of cases the portal vein may be partially or completely obstructed by thrombosis, although the liver remains macroscopically normal (McNee, 1932b) ; and in a third splenomegaly may be the only gross pathological feature (McNee, 1932b). In these circumstances I propose to evade the aetiological issues and to consider the surgical problems in certain groups of cases in which the diagnosis is either tolerably certain or definitely established on the operating table or at necropsy.

(1) Splenomegaly with Hepatic Cirrhosis

In such cases the risks to the patient are protracted anaemia, persisting ascites, gastro-intestinal haemorrhage, and hepatic failure. Within the limits set by the title of this paper the possible operations are splenectomy, and splenectomy with anastomosis of the splenic vein to the left renal vein.

(a) Splenectomy Alone

In cases of hepatic cirrhosis there is no question of any beneficial effect of splenectomy on ascites.

That the presence of the enlarged spleen (hypersplenism) may reduce all the cellular elements of the blood is undoubted ; the anaemia may persist in the absence of even occult bleeding from the alimentary tract. It is normocytic and characterized by a continuing normal ratio between polymorphonuclear leucocytes and lymphocytes ; thus in cirrhosis the haemopoietic principle is normally produced and normally stored (Huang and Wang, 1949). Davidson (1934) has shown that anaemia resulting from gastro-intestinal bleeding responds to the administration of iron, and we have found the response to be most rapid when the iron is given intravenously, possibly because when it is given by mouth its absorption by the congested mucosa of the jejunum is incomplete. It has been my experience that the panhaemocytopenia of congestive splenomegaly is relieved by splenectomy in about 75% of cases, and this has been confirmed by Robson (1949a). This has altered my attitude to splenectomy in early cases, because it is obviously desirable that a damaged liver should receive the best possible qualitative supply of blood. Between 1932 and 1938 I operated on seven patients for Professor Davidson, all in the late stages of the disease, and of these three died. We gave up splenectomy for six years. Since 1944 I have performed splenectomy on 25 patients suffering from this condition, most of whom had reasonable hepatic function ; of these one died.

When the condition is more advanced and hepatic function somewhat reduced, I believe that it is still justifiable to perform splenectomy for the relief of hypersplenic panhaemocytopenia. In these circumstances it is advisable to attempt to improve hepatic function during the pre-operative period by arranging for the patient to have a diet high in carbohydrate and protein and low in fat and sodium. The Dietetic Department of the Royal Infirmary of Edinburgh provides a palatable daily diet containing : carbohydrate, 260 g. ; protein, 135 g. ; fat, 71 g. ; and sodium, 0.5 g.

The arguments for performing splenectomy in an endeavour to prevent bleeding from oesophageal varices are less secure. On the one hand it has been stated that the operation reduces the arterial input into the portal system by about 20%, and interrupts the direct connexions by way of the vasa brevia between the splenic effluent and the oesophageal varices. On the other hand it has been contended that splenectomy interrupts the many collateral venous channels between the portal and systemic systems formed in connexion with the enlarging spleen. I have considered these pathways in detail elsewhere (Learmonth, 1951), but here I give my impression, gained from observation at operation on such patients, that the enlarged veins between the spleen and its surroundings which are divided in the operation of splenectomy are dealing almost solely with the splenic effluent. Valuable channels connecting the main splenic vein to the pancreatic and adrenal veins remain undisturbed, and their contents ultimately reach the left renal vein by way of the left adrenal vein and the left ascending lumbar vein. This is not the only factor to be taken into consideration : after splenectomy oesophageal varices are still exposed to the increased portal pressure because of their connexions with the left gastric (coronary) vein. To divide the left gastric vein also gives only temporary relief because of the relatively rapid formation of new venous channels to replace it.

Statistics give an equivocal answer. When all published cases are considered it appears (Howells, 1938) that splenectomy does not add to expectation of life. On the other hand, Pemberton and Kiernan (1945), whose series is large enough for statistical analysis, have shown that if haematemesis has occurred before splenectomy it will recur in 53.7% of cases, whereas if haematemesis has been absent before splenectomy it will occur afterwards in only 15.2%. It has been contended that peptic digestion of the walls of the uncovered or thinly covered oesophageal varices provides another variable in the incidence of haematemesis, but in most of these cases gastric analysis shows hypochlorhydria or even achlorhydria. It is generally held that after splenectomy the early occurrence of haematemesis, or its repetition at short intervals, is of grave prognostic significance.

