SPLENECTOMY IN EGYPTIAN SPLENOMEGALY.

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The literature of splenectomy consists mainly of reports from different surgeons who have operated on one or two cases, and collections of these accompanied by a discussion of the results. The best examples of the latter which I have been able to find are the excellent papers of Armstrong,[1] Torrance,[2] and Johnston.[3]

But it is clear that while such collections possess great value in some respects,

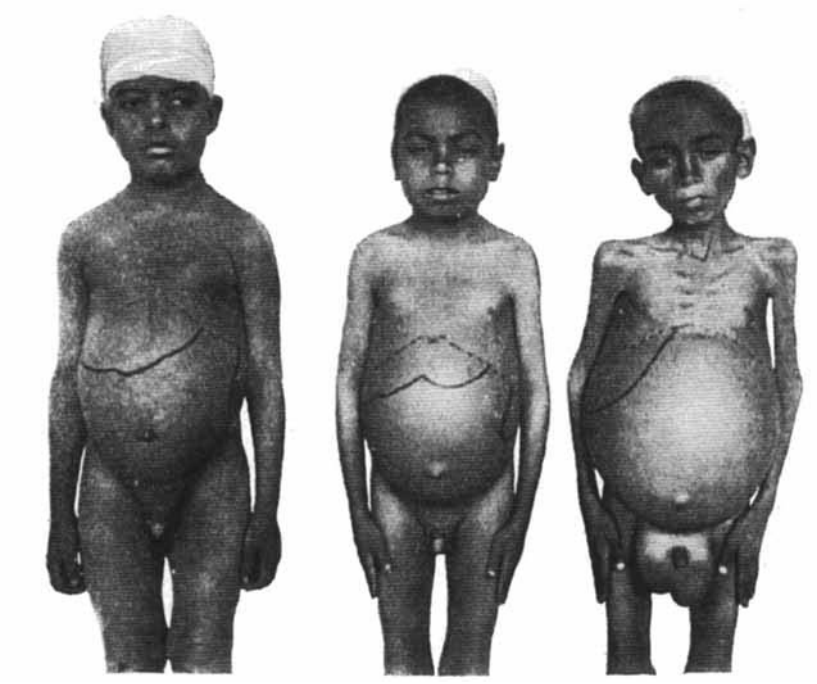


Fig. 267.—These photographs, and Fig. 268, show three stages in the progress of the disease. Note the gradual opening out of the costal angle, and the progressive emaciation. The most advanced case has developed a double hernia full of ascitic fluid.

they have two inherent defects. As one of these authors points out, a surgeon who has only performed the operation once, and has been unfortunate enough to have had a fatal result in that case, is not likely to report it; so that where so much of the literature consists of single cases, any conclusions as to mortality and results drawn from the published material can only apply to a part—and that the most successful part—of the operations actually performed.

Another defect of such records is due to the unsatisfactory nature of our present knowledge of the pathology of splenomegaly. When all the known and understood forms of this condition have been subtracted, there remains a considerable residue of cases, classed together for the sake of convenience as splenic anæmia and Banti's disease, which probably represent several distinct conditions; and if we are to estimate the value of any operation, we must first have a clear idea of what disease it is applied to.

A contribution which meets both these difficulties as far as possible is the remarkable series of cases recently published by Mayo,[4] which consists of twenty-seven consecutive operations from the same clinic. In addition, there are papers on the pathological and clinical side of the same cases by Wilson[5] and Giffin.[6] The whole forms a comparative study of a considerable mass of material at first hand by the same observers. Nine of these cases are heterogeneous, wandering spleen, tubercle, etc. The most homogeneous and, I venture to think, the most interesting part of the series are the 18 cases of the splenic-anæmia group, 8 of which showed changes in the liver (Banti's disease).

FIG. 268.—The same subjects as FIG. 267.

These cases, clinically uniform, showed considerable differences in their morbid anatomy, and are arranged by Wilson in three classes. But he concludes that it is unnecessary to assume three different causes for their production, and is inclined to think that they are all due to the presence of a slowly acting local toxin. This conclusion is in agreement with the excellent results reported after removal of the diseased spleens.

In Egypt, a disease which produces a very similar clinical and pathological picture is of frequent occurrence. It has already been described in a paper by Day and Ferguson[7] under the title of "Endemic Splenomegaly with Cirrhosis," and subsequently by Day and myself[8] under the name of "Egyptian Splenomegaly." Both these names are purely descriptive and provisional. In its main features it agrees with Banti's disease as this is usually understood, though I have been unable to consult Banti's original paper. But it presents certain marked differences from many of the cases which have been grouped under this heading. It is a very definite and uniform disease, and to call it Banti's disease would lead to its confusion with cases, some of which are probably of quite a different nature.

