In a Carpenter Lecture, William J. Mayo (Med. Record, 27th October 1917) discusses the relation of the spleen to certain obscure clinical phenomena. He points out that pathology and experimental medicine have contributed very little to our knowledge of the organ, and that what we know and what we think we know about the spleen is largely the result of circumstantial evidence, having its origin in comparative anatomy and physiology, and the results of clinical experiences capable of different interpretations.

The spleen is derived from mesoblastic tissue and is probably concerned largely with filtration of certain substances from the blood, and the product of its activities is delivered to the liver through the splenic vein.

The organ has a very scanty nerve supply from the sympathetic system, but it does contain a very considerable amount of non-striated muscle fibre. Keith has demonstrated that the non-striated muscle has the power of originating contractions independent of nerve supply, and exhibits a most primitive form of control. The spleen has what might be called a beat, which consists of an enlargement following food intake, with a gradual resumption of its normal size after several hours, showing a definite connection with the digestive function. This is further indicated by the fact that its blood-supply is from the celiac axis—the same source as that of the derivatives of the foregut, the stomach, the liver, and the pancreas. The blood entering the spleen comes into direct contact with the splenic pulp, the smaller blood-
vessels having lost their elastic coats. This renders the organ friable and vulnerable to trauma, and also extraordinarily susceptible to the influence of radium.

The enormous blood-supply of the spleen clearly proves it is not a retrogressing organ. It may be surmised the spleen does not possess an internal secretion of importance from its extremely limited sympathetic nerve supply and from the slight metabolic disturbance caused by the removal of the organ. On hypothetical grounds it seems probable that the spleen develops certain enzymes which are important to its function, but it is equally evident that the function of the spleen is shared by other lymphoid and adenoid structures, and that on the removal of the organ the function is continued by these collaborating structures.

In discussing the relations of the spleen to the blood he states that Robertson and Rous (Journ. Exp. Med., 1917, xxv. 651, 664) have come to the conclusion that fragmented erythrocytes are not only removed there but that fragmentation must take place in the spleen.

In some of the anæmias, notably hæmolytic icterus, the enlargement of the spleen may be a work hypertrophy—as suggested by Chauffard and Widal—the fragility of the corpuscles in this condition leading to undue fragmentation and destruction in the spleen.

Relation of the Spleen to the Liver.—Cirrhosis of the liver secondary to splenic disease, if portal, is of the atrophic or hob-nail type, showing that the cause concerns protein, not fat, metabolism. In biliary cirrhosis, on the contrary, the liver is always enlarged. This form is even less well understood than the portal type, and there are various subdivisions, such as the pigmented (hæmochromatosis) and the so-called Hanot’s cirrhosis; the latter designation evidently includes cases of hæmolytic icterus (acholuric jaundice), and serves only still further to confuse the issue.

The enlargement of the spleen which so often accompanies portal cirrhosis suggests that the primary source of these poisons may be in the spleen, and in some cases of portal cirrhosis in which Mayo and his co-workers have removed the enlarged spleen the results, he says, justify the presumption. In Banti’s syndrome the portal cirrhosis is a late stage of splenic anæmia, and even in advanced disease the removal of the spleen often eures, the liver regenerating to a marked degree. In portal cirrhosis supposed to be primary the spleen may be enlarged to a considerable extent without being manifest until a late stage. Sometimes it is a moot question whether the liver or the spleen was first affected. All this goes to show how little is definitely known about the subject.

The biliary cirrhoses, in many cases at least, have their origin in infections in the common duct, associated with gall-stone disease. In such cases the spleen may or may not show great enlargement. In
other cases in which no such infection exists in the common duct the spleen may be found to be very large, and suggests the possibility that it has carried to the liver toxic materials which have safely passed through the portal side but have exercised a large influence on the hepatic cells and the smaller bile ducts. This, Mayo says, we know to be the fact in connection with hemolytic icterus, as the removal of the spleen promptly relieves the jaundice. The enlarged liver thus may not be a true biliary cirrhosis but in part at least an excess of function, causing hepatic hypertrophy as well as a cirrhosis in the attempt to care for the erythrocytic débris destroyed in the spleen and carried to the liver by the portal vein.