It seems to me that removal of the spleen should be considered when its enlargement is associated with hepatic cirrhosis. Pemberton and Kiernan (1945) hold that splenectomy may, at least in some cases, remove the site of formation of a possible toxic substance, a view to which McNee (1932b) also subscribed. The hazards of the operation are said to be reactionary haemorrhage, thrombosis in the portal system as a result of an unusually great post-operative increase in the number of circulating platelets, and hepatic failure. Of the three deaths in my early series (seven cases), two resulted from respiratory complications and one from reactionary bleeding. The death in my second series (32 cases) provided the only example of portal thrombosis following splenectomy that I have seen. It was widespread and involved even the natural portal-systemic communications of the azygos veins.

Many of my patients had sclerotic changes in the walls of the splenic or portal veins, but I cannot correlate this finding with a tendency to post-operative thrombosis ; in one patient the portal vein was patent, although

it was so calcified that it could be identified in a radiograph. It has been held that the danger of widespread thrombosis of the portal system after splenectomy, as a result of a rapid increase in the number of platelets, imposes some less radical surgical procedure when the pre-operative platelet count is normal or nearly normal. McNee (1931c) suggested ligation of the splenic artery, but there is an anatomical objection to this, because a collateral circulation is provided by way of the gastro-epiploic arterial arcade. I have tied and divided the splenic and right gastro-epiploic arteries on four occasions; in one case I removed the spleen later. The spleen shrinks and the blood count improves, an observation which I regard as of some importance in explaining the part played by the organ in producing haemocytopenia. Later the spleen enlarges again, and haematemesis and haemocytopenia may recur. Thus in one patient the figures were :

Time	Hb	R.B.C.	W.B.C.	Platelets
Pre-operative ..	65%	4,170,000	1,600	156,000
After ten days ..	50%	2,820,000	22.000	615,000
After five months	70%	3,600,000	4,300	209,000

I do not think that the ligature operation need be retained, except in such rare cases as one in which I found the portal and splenic veins thrombosed, and a huge vein passing down the hilum of the spleen to the retroperitoneal tissues; this appeared to be draining most of the portal blood.

I believe that estimates of hepatic function deduced from the results of several tests are of more value in ultimate prognosis than in defining operative risk, provided that anaesthetist and operator are experienced, and that any lost blood is rapidly replaced by transfusion, in order to avoid anoxia of the liver. It is certainly unwise to offer or to withhold operation according to the result of any one test—for example, estimation of serum proteins.

My view is that in the present state of knowledge the value of splenectomy alone in this group of cases will be found to depend largely on the improvement it produces in the blood picture. The causes of hepatic cirrhosis are so numerous and its evolution is so variable that it is almost impossible to compare any one case with others. Fortunately the risk of the operation has been so reduced that it can be used with judgment for this single purpose. However, it must be remembered that when the patient is a child the prognosis is bad; it has been shown that three out of four children die within five years of the onset of the disease (Logan, 1950), and my own experience is similar.

It has been stated by Robins (1941) that when a woman suffering from this condition becomes pregnant the evolution of the disease is more rapid and there is an increased liability to post-partum haemorrhage. He recommends that the pregnancy should be terminated if the complicating condition is diagnosed before the foetus is viable. On the other hand, I record through the courtesy of Professor John Craig the unusual late history of one of my cases in this group.

In 1926, at the age of 7 years, the girl had melaena; the spleen was not palpable. She was well until 1935, when she had several haematemeses, and in June of that year I removed a large spleen. In November, 1935, there was another haematemesis, and in 1937 still another. Between 1926 and 1937 she had several transfusions, the details of which are not now available. Thereafter she remained well, married, and in 1944 had a normal pregnancy, the baby being normal. In 1949 she gave birth to an infant which showed severe erythroblastosis with jaundice, from which it recovered. The mother, who is Rh-negative, is alive and well.