Possibly, with fuller knowledge, these cases and the greater part of those now called Banti's disease will be given some name derived from their cause. Meanwhile neither name connotes anything except a group of symptoms; but

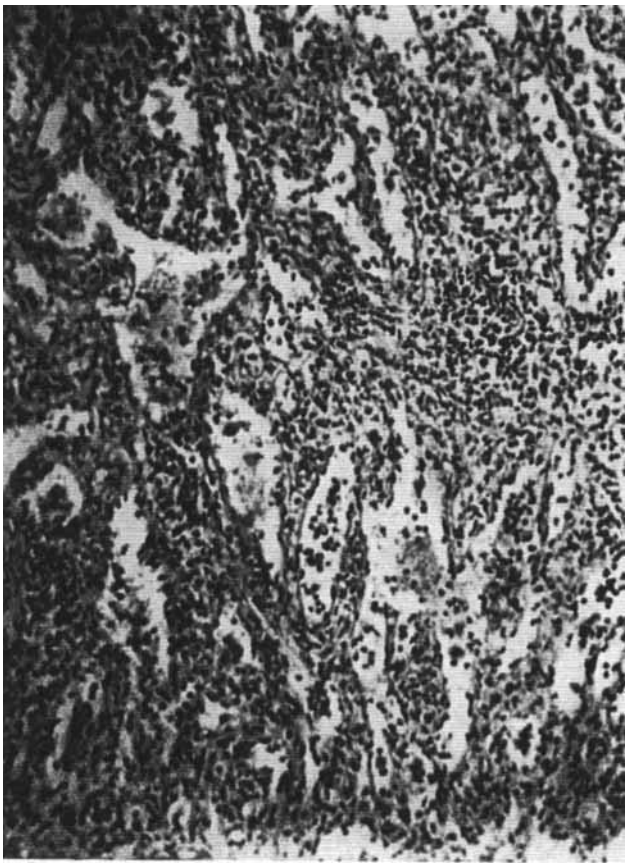


FIG. 269.

Notes and Photograph by Professor A. R. Ferguson.

Microphotograph of Case No. 8 (about 150 diameters).—The microscopical characters displayed are those shown by the organ in the later stages of the disease. The general dilatation of the vascular spaces is well shown. They are of most irregular form, and in the fresh state are always distended with blood. A large quantity of the contained blood is lost on opening a spleen received from operation. The leucocytes in the sinuses are practically all of the mononucleated variety. Polymorphonuclear leucocytes are exceedingly sparse in the fresh spleen in this disease, and these cells are always notably diminished in the circulating blood during life. There is a considerable development of young connective tissue in the reticulum of the spleen, and a corresponding degree of disappearance of the lymphoid elements proper, the result probably of pressure-atrophy. The Malpighian follicles are reduced in number; those surviving are indefinite in contour, and share in the general fibrosis. This is the case in the particular spleen from which the microphotographs were taken, though the fact is not illustrated in the photographs.

the denotation of Egyptian splenomegaly is at any rate precise, and it refers to a group of cases all of the same kind, and to no others. In this disease splenectomy, of which I am able to report a series of 22 cases, yields excellent results.

The patients who were selected for operation all showed an enlargement of the spleen, anæmia, cirrhosis of the liver, and a low leucocyte count. They ranged between the ages of 7 and 50, but only one case was over 30, and the average age was 20. Few of the patients knew their ages accurately, so these

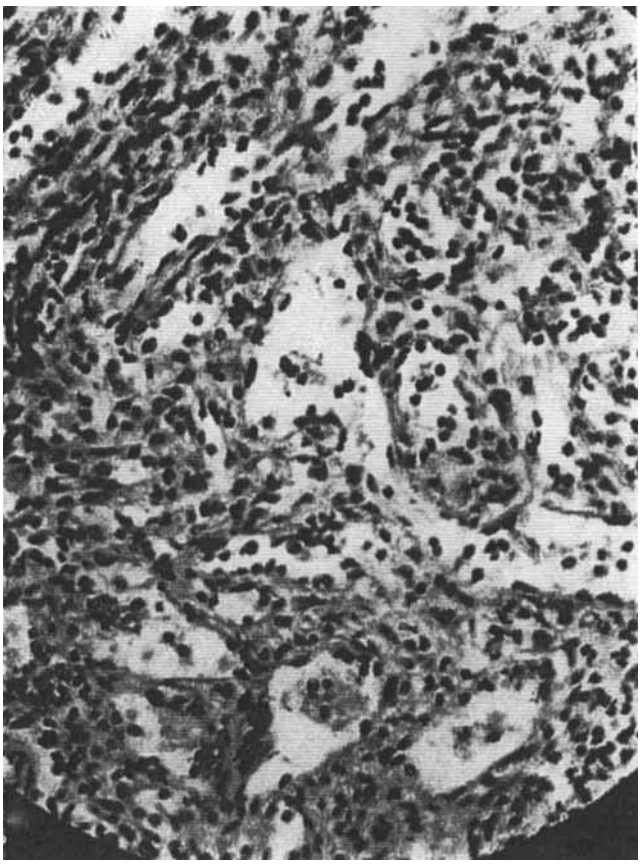


FIG. 270.

Notes and Photograph by Professor A. R. Ferguson.

Microphotograph of spleen from Case No. 8 (about 400 diameters).—The dilatation of the vascular sinuses and the general cellular fibrosis of the spleen pulp are well seen. It may be observed that many of the newly-formed connective-tissue corpuscles have nuclei of oval or pyramidal form as well as the more usual spindle-shaped type of nucleus. Many of these corpuscles, judging from their form and relation to the vascular sinuses, take their origin from proliferating endothelium. Note the exclusively mononuclear character of the leucocytes lying within the sinuses. The relative disappearance of the lymphoid tissue of the spleen is more strikingly apparent.

figures are only approximate. All except three were males, and they were all of the labouring class.