Relation of the Spleen to Bacteria and Protozoa.—Mayo states that spirochetal “hibernation” in the spleen is not unusual. Failure to eradicate the disease by salvarsan and prolonged mercurial treatment may result in a syphilitic spleen, which permits not only luetic reinfection of the body but also causes a high grade of chronic anæmia. In four cases of this type, removal of the spleen promptly cured the anæmia, and the lues thereafter quickly responded to renewed treatment. In all of these spleens either spirochetes were found or gummata in spleen or liver were demonstrated, showing again the relation of the spleen to hepatic disease.

Hæmolytic Icterus.—The Mayos have performed splenectomy nineteen times for hæmolytic icterus and the results have been astonishingly good. William J. Mayo says he does not know of an operation giving more gratifying results. The jaundice which the patient has had for perhaps years will be perceptibly less in forty-eight hours, and within four days will have quite disappeared. Sixty per cent. of his patients had complicating gall-stones, apparently due to the greatly thickened bile, the result of pigments derived from the disintegrated erythrocytes. Splenectomy, as a rule, is not difficult in these cases, for although the spleen may be quite large it is seldom adherent to a marked degree. There was one operation death in the series of nineteen cases. This patient was operated on during a crisis, and death probably would not have occurred had the operation been performed in the interval between crises.

Splenic Anæmia and Banti’s Disease.—The gastric hemorrhage of splenic anæmia is a symptom well worthy of attention. It is not different from that which occurs in connection with hepatic cirrhosis, and it is altogether probable that many of the unexplained hæmorrhages from the stomach, in which no local lesion of the gastric mucosa is to be found, are a result of the toxic condition which precedes, accompanies, or is caused by splenic anæmia and cirrhosis of the liver. In gastric hemorrhage we must think of the spleen and the liver as causative factors, just as in the differentiation of the causes of jaundice the spleen must be thought of as well as the liver. The Mayos have removed
the spleen in forty-three cases of splenic anæmia with four deaths. The operation is more difficult and dangerous than in any of the other diseases for which splenectomy is indicated.

As E. Moschcowitz (Journ. Amer. Med. Assoc., vol. lxix. No. 13) points out, the literature on Banti's disease affords a most disjointed impression and leaves little material on which to base an estimate of the validity of Banti's disease as a nosological entity. Practically the only criticism of Banti's views has come from pathologists, and, significantly enough, the general tone of it is one of guarded scepticism. According to Moschcowitz, nobody, not even Banti himself, knows definitely what Banti's disease connotes.

In analysing the clinical and pathologic features of the disease called by his name, we find that Banti has hedged his malady about by so many criteria that it is almost unreasonable to expect any single case to conform to every requirement. These requirements are clinical and pathologic, and may be classified as follows:—(1) The disease must be without known etiology. As soon as a definite cause for the malady is established, the case is no longer regarded as a possible "Banti," but is at once thrown out of court. (2) The blood picture must be typical. (3) There must be three stages, two of which are fairly well defined clinically. (4) The signs of anæmia and splenomegaly must precede the cirrhosis.

Pathologically the data which make a diagnosis of Banti's disease possible are the following:—(1) A splenomegaly of considerable size; (2) a cirrhosis in the liver of the Laennec type; (3) a histologic fibrosis of the spleen arising without the interposition of fibroblasts; (4) a progressive eccentric fibrosis of the Malpighian follicles; and (5) a usual but not constant endophlebitis of the splenic vein.

Banti admits there is nothing pathognomonic in the pathological findings, but no matter how clear the clinical diagnosis of Banti's disease may be, unless the pathological findings are those premised by Banti, the disease is not Banti's disease. Moschcowitz admits that the clinical picture described by Banti is sometimes seen, and that at