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DISCUSSION.—DR. LON GROVE, Atlanta: I have before me a summary of a personal experience in 52 consecutive splenectomies, covering a period of 20 years from 1927 to 1948. Four of these were done for trauma and will not be discussed.

Our results in the remaining 48 cases parallel very closely the experience related by Doctor Cole. I might say here that we are thoroughly in accord with what Doctor Cole said about the absolute necessity of having a hematologist of experience associated with you on these cases. I do not see how anyone could approach this type of surgery without the help of a really experienced hematologist. All these cases have been studied by a hematologist of experience, most of them by Dr. Roy Kracke.

Twenty of this series were diagnosed as familial jaundice but now, in the light of subsequent follow-up, we should change the diagnosis in one patient to acquired jaundice, because she has not done well.

In the purpuras, 13 were diagnosed as idiopathic or primary purpura and one as secondary, and we would like to give in some detail the history of this latter patient. This case of secondary purpura was under observation for a long time. Even though she showed all the clinical and laboratory findings that would fit a primary purpura, including repeated bone marrow studies; with a history of two sisters and one brother having died with the disease, we were very hesitant to classify her as a primary purpura. She responded to no form of conservative treatment and finally was splenectomized. She did well for a time, but returned in nine months with all the symptoms she had shown primarily, and died from a subdural hemorrhage.

There have been nine patients with Banti's syndrome, two of whom we have lost track of. Of the remaining seven, we know five have had subsequent hemorrhages and, of that five, three have died. It is interesting that one of these patients went nine years before he had his first hemorrhage; this occurred approximately a year and a half ago and he has not hemorrhaged since. This is a poor showing in Banti's syndrome. As you see from the age incidence most of these patients were children; the oldest was aged 28 years and the youngest 7 weeks. We believe that most of them were diagnosed early but, notwithstanding, they have not done well.

The patient with tumor-fibroangioma was reported before this Association at Biloxi in 1936. At that time she was four months of age. She has developed normally, has shown no evidence of metastases, and I believe we are justified in classifying her as a true fibroangioma.

There was one case with so-called primary tuberculosis with no demonstrable evidence of a primary lesion. She was operated upon because of the enormous size of the spleen. She made a good convalescence and did well for two years, then declined rapidly with evidence of miliary tuberculosis and died.

In the pancytopenia purpuras our experience has been the same as Doctor Cole's. One patient died after six months; one died after five years, in another hospital; we were unable to get an autopsy but were informed that the diagnosis was atypical leukemia. The third patient still shows some evidence of anemia with occasional hemorrhage from the gastrointestinal tract. We have had difficulty in evaluating this case, however, because she has a severe radiation proctitis from which she bleeds, and it has been difficult to determine the source of her anemia. She is still living after approximately a year.

As to technic: In adults we have preferred the subcostal incision, while in children we definitely prefer the left rectus incision, as we think it gives adequate exposure. In this group of 48 patients who had splenectomies there were no hospital deaths.

DR. G. T. MCCUTCHEN, Columbia, S. C.: I would like to call attention to one fact: I believe internists and hematologists make a distinction between primary splenic disease associated with neutropenia and so-called Felty's syndrome. There has been a very limited number of true Felty's syndrome reported. True Felty's syndrome is characterized by the triad of neutropenia, arthritis and splenomegaly. Recently I was embarrassed by being ignorant on the subject. I saw a patient in consultation with an internist; this woman was 80 years of age and, about eight months before, I had opened a huge abscess on her shoulder. I knew at the time that she had neutropenia but it did not occur to me that she might have Felty's syndrome although she complained of her joints also. The internist had observed her over a long period in an effort to detect an enlarged spleen but could not do so. About three months ago he did find an enlarged spleen, and at that time splenectomy was done. She sailed through it without any trouble and has had no recurrence of boils, her arthritis is better, or improved—apparently sometimes it is more or less permanent—but the neutropenia, which had been persistent for at least a year of observation, within 48 hours returned to normal and has remained so.

I would like to make the distinction that true Felty's syndrome is, I believe, not splenomegaly with neutropenia, but is splenomegaly with neutropenia and arthritis. That is the description given by Felty.