In nearly all cases they gave a history of attacks of fever, and of splenic enlargement, first noticed after these attacks and gradually increasing, with or without recurrence of the fever. Without exception they suffered from cirrhosis

of the liver, and in advanced cases from ascites. Their anæmia was often aggravated by coincident bilharzia or ankylostoma infection. The complaint for which they sought relief was usually the size of the spleen, and pain caused by adhesions round it. Several were accustomed to provoke vomiting to relieve the pain caused by filling the stomach with food, and one man complained that when he walked about, his spleen swung like the pendulum of a clock. Several had attacks of fever in the hospital.

There seems to be a seasonal variation in the accesses of the disease, for with four exceptions all the cases applied for admission in the six months from March to August, that is to say, in the hot weather.

The condition gradually progresses to ascites, emaciation. and death. Anæmia is never severe enough to cause death, and hæmatemesis is rare. The sufferers die apparently from exhaustion, the result of the long-continued drain brought about by intrahepatic obstruction of the portal system. In favourable cases the disease may be arrested spontaneously, but these patients do not come to hospital, or come for some other reason.

The illustrations (*Figs.* 267 and 268) give a good idea of the successive stages of the disease.

The patients have not been benefited in any way by quinine or salvarsan; they do not have malarial parasites; and careful and repeated search has failed to reveal any organism of the Leishman-Donovan type.

The nature of the complaint is unknown, and it would be out of place to attempt to discuss it here. Full details will be found in the papers referred to above. The changes in the spleen are a general hyperplasia, that in the liver a multilobular cirrhosis. It is clear that it constitutes a definite and consistent disease, probably of infectious origin. It resembles in many respects the cases grouped by Mayo under splenic anæmia, but differs from them chiefly in the accesses of fever, the seasonal variation, and the early and invariable cirrhosis.

FIG. 271.—*Case* 11.—An advanced case with ascites and jaundice. The boy weighed 6 st. 12 lb., and the spleen over 6 lb. He made a good recovery, but suddenly developed portal thrombosis on the seventeenth day, and died next day.

Professor Ferguson has kindly prepared microphotographs (*Figs.* 269, 270) from one of the spleens removed. They show clearly the changes which take place in these cases.

The total number of splenectomies performed for this condition in my section of the hospital is 22, of which 4 were fatal in the sense that the patients died before they left hospital. In 2 cases operation had to be abandoned, and both

patients recovered. These two cases are reported, but of course not counted as splenectomies. No other splenectomies have been performed in my section during the period covered by this series (August 1910 to October 1913).

Of these operations, 5 were performed by my assistant surgeon, Dr. Aly Bey Ibrahim Ramez, who also gave me most valuable help in nearly all the others. I have to thank him for kindly allowing me to include his cases in the series, which thus represents our total combined experience.

The first 11 cases have already been published elsewhere.[8]

Fatal Cases (*Cases* 7, 8, 11, 16).—These are four in number, of which two only (*Cases* 7 and 11) had progressed to the stage of ascites. Each had yielded over six litres of fluid on tapping. *Case* 7 succumbed on the third day, with delirium and coma. *Case* 11 (*Fig.* 271) made an apparently complete recovery, and was transferred at the end of a fortnight to the medical side. On the eighteenth day he developed portal thrombosis, and died very soon. I have since made it a rule not to operate on any case which has progressed to the stage of ascites.

FIG. 272.—*Case* 8.—This patient died of acute dilatation of the stomach.

The third (*Case* 8) died with a typical acute dilatation of the stomach, with copious secretion of a greenish watery fluid (*Fig.* 272). The fourth case sank steadily, and died the next day. He had double chronic pleurisy, a very fatty heart and kidneys, ankylostoma infection, and extensive bilharziasis of bladder, ureters, and rectum. Eggs were also found in the lungs, while the cirrhosis of the liver was periportal and contained calcified ova. I was at first inclined to consider this an error of diagnosis, and that it was a case of enlarged spleen secondary to a bilharzial cirrhosis; but Professor Ferguson is of opinion that the splenic enlargement was too great to be due to this cause, and that the case was really a primary splenomegaly complicated by bilharzial infection. Since 60 per cent of the males between the ages of 5 and 60 dying in this hospital have this infection in some degree, the coincidence is not remarkable.[9]

This mortality, 18 per cent, seems at first sight a high one. If I had been wise enough at the time to exclude the two advanced cases with ascites, it would, however, have been reduced to 10 per cent, and this, I think, about represents the real risk of the operation, when its application is restricted to cases in the

pre-ascitic stage. Mayo, in his series of splenic anæmias, had 2 deaths in 18 cases, a very similar proportion.

It must be remembered that these patients are always weak and anæmic, and often the subjects of other infections and diseases. When the improvement in the successful cases is considered, the risk is not excessive, and with proper selection of cases will probably be slightly reduced. In our last 11 cases there has been 1 death.