(b) Splenectomy as a Preliminary to Venous Shunts

In 18 cases I have performed splenectomy as a preliminary to splenic renal venous anastomosis, and in another case, in which the arrangement of the splenic vein proved to be unsuitable for this operation, I added an anastomosis between the portal vein and the inferior vena cava. Five of the former 18 patients died after operation. In two the cause of death was hepatic failure, and in retrospect the operation was ill-judged: in the remaining three death was due to massive haemorrhage—in two instances into the gastro-intestinal tract from pre-existing oesophageal varices and in one instance in the field of operation.

The first 13 of these operations were end-to-end anastomoses, 11 made over a vitallium tube and two direct. In my hands the results of this technique have been unsatisfactory; of the eight patients who survived splenectomy with end-to-end anastomosis, haematemesis has recurred in five, at intervals of from 12 to 42 months. The first patient on whom I performed this operation died from massive haematemesis 42 months later, having cycled 50 miles and played in a football match on the previous day. At necropsy the anastomosis, which had been made over a vitallium tube, was blocked by old thrombus. The tube method has not been widely adopted, but great credit is due to Blakemore and Lord (1945) for the idea, which has been to vascular surgery what Murphy's button was to intestinal surgery. It is probable that an end-to-side anastomosis between the splenic and left renal veins (Linton *et al.*, 1947) will remain patent in a higher proportion of cases; I have performed this operation in five cases without operative mortality.

My present opinion is that if it is desired to add a "shunt" operation to splenectomy, end-to-side splenic renal anastomosis should be chosen. But I am convinced that the final treatment of splenomegaly with hepatic cirrhosis must await more accurate methods of assessing the probable rate of evolution of failure of hepatic function, and consequently more accurate conceptions of its pathological causes. I remember vividly one patient I saw, an elderly man who had undergone splenectomy at the hands of Lord Moynihan. He bled at intervals for five years after the splenectomy, and thereafter had remained well for 21 years : if he had had a shunt operation he would have been equally cured.

The patient who was treated by splenectomy with portal-caval anastomosis 10 months ago survived operation, and has not as yet bled again.

(2) Splenic Vein Thrombosis

Thrombosis limited to the splenic vein may result from any of the causes of thrombosis, but is probably rare. In such cases increase in pressure is limited to this vein, to its tributaries, and to its collaterals. It seems the most likely anatomical diagnosis in a patient referred to me by Dr. F. G. Hobson.

As a boy of 2 years he had an infection which led to thrombosis of the inferior vena cava. At 14 he was acutely ill with abdominal symptoms, and it was concluded that further venous thrombosis had occurred. At 29 he had a sudden severe haematemesis; after he had recovered large oesophageal varices were demonstrated by radiography and

his spleen was found to be enlarged. Dr. Hobson and Professor Witts thought that he might be suffering from partial blockage of the hepatic veins (Chiari's syndrome) or thrombosis of the portal vein or splenic vein. After a good deal of discussion I explored his abdomen. The liver was quite normal; all the collateral venous channels began in the spleen and passed chiefly towards the cardia. The splenic vein as such was absent, a number of small veins taking its place. The spleen was removed. He recovered from a most difficult operation and has had no further bleeding for 29 months, although his health has deteriorated for other and probably unconnected reasons.

This is the only case in my series in which I have been reasonably certain of the diagnosis of thrombosis of the splenic vein; it was not associated with hypersplenism. I have made this diagnosis on several occasions, only to be proved wrong by the subsequent course of events or by necropsy, which showed either progressive hepatic cirrhosis or, in one case (see below), a cavernous haemangioma in the portal fissure.

Comment.—Splenectomy should be considered in "congestive splenomegaly" when haemocytopenia is present and it precedes splenic renal end-to-side venous anastomosis when that operation is chosen to attempt to reduce portal hypertension. It is also indicated in thrombosis of the splenic vein.

Miscellaneous Conditions

1. *Splenomegaly of Undetermined Origin.*—This case presented many unusual features.