DR. FRANK H. LAHEY, Boston: The mortality of splenic diseases for which splenectomy is done will be controlled much more largely by the hematologist than by the surgeon. The mortality will depend upon how well the cases are selected by the hematologists. The more they deal with these cases the better will the hematologists become in aiding in their selection. The mortality in splenectomies for diseases of the spleen will occur in patients who are in the late stages of Banti's disease in which the operation was probably never indicated, in the cases of hemolytic anemia of the acquired variety, in thrombocytopenias which are not truly idiopathic thrombocytopenias, and in those cases even of idiopathic thrombocytopenia which have been permitted to progress into acute states. The mortality will also be in those cases of secondary panhematocytopenias in which the spleen has become so large and the patient has progressed into such a state of depletion that any operative procedure will have an associated high degree of risk.

We have just reported in the ANNALS OF SURGERY 83 splenectomies with fatality in only two cases. Both deaths were in patients with late congestive splenomegaly for which the operation probably should never have been done, and occurred before we knew enough about these states to avoid doing operative procedures.

One thing that our experience has taught us is that once the decision is made by the hematologist that the patient with hemolytic anemia, primary neutropenia, idiopathic thrombocytopenia, panhematocytopenia or congestive splenomegaly, is a suitable case for splenectomy, the sooner the operation is performed the better. If one delays, any one of these patients can go into an acute phase of the disease so that the opportunity of doing a splenectomy with almost no risk is lost, and the situation is then approached with a high rate of risk.

Another point I wish to make in connection with our own experience is that hematologists will have to take the responsibility for the very painstaking and intricate determinations as to whether the lesion is really of primary origin or secondary to some other cause for which splenectomy would not in any way be helpful.

I do not know of anything that is more interesting in all these splenic states than secondary panhematocytopenia. It is extremely interesting that anything producing enlargement of the spleen can bring about this panhematocytopenic effect and produce the three common blood changes that one sees in relation to splenic effects upon bone marrow or abnormal red cells; that is, hemolytic anemia, idiopathic thrombocytopenia or secondary neutropenia. It is extremely interesting to realize that even the enlargement which goes with congestive splenomegaly, the tumor infiltration that one sees with Hodgkin's disease, or the enlargement as a result of the infiltration which one sees with Gaucher's disease, can produce these blood abnormalities which individually can occur as abnormal blood states; but in this state, panhematocytopenic effects can all occur in one picture. We do not cure Hodgkin's disease or Gaucher's disease by splenectomy, but we can, temporarily at least, make these patients much better, not only by the removal of the large spleen and its pressure effects but also by doing away with these secondary blood changes which come with these panhematocytopenic effects of enlargement of the spleen.

Regarding Banti's disease, we feel that we may have been hasty in the past in condemning splenectomy entirely. Our only fatalities in splenectomy for congestive splenomegaly have been in those done unwisely in the third stages of the disease. In the early stages we believe that splenectomy still has real value, unless one feels that immediate shunt should be done.

There is one other thing we have said in a paper recently published on this subject, which is a little dangerous to state, but I believe it is sound. When one has a patient with splenomegaly of unknown origin, provided there can be no demonstrated cause for the splenomegaly by a competent hematologist, and it does not occur in a geographic region where malaria and other such diseases are prevalent, then the mortality of splenectomy is so low that we believe splenectomy is indicated. It has been shown that people exist well and without difficulty after splenectomy, and such is the hazard of later changes in blood states due to a large spleen, that we believe, if adequate blood studies have been done and nothing has been found, and the patient is a good risk, splenectomy is justifiable.

DR. FRANK WILSON, Birmingham: In the last two years I have had five cases of splenectomy in children at the Children's Hospital in Birmingham. Their ages were four years, two years, 16 months, 15 months and, the youngest, eight weeks. Our experience has been very similar to that of Doctor Cole, in that the hematologist is responsible, to a great extent, for the correct diagnosis in these cases.

One of the cases was a thrombocytopenic purpura, three were familial hemolytic jaundice, and one was diagnosed as acquired hemolytic jaundice. This child was the oldest in the group and at operation the spleen, approximately twice normal size, was removed along with two accessory spleens about 2 centimeters in diameter. All the

children except the last one have been entirely relieved of symptoms. This child has continued to require transfusions with the intervals getting farther and farther apart. We have thought that probably she has another accessory spleen which was not found at the time of operation.