The mortality for splenectomies up to 1908 given by Johnston,[3] is 27·4 per cent, and for 61 cases of Banti's disease in the same period, 19·5 per cent. I have already given reasons for thinking that the mortality deduced from published cases is unduly low. In the cases quoted by Armstrong[1] (32 cases, 9 deaths=28 per cent), the cause of death seems usually to have been hæmorrhage and its resultant shock. This risk, always present, can be largely reduced by a careful technique. It has not contributed to the mortality of this series, and the same is true of sepsis.

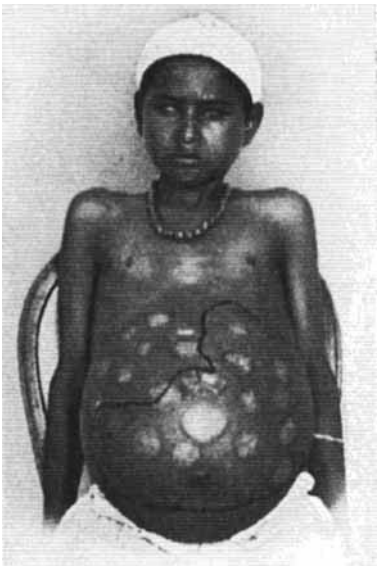


FIG. 273.—*Case* 1.—This was the first case attempted, and the operation had to be stopped owing to heart failure. The patient recovered, and died at home about six months later. The marks on the abdomen are cautery scars, and represent the native treatment of chronic pain.

Incomplete Operations (*Cases* 1, 9).—In *Case* 1, a girl with an enormous spleen (*Fig.* 273), the heart stopped as soon as I endeavoured to dislocate the lower pole of the spleen. It was induced to begin again by massage through the diaphragm; the abdomen was closed, and the girl made a good recovery, and died at home six months later. Considering her condition, I doubt if death was hastened by operation.

Case 9 had such universal adhesions that removal was found to be quite impossible; the wound was closed, and the patient left hospital a fortnight later. These cases call for no comment.

Successful Cases.—The senumber 18, and were all successful in the sense that the patients left hospital in better condition than they entered it.

As regards their ultimate success, they have all been operated on since March, 1911, so that many of them have only been done a short time. Moreover, it is difficult to keep track of poor patients who cannot read or write, who have no surnames, and many of whom come from remote villages. However, by the aid of the police and backsheesh, two great forces in Egypt, several of these have been induced to come up for inspection once or twice, and four were shown to the local medical society last April.

Six of the first 10 have been seen at intervals varying from two and a half years downwards, and the results have been very satisfactory.

Case 2 was seen eleven months after operation; he was well, and his red blood corpuscles had risen from 1,808,000 to 3,680,000.

Case 4 was seen twenty-three months after operation, and was very well and grateful, living an active life (*Fig.* 274).

Case 5 was seen eight months, and again two and a half years after operation. He had gained 19 lb., or, since the spleen weighed 4 lb., 23 lb. in all. He is now doing full work as a field labourer, which he had been unable to do for some years before operation.

Case 6 was seen a year and nine months after operation, and was doing a fair amount of work, about half a full day; he looked well and cheerful.

Case 14, who had been ill for two years before operation, and

Case 15, who had been under treatment for the same time (including injection of salvarsan) were both shown in good condition nine months later.

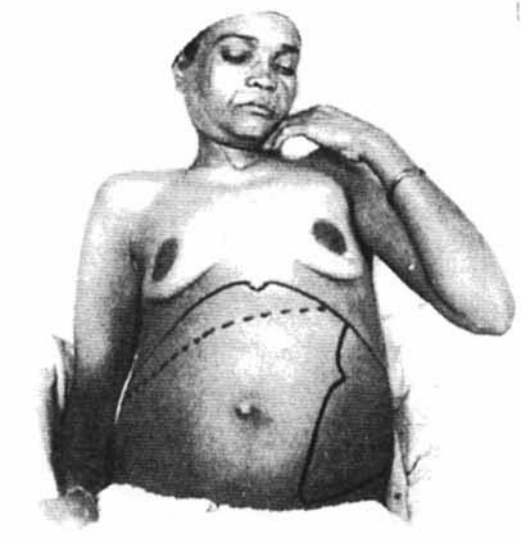


FIG. 274.—*Case* 4.—This woman made a good recovery, and was shown well and active at the end of nearly two years.

It is as yet too early to say much about the later cases, but one of them, *Case* 20, is worth mentioning. She was a girl, aged 15, with a spleen weighing over 3 lb., had been ill for more than two years, and had so much pain after food that she used to fast in order to avoid it. She made a rapid recovery, engaged herself as ward-maid in the hospital, and earned her pay at this fairly heavy work till she left after four months in good condition. She is marked in the matron's register as "a good worker."

These results may be summarized as follows:—

Of the first 10 patients who survived the operation, one (*Case* 3) was found to have died at home five months after the operation. There is no information as to how he died; three others (*Cases* 10, 12, 13) could not be traced, dead or alive; six others (*Cases* 2, 4, 5, 6, 14, 15) were all well at periods varying from two and a half years to nine months after operation, and several of them doing hard physical work.