At the age of 4 this girl was admitted to the Royal Hospital for Sick Children, Aberdeen. Her abdomen had been protuberant since birth, and the cause of this was a large spleen. Neither then nor subsequently was there any evidence of hypersplenism. Two years later she had two episodes of haematemesis and proved to have extensive oesophageal varices. At laparotomy in the Royal Infirmary of Edinburgh the liver appeared to be normal, and this was confirmed by microscopical examination of a portion removed at operation. The splenic vein was represented by a number of small veins which disappeared into a hard pancreas; in one of these the pressure was 190 mm. of saline solution. Splenectomy was performed. She did well for 33 months, when she was again admitted to the Royal Hospital for Sick Children, Aberdeen, with an obscure febrile illness of which gastro-intestinal bleeding was one sign. Later, ascites appeared. Finally she died, and I am indebted to Professor John Craig for the findings at necropsy:

"Despite the intense fatty change, the general liver architecture appeared normal, and there did not seem to be any evidence of a former cirrhosis. The larger portal tracts contained lesions of polyarteritis nodosa involving branches of the hepatic artery. Not only did the lesions vary in age but the size of the artery affected varied. In the kidneys the lesions of polyarteritis nodosa of varying stages were seen in arteries of varying sizes. The gastric mucosa showed mainly post-mortem autolysis, but situated almost immediately under the mucosa was a small artery showing polyarteritis nodosa. In the small intestine the changes were those of non-specific mucosal ulceration. It was of interest to note that almost invariably a lesion of polyarteritis nodosa was seen in the underlying submucosa and there was much distension of the neighbouring vessels."

Sulphadimidine had been given the day before admission to hospital, but only after the illness had assumed its final form. It is to be noted that microscopical examination of a portion of the liver at the time of the splenectomy had shown the organ to be quite normal. It is just possible that the original lesion may have been thrombosis of the splenic vein.

2. *Cavernomatous Transformation of Portal Fissure*:

The patient, a girl of 16, had had previous haematemeses at 5 and 8 years of age. Haematemesis recurred without warning and at first was treated by transfusion. The spleen was enlarged, but the only evidence of hypersplenism was moderate thrombocytopenia (170,000). There was no radiological evidence of oesophageal varices. At operation the liver appeared to be normal, and the only visible collateral venous channels were in relation to the enlarged spleen. The condition was thought to be due to thrombosis of the splenic vein, and splenectomy was performed. The patient was discharged from hospital three weeks later, when the platelet count had risen to 575,000. She remained at work on a farm for 23 months and then suddenly had a large fatal haematemesis. The necropsy revealed large oesophageal varicosities. The reason for the portal hypertension was not found until the liver was cut open, when a haemangiomatous mass 5 cm. in diameter was found replacing the two terminal branches of the portal vein. The mass was examined microscopically by Professor Murray Drennan, who considered that it was a congenital malformation (Pick, 1909) and not a collection of collateral veins replacing thrombosed portal branches. In the wall of the portal vein itself was a large calcified ulcer. Microscopical examination of the liver showed it to be normal in every respect.

3. *Tumours.*—It is well known that the incidence of tumours in the spleen is low; Krumbhaar (1927) stated it to be 0.1% of primary tumours and about 0.6% of secondary tumours. The reason for this low incidence is unknown. My only experience of splenectomy for tumour was in a patient whose spleen was enlarged as part of the picture of histiocytic medullary reticulosis; the diagnosis was made only after microscopical examination of the spleen. This patient died of the primary disease a year after her splenectomy.

4. *The Lipoidoses.*—Splenomegaly may occur in certain disturbances of metabolism as a consequence of the excessive storage of lipoid substances in the reticuloendothelial cells of the spleen. These conditions require splenectomy only if the bulk of the spleen is disabling, or if there is pronounced anaemia, leucopenia, or panhaemocytopenia from hypersplenism. I have removed the spleen for the second reason in Gaucher's disease; an aneurysm of the splenic artery was also present. Dr. Sherlock and I (1942) reported the case in detail. As is usual in such cases, splenectomy restored the blood picture to normal.

5. *Tuberculosis.*—I have had one case of tuberculosis of the spleen, in a middle-aged woman whose presenting clinical features were anaemia and splenomegaly. Unfortunately her record has been lost, and I have been unable to trace her subsequent history. The operation made a considerable impression on me, because the liver was also affected, but only its left lobe, which was studded with tubercles. Although a single one, this finding lends some support to the view that the flow of blood in the portal vein is streamlined, a view discussed in detail by McNee (1932a). The literature of tuberculosis of the spleen has recently been reviewed by Guild and Robson (1950), who record a case associated with polycythaemia vera. It would appear that enlargement of the spleen as the result of tuberculous infection leads to the same reduction in the numbers of erythrocytes, leucocytes, and platelets as is found in many other forms of splenomegaly.