There were two sisters with familial hemolytic jaundice, the older 16 months and the younger eight weeks of age. Their father had his spleen removed at the age of 13 and an aunt also had a splenectomy. The older child's symptoms appeared very early, but as her father was a paratrooper in Germany we waited for his return home before operating on her at 16 months of age. After the father returned, the mother soon became pregnant and the second child, a girl, was born. She obviously had hemolytic jaundice, which was diagnosed soon after birth, and we were unable to keep her blood count up without a transfusion at least once a week.

The mother was much concerned because of the economic phase and the cost of transfusions, and she wanted to know whether or not we could operate on this child as soon as possible. We had never done a splenectomy on a child so young, but we decided to operate on her at the age of eight weeks. The mother is again pregnant and is most anxious to know what can be done for her child and how soon the spleen can be taken out, provided the child is born with familial hemolytic jaundice.

These children upon whom we have operated have all made an uneventful postoperative recovery with no postoperative complications.

We have taken all these spleens out through a subcostal incision, as described by Doctor Cole, with the left shoulder slightly elevated, and I think it makes the operation a great deal less difficult than with a right rectus incision.

DR. ROBERT S. SPARKMAN, Dallas: I wish to make a few remarks concerning Gaucher's disease, which Doctor Cole has listed as an occasional indication for splenectomy. Our interest in this condition has been stimulated by the fact that we have four cases of Gaucher's disease under observation at the present time. There are perhaps three very good reasons why Gaucher's disease may constitute a specific indication for splenectomy. The first of these, as has been pointed out by Doctor Lahey, is the fact that patients with Gaucher's disease regularly demonstrate either panhematopenia or one of its components, of which neutropenia is particularly frequent. In fact, in Doan's original report of splenic panhematopenia, one case was in a child with Gaucher's disease. The second reason is the immense size the spleen can attain; one case is reported in which the spleen weighed 7200 grams. This slide shows the size of a spleen in a 5-year-old boy with Gaucher's disease. The third reason is the fact that frequently in children growth becomes completely retarded, to be restored after removal of the spleen.

I offer these as fairly consistent indications for splenectomy in this disease.

DR. ARTHUR H. BLAKEMORE, New York: In cases presenting the clinical picture of Banti's syndrome, the surgeon should suspect splenomegaly of the congestive type, and be prepared to locate the site of the obstruction in the portal system. For upon this will depend the correct surgical therapy in a given case of Banti's syndrome.

I make a strong plea that those surgeons interested in splenectomy have a simple venous pressure apparatus boiled up with their instruments in each case. It takes only a few minutes to cannulate an easily accessible, known radicle of the portal vein (e.g., the gastroepiploic) and take a portal pressure reading. If a correct pressure reading thus taken is 150 mm of water or more, it means that the site of portal block is either in the portal vein itself or in the liver. In either case there is portal hypertension not only in the splenic vein but in all other portal radicles as well.

This finding of generalized portal hypertension in association with congestive splenomegaly is by all odds the common finding in cases of Banti's disease. Splenectomy alone will not cure such cases of a tendency to have recurring hemorrhages, whether

they be considered "early" or "late" cases. Our experience at the Spleen Clinic of Presbyterian Hospital, over its nearly 30 years existence, suggests that if a so-called early case of generalized portal hypertension has not had a recurrence of hemorrhage following splenectomy, it simply means that there has not been a sufficient follow-up period. We do not contend that there is not the exceedingly rare case in whom, because of an unusual capacity to develop collateral vessels, hemorrhage may be postponed seemingly indefinitely. But we do not consider that one should necessarily credit splenectomy with the entire role in these rare instances.

Much has been said of splenectomy as removing a burden of blood from the already engorged portal system. In an attempt to evaluate this possible factor we have made some portal pressure readings before and after ligation of the arterial supply while doing splenectomies. The results, so far, have varied from zero to 25 mm. (water) change in portal pressure following interruption of arterial blood flow of the spleen. These findings would not seem particularly significant in the usual cases who have portal pressures varying from 300 to 600 mm. of water.

The rational attack upon the problem of portal hypertension is the direct lowering of portal blood pressure by the establishment of portacaval shunts. Our experience with this procedure now embraces 75 operations and we are well pleased with the overall results.