I think it is quite clear that splenectomy in these cases produces a degree of improvement amounting in favourable cases to a cure. And this is quite in accordance with the theory that the disease is a progressive cirrhosis of the liver, due mainly to the presence of a focus of infection in a diseased spleen. On this view the spleen is in fact removed to save the liver. Of course, no operation whatever will give the patient a new liver, which is what he wants to make him a healthy man. But if a patient gets rid of the complaint for which

he comes to hospital, returns to active life, and earns full pay, he may be considered, from a practical or economic point of view, as cured. It is in this sense only that operation is claimed to cure some of these cases. As to the spleen, it has probably ceased to have any useful function long before it comes to operation (*see Figs.* 269, 270).

Diagnosis.—Diagnosis in this country is relatively easy. The clinical picture is a common and definite one. Alcoholic cirrhosis is practically unknown among a Moslem population. Bilharzial cirrhosis is very rare; it only occurs in 4 per cent of all cases with marked infection; moreover, it does not as a rule cause much ascites, and never any great splenic enlargement (Ferguson). Malaria is also very rare, and is excluded by a blood examination such as has been made in all these cases, and which also serves to exclude leukæmia. Though syphilis is common enough, syphilitic cirrhosis is rare, and salvarsan does these patients no good. Repeated examination of the spleen pulp has not revealed the Leishman-Donovan body in any case. Therefore, if cirrhosis, splenomegaly, and a low leucocyte count are present together, I think the chances of a wrong diagnosis are very small. Anæmia is always present, but it is so common here from bilharzia and ankylostoma, that it is of very little diagnostic value.

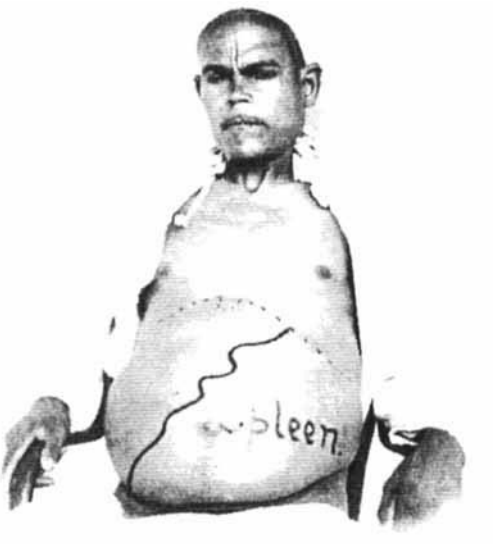


FIG. 275.—*Case* 6.—This patient was shown a year and nine months after operation. He was doing work in the fields.

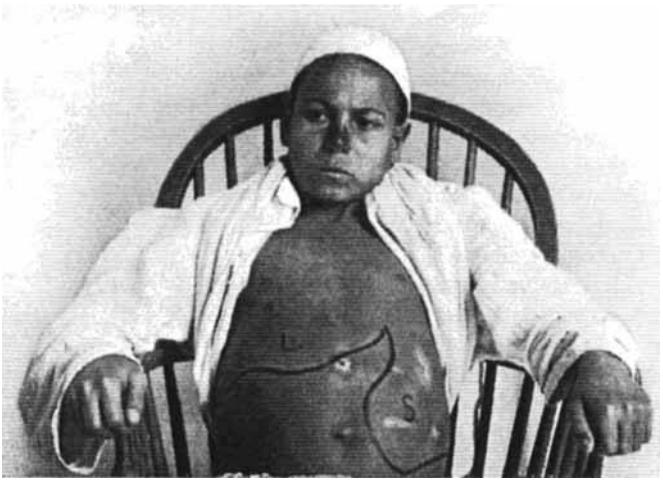
Selection of Cases.—The first question which arises is, what cases of this disease should be operated on. In deciding this, the state of the liver is the one essential factor. The size of the spleen is of relatively little importance, though of course, the larger the spleen the greater the relief given to the patient by its removal. What kills the patient is not splenomegaly or anæmia, but cirrhosis.

Obviously mild and early cases should always be allowed the chance of spontaneous recovery, and anyhow they seldom apply for relief. On the other hand, the very advanced cases, with ascites and emaciation, are in my opinion unfit for operation. Removal of the spleen cannot cure a cirrhosis which is already so advanced as to be visibly killing the patient, while the shock of the operation and the poison of the anæsthetic are only too likely to hasten the end. Two of my deaths were due to this mistake, and I feel sure that it is a sound rule

never to operate in the presence of ascites. One must draw a line somewhere, if only for teaching purposes, and the occurrence of ascites is a definite landmark in the progress of the vitally important part of the disease, namely, the cirrhosis.

I have described elsewhere[8] my attempts at dealing with the ascites itself by Talma-Morrison operations and Paterson's button ; since they were uniformly unsuccessful, they need not be referred to again. These cases can be relieved by repeated tappings ; in some, the ascites diminishes or disappears in hospital ; but the improvement is usually only temporary, and they die, as a rule, in about six months (Day).

Broadly, one may say that the cases most suited for operation are those with big spleens and big livers, in which the disease is severe and progressive, but which have not yet reached the stage of ascites.



[FIG. 276.—*Case* 10.—Weight of spleen about 5 lb. Recovery. Not traced.