6. *Splenectomy in Course of Other Operations.*—On nine occasions the spleen has been removed during gastrectomy: in four patients—one of whom died—the operation was for peptic ulcer; in four for carcinoma

of the cardia, and in one (who died after operation) for an extensive gastric neoplasm which proved to be a sarcoma; on two occasions the spleen has been removed with a carcinoma of the splenic flexure of the colon.

Conclusions

My whole experience is summarized in Table II. Clearly it has been uneven, and it cannot be called venturesome or even exploratory. Except for certain mistakes in diagnosis the indications for splenectomy have been orthodox. The operative mortality has not been prohibitive.

TABLE II

Condition	Operation	No. of Cases	Operative Mortality
Rupture of spleen	Splenectomy	1	0
Thrombocytopenic purpura	,,	19	1
Symptomatic purpura	,,	3	2
Congenital haemolytic anaemia	,,	11	0
Acquired haemolytic anaemia	,,	15	2
Splenic neutropenia	,,	2	0
Congestive splenomegaly	Splenectomy	32	4
	Ligation of splenic artery	4	0
	Splenectomy and splenic renal anastomosis	18	5
	Splenectomy and portal-caval anastomosis	1	0
Splenic thrombosis	Splenectomy	1	0
Splenomegaly of undetermined origin	,,	1	0
Cavernous haemangioma of liver	,,	1	0
Histiocytic medullary reticulosis	,,	1	0
Gaucher's disease	,,	1	0
Tuberculosis of spleen	,,	1	0
Splenectomy in course of other operations		11	2
Totals		123	16

As I have reread my case notes, certain conclusions—perhaps not all new—have been forced upon me.

1. The rapidity with which symptomatic improvement usually occurs after splenectomy for idiopathic thrombocytopenic purpura and for congenital haemolytic anaemia. If one ligates the splenic artery at operation for idiopathic thrombocytopenic purpura, and waits long enough before completing the removal of the spleen, the bleeding-time falls although the spleen is receiving enough arterial blood for its nutrition.

2. The curious lack of uniformity in size of spleens concerned in pathological processes. (*a*) In idiopathic thrombocytopenic purpura the spleen is rarely much enlarged. Yet an accessory spleen reproduces the disease clinically only after its size has increased. (*b*) In congenital haemolytic anaemia the spleen is enlarged. Again an accessory spleen must grow before it reproduces the disease clinically. There is something quantitative about this—quantitative in toxic substance, or in volume of spleen pulp, or in amount of reticulo-endothelial tissue.

3. The temporary disappearance of hypersplenism after ligation of the splenic artery. There are two possible explanations: cessation of most of the blood flow through the spleen and reduction in size of the spleen. Whether one chooses the first or second (or both), again there is something quantitative about the sequence of events.

4. The catastrophic rapidity with which splenectomy in a case of acute leukaemia is followed by the death of the patient, although the operation has been easy and the macroscopic appearance of the tissues at necropsy (including the site of operation) possibly quite uninformative.

5. In contrast to paragraph 4, the rapidity with which splenectomy will terminate acquired haemolytic anaemia in a proportion of cases of undetermined aetiology.

These problems await complete solution by haematologists. In the meantime I propose to continue to use splenectomy as a therapeutic measure when it is not definitely contraindicated.

I am grateful to the many physicians and surgeons in Edinburgh and elsewhere who have entrusted patients to my care. The majority of the blood counts have been done by Professor Davidson's staff; Mr. A. I. S. Macpherson gave assistance in tracing some of the patients, and Professor W. C. Wilson kindly furnished me with particulars of some of my Aberdeen cases. Certain of the operations were performed in my unit by Mr. T. McW. Millar, Mr. A. G. R. Lowdon, Mr. A. W. Wilkinson, and Mr. A. I. S. Macpherson. The coloured photomicrographs were done by Mr. T. C. Dodds.

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