In cases of portal hypertension secondary to intrahepatic portal block (cirrhosis) it is our policy to employ the portal vein to venacava type of portacaval shunt, preferably, as a side-to-side suture anastomosis. This type of portacaval shunt, because of its large size, is capable of returning the portal pressure to normal and, what is even more gratifying, regularly results in complete disappearance of esophageal varices demonstrable by x-ray. We have accomplished this type of operation with a postoperative mortality of 12.5 per cent. This we think is reasonable when one considers that disease of the liver was present in every case.

The splenorenal type of portacaval shunt, preferably as an end-to-side anastomosis of the splenic vein by suture with the left renal vein (Blalock) is decidedly the next most efficient set of vessels to employ in the establishment of portacaval shunts. The truth of the matter is that conservation of the splenic vein for anastomostic purposes is the one best hope of cure for that large group of portal hypertensives having normal livers, in which the seat of portal block is in the portal vein itself, thus precluding its use for shunting purposes.

In view of the above facts, in cases in which careful hematologic and liver chemistry studies would indicate the likelihood of portal hypertension due to extrahepatic portal block, it is to the best interest of the patient that the surgeon be prepared to make confirmatory portal pressure readings at operation. Certainly it is only by recognizing the presence of portal hypertension when confronted with it (one cannot often tell by the appearance of the veins) that we will ever be able to stop adding to that pathetic group of post-splenectomy bleeders—cases whose best hope may lie in the conservation of the splenic vein for shunting purposes. In such cases if, upon exploration, the surgeon be unprepared to proceed with splenorenal anastomosis at the time, in fairness to the best interest of the patient he should close the abdomen and thus conserve the integrity of the splenic vein for subsequent use.

In closing, I wish to discuss the surgical handling of cases which, in our overall series, constitute decidedly a minority group. I refer specifically to cases of congestive splenomegaly secondary to obstruction of the splenic vein. Pathologically, the obstruction is commonly located in the splenic vein at its junction with the superior mesenteric vein, the result, apparently, of congenital atresia. A more sudden onset, particularly in cases giving a history of trauma, suggests thrombosis as the more likely cause of the obstruction.

Of more importance is the ability to distinguish at the operating table two types of cases in this group. For upon this distinction the correct surgical therapy will depend. Differential portal pressure readings are the practical solution of this problem. In the first place, in a case of suspected congestive splenomegaly a normal portal pressure reading,

when taken from a known branch of the superior mesenteric vein, will rule out the liver and the portal vein as the seat of obstruction. Next, if a reading taken from a distal branch of the splenic vein is elevated this confirms the diagnosis of congestive splenomegaly and localizes the site of obstruction to the splenic vein. As previously stated, there are two types of cases which must be differentiated when the splenic vein is the site of obstruction; namely (1) those identified by elevation of pressure in the splenic vein only—cases in which a simple splenectomy will bring about cure; (2) those identified by elevation of pressure both in the splenic vein and in the coronary system of veins—cases in which, to relieve hypertension in the coronary system of veins, a splenorenal shunt is necessary following splenectomy. To identify this second type at the operating table it is only necessary to pull down the stomach, cannulate a known branch of the coronary vein, and take a third venous pressure reading. If the pressure is higher than normal you know at once that the coronary vein joins the splenic vein at a point distal to the obstruction in the splenic vein itself. The reason for the existence of this second type of case is the anatomic frequency (65 per cent) with which the coronary vein joins the splenic vein.

Early in our experience, through venography, we confirmed the existence of these two anatomic types. The importance of these findings was well demonstrated in the case of an 18-year-old girl who had a massive hematemesis three months following a simple splenectomy. A venogram showed a greatly enlarged, tortuous coronary vein joining the splenic vein, in which the latter was obstructed at its junction with the superior mesenteric vein. At the time of splenectomy, had the stump of the splenic vein been anastomosed to the left renal vein, the girl would probably be alive today. It is a fact that branches of the coronary vein inosculate with branches of the esophageal veins and this, most likely, accounted for the hemorrhage in this case.

In cases in which return blood flow is impeded in the coronary system of veins, for anatomic reasons it is not logical that simple splenectomy should be of benefit. The facts are that the entire arterial supply for the coronary system of veins is gastric, not splenic. Decompression of the coronary system by shunting is the logical solution in such cases.