Preparation of the Patient.—It is essential to give these patients a rest in hospital in order to establish the diagnosis, discover and treat ankylostoma, and improve the anæmia by drugs and food. Nearly all the cases have been for varying times under the care of Dr. Day before coming to my section, and their general condition has been much improved. *Case* 13, weighing 3 st. 8 lb., gained 4½ lb. in the month which he spent in the medical wards, and was practically converted from an inoperable case into an operable one. He was in any case on the border-line, as he had a trace of ascites, and the successful result was mainly due to his period of preparation, and especially to the expulsion of his ankylostoma worms.

OPERATION.

Surgeons of wide experience in abdominal work may find a detailed description of the technique superfluous, but as these large spleens are rare in England, I think it is worth while to discuss the methods Dr. Aly Bey and I

have found most useful, in the hope that they may be of help to some surgeon who is called on to deal with a case of this kind for the first time.

The difficulties met with differ widely from those in cases of normal or slightly enlarged spleen, without adhesions, which form the basis of the text-book descriptions, and none of these remarks apply to such cases, of which I have no experience.

As regards the incision, a vertical one is the best, starting from a point half-way between the costal angle and the lower border of the thorax, about two or three inches from the middle line. It descends vertically downwards, splitting the left rectus muscle for six or eight inches.

If the incision is placed in the middle line there is not good access to the outer side of the spleen, and it is difficult to bring it out of the incision; if, on the other hand, it is placed further out, the costal margin prevents its being extended high enough to give access to the vault of the diaphragm, where much of the difficulty from adhesions is likely to occur. For the same reason, even if the enlarged liver descends low, as it often does, the incision must still be carried right up to the rib cartilages. A transverse incision added to this is practically never necessary; I have used it only once.

The left rectus is often separated from its fellow, and spread out over the tumour, so that its relation to the incision varies.

The fibres of the muscle are not divided, but its nerves are, and this leads later to a degeneration of the strip lying median to the incision. In *Case* 2, seen eleven months later, this was marked, but the case was one of a small boy, aged 12, and his spleen weighed 4½ lb., so that the incision was necessarily long in proportion to the boy. There was, however, no hernia, even in him, and in the other cases the weakening was not marked, and led to no visible bulging.

Theoretically, this division of nerves is regrettable. But the paralyzed strip is covered by intact sheath, and lies in the upper part of an abdomen which is lax after removal of the tumour, so that practically no harm results from it, even in the cases which do hard work in the fields.

After exposure of the spleen, the first difficulty is that of adhesions. These follow no rule: they may be negligible, or they may bind the spleen so closely to parietal peritoneum, omentum, diaphragm, stomach, and small and large intestine, that removal, as in *Case* 9, is rendered quite impossible. Most difficulty is met with high up under the diaphragm, where it is impossible to do anything except work in the dark. In *Case* 8 the pedicle had first to be divided and the spleen swung right out, in order to deal with a dense area of adhesion beneath the lower ribs.

As a rule, however, the adhesions can be torn down or divided with scissors without much difficulty. The adhesions themselves bleed only moderately; if there is much blood, it generally comes from a tear in the capsule of the spleen, and the only way of stopping this is to go on and turn the spleen out as quickly as possible. When the outer surface and upper pole are free, the rest is fairly easy. The adhesions at the vertebral border behind are never dense, the lower pole can be pulled up and dealt with in the light, and the same is fortunately the case with the more important adhesions to the stomach and other viscera.

Practically, the bleeding from adhesions is not profuse or important. It soon stops when the spleen is removed, and never requires drainage. The difficulty is rather the mechanical one of getting the spleen free. A spleen without adhesions can be removed in half an hour; an adherent one may take over an hour.

When all the adhesions have been dealt with, the lower pole is pulled out of the wound. There is a constant leash of big vessels running down to a sort of secondary hilum near the lower pole, and carrying with them a fold of peritoneum from the pedicle. These vessels are divided between ligatures, and the peritoneum stripped up to the pedicle proper. The spleen is then only attached by the pedicle, and the next step is to bring it outside the abdomen. To do this the assistant pulls out the lower pole, and the operator passes his right hand up between the spleen and the ribs till he can grasp the upper pole in his palm, while with his left hand he depresses the outer lip of the wound. Then, by squeezing, pulling, and pressure, the spleen can be levered gradually out. It is bigger than the incision, but is elastic, slippery, and capable of rotation. It is important to prevent its coming out with too much of a jerk, and once out it must be supported by a dresser to prevent undue strain on the pedicle.

The remaining step is to ligature and divide the pedicle, and this, to judge from the reported cases of death from hæmorrhage, is the crux of the operation.

The difficulty is that the vessels are large, with thin walls, and since the spleen is an elastic bag full of blood under pressure, they are ready to bleed furiously from either end or both. Bleeding from the splenic end is no loss to the patient after the artery has been tied, but it is messy and demoralizing, for in a very big spleen it may amount to about a quart (*see* p. 431). To avoid this, it is necessary to secure both ends of the divided vessels. For this purpose forceps are unsatisfactory; they retract into a dark cavity after division of the vessels, and it is difficult to pull them up for ligature afterwards without putting undue strain on thin vessels lying in loose connective tissue. Moreover, they are very much in the way, especially as they have to be big in order to clip securely large vessels like the splenic vein.

Many good surgeons use big clamps which grasp the whole pedicle, and apply ligatures to the individual vessels either proximal or distal to these. I have tried this method and do not like it. It gives apparent security, but the clamp is very much in the way on a short pedicle, and flattens out the tissues so that it is harder to tie the vessels securely.

For the reasons given above, double ligature is advisable, and a clamp makes this much more difficult of application.

The method which Dr. Aly Bey and I have found the best, is to isolate the vessels successively and divide them between ligatures. Two pairs of forceps are first applied to the upper and lower edges of the central end of the pedicle respectively, so as to enable it to be pulled up readily for inspection afterwards.

The mass is then dissected, chiefly with closed forceps and gauze, with a little help from the knife. It consists of connective tissue and fat, with occasionally lymphatic glands or the tail end of the pancreas, and often some splenculi. The smaller vessels running to the stomach lie in front; the splenic artery, as a white tortuous trunk, lies deeply near the upper border, and the mass of the central part consists of veins joining to form the splenic vein.

As each vessel or small cluster of vessels is isolated, it is cleared for an inch or more, and a loop of stout silk passed under it. The loop is cut, and the resulting two ligatures are pulled in opposite directions and tied as far apart as possible. The vessels included are then cut nearer the splenic end, so as to leave a longer stump centrally.

In this way, with about half a dozen ligatures or less, all the main vessels are tied separately. The spleen is then taken away. The stump is pulled up by the forceps for inspection; it consists of a long narrow strip of raw surface, chiefly fat, with the ends of the vessels lying at intervals along it. If broad, it can be overrun with a fine continuous suture of catgut, care being taken not to prick the vessels, while any opening into the lesser sac is closed in the same way.

Pedicles differ in density, and it is sometimes impossible to get the vessels quite clean. Usually it is easy enough; but if not, the pedicle must be tied in as many small parcels as possible.

This method seems to me neater, safer, and more surgical than any which draws the whole pedicle up into a bunch, and leaves a dead stump distal to the ligatures. It also minimizes bleeding, and I have seen Dr. Aly Bey remove a large non-adherent spleen with a loss, exclusive of abdominal incision, of about two teaspoonfuls. The spleens thus removed are full of blood under pressure. The cavity left is then swept clear of clots if any have formed from the oozing of adhesions, and closed without drainage.

Shock is usually slight, if no blood has been lost, and recovery rapid. A number of detailed accounts of operations will be found in my previous paper,[8] and are fairly typical of the rest; there is therefore no need to add to their number.

The Spleens Removed.—The spleens ranged in weight from 2780 grams (over 6 lb.) to 535 grams (a little over 1 lb.), with an average weight of 1350 grams (3 lb.). The normal weight of the spleen is 170 grams, and consequently the largest spleen was sixteen times the normal weight, the smallest more than three times, and the average eight times.

These weights were taken immediately after removal. The spleens removed by the method described above were nearly all full of blood under pressure, and when the vessels were opened after removal, they discharged blood up to the amount of some 40 per cent of the total weight in the course of a few minutes. With a view to saving some of this blood, I once tried ligaturing the splenic artery at the beginning of the operation, but the loss in this case was also just over 40 per cent. After all, the blood in the rest of the body is not diminished by removing any organ with its contained blood, so the point is not very important.

In appearance, these spleens had the normal shape, with patches of fibrous thickening in the capsule, and the remains of adhesions. Their structure is shown in *Figs.* 269, 270.

After-treatment.—This is the same as in any abdominal operation. It is an advantage to keep the patient propped up.

SERIES OF TWENTY-FOUR CASES, IN TWENTY-TWO OF WHICH SPLENECTOMY WAS PERFORMED.

Case	Sex	Age	Date of Operation	Notes	Operator	Weight of Spleen	Results
1	F	15	Aug. 22 1910	Ill two years	O. R.		Operation not completed owing to heart failure. Recovery. Death at home 6 months later (*Fig.* 273)
2	M	12	March 6 1911	Ill three years	O. R.	2040 gr (4½ lb.)	Recovery: pneumonia: well 11 months later. Rise of R.B.C. from 1,808,000 to 3,680,000
3	M	22	April 4 1911	Bad bilharzia	O. R.	1130 gr.	Recovery: died at home 5 months later. Cause unknown
4	F	25	May 9 1911	Bronchitic	O. R.	1340 gr.	Acute bronchitis; recovery, shown in good health 2 years later (*Fig.* 274)
5	M	25	May 30 1911	Unable to work three years	O. R.	1810 gr.	Recovery: 5 months later had gained 23 lb. and was doing full work. Seen again well, after 2½ years
6	M	18	July 25 1911	Ill four years	A. B.	1340 gr.	Recovery: 5 months later was doing half work. Seen again well, after 1¾ years, in good condition (*Fig.* 275)
7	M	20	Sept. 5 1911	Ascites tapped, seven litres	O. R.	Not noted	Died comatose third day. Œdema of brain. No abdominal hæmorrhage or peritonitis
8	M	14	Nov. 7 1911	Ill two years	O. R.	1250 gr.	Died of acute dilatation of the stomach (*Fig.* 272)
9	M	50	Aug. 3 1911	—	A. B.	—	Operation abandoned owing to density of adhesions
10	M	16	Feb. 27 1912	Ill seven years	A. B.	2200 gr.	Recovery. Acute bronchitis, discharged well in 3 weeks. Cannot be traced (*Fig.* 276)
11	M	15	March 19 1912	Jaundice. Ascites tapped, six litres	O. R.	2780 gr. (6 lb.)	Death on the 18th day from portal thrombosis (*Fig.* 271)
12	M	28	April 4 1912	Pain after food four months	O. R.	1585 gr.	Recovery. Erysipelas. Not traced
13	M	7	May 21 1912	Ill all his life, emaciated, 3st 8	A. B.	1675 gr.	Recovery. Not traced
14	M	16	July 2 1912	Ill two years	A. B.		Recovery. Well 9 months later
15	M	12	July 23 1912	Treated two years, 606 etc.	O. R.	950 gr.	Recovery. Well 9 months later
16	M	20	Aug. 27 1912		O. R.	1200 gr.	Died. Extensive bilharziasis, bilharzial cirrhosis
17	M	15	May 13 1913	Two years fever etc.	O. R.	855 gr.	Recovery
18	M	15	May 18 1913	Ill over one year	O. R.	725 gr.	Recovery
19	M	30	May 22 1913	Ill over one year	O. R.	2258 gr. (5 lb.)	Recovery
20	F	15	June 5 1913	Splenomegaly over two years. Pain after food	O. R.	1528 gr.	Recovery. Well after 4 months and earning full wages
21	M	30	June 17 1913	Bronchitis	O. R.	983 gr.	Recovery
22	F	14	June 26 1913	Ill three years	O. R.	965 gr.	Recovery. Broncho-pneumonia
23	M	14	Aug. 5 1913	Pain one year	A. B.	535 gr.	Recovery
24	M	30	Oct. 21 1913	Ill five or six years	O. R.	1200 gr.	Recovery

Complications.—The only complication of any frequency is bronchitis or pneumonia, which occurred in five cases. This might be attributed to paralysis of the vault of the diaphragm if it always occurred on the left side, but it does not. Probably the fact is that the patients have, before operation, some trouble in the lung due to abdominal distention, and the exposure and shock of the operation lights it up. It has never proved fatal, but easily might have. The effect on the leucocyte count is to send it up very rapidly. The rarer complications are noted under the fatal cases.

CONCLUSIONS.

1. A disease exists in Egypt, of which the essential features are progressive cirrhosis of the liver, enlargement of the spleen, and anæmia.

2. This disease closely resembles that described in the literature as Banti's disease, but it is associated with fever, is probably due to an infection, and the hepatic cirrhosis, instead of being a terminal stage of a "splenic anæmia," is an essential part of the disease from its commencement.

3. Slight cases may recover; more severe cases end in death from hepatic cirrhosis with ascites and emaciation.

4. Cases which have progressed to the stage of ascites are unfit for operation; they usually die in about six months.

5. Severe cases which have not yet reached the stage of ascites may be much benefited, and sometimes cured, by removal of the spleen.

6. The mortality of this operation in such cases is about 10 per cent.

7. The chief operative risk is hæmorrhage, and this may be best met by a systematic division of individual vessels between ligatures.

REFERENCES.

[1] ARMSTRONG, Splenectomy and Banti's disease, *Brit. Med. Jour.*, 1906, ii, 1273.
[2] TORRANCE, Splenectomy in Banti's disease, *Ann. Surg.* 1908, 1, 41.
[3] JOHNSTON, Splenectomy, *Ann. Surg.*, 1908, ii, 50.
[4] MAYO, Surgery of the Spleen, *Jour. Surg. Gyn. and Obst.*, 1913, 233.
[5] WILSON, The Pathology of Splenomegaly, *Ibid.*, 240.
[6] GIFFIN, Clinical Observations concerning twenty-seven Cases of Splenectomy, *Amer. Jour. Med. Sci.*, 1913, No. 6, 781.
[7] DAY AND FERGUSON. An Account of a Form of Splenomegaly with Cirrhosis, Endemic in Egypt, *Ann. Trop. Med. and Parasit.*, 1909, Nov. No. 3.
[8] RICHARDS AND DAY, Egyptian Splenomegaly and its Surgical Treatment, *Trans. Soc. Trop. Med. and Hygiene*, 1912, No. 8, 333.
[9] FERGUSON, The Lesions of Bilharzial Disease, *Glasg. Med. Jour.*, 1913, Jan.

Some other Papers of interest;—

BLAND-SUTTON, Observations on the Surgery of the Spleen, *Brit. Jour. Surg.*, 1913, Oct.
SUTHERLAND AND BURGHARD, The Treatment of Splenic Anæmia by Splenectomy, *Lancet*, 1910, ii, 1819.
OSLER, ROLLESTON, etc., Discussion on Splenic Enlargements other than Leukæmic, *Brit. Med. Jour.*, 1908, ii, 1131.
SUMMERS, Splenectomy in Banti's disease, *Ann. Surg.*, 1908, i, 1006.
CLIFFORD ALLBUT, *System of Medicine*, v, 777 *et passim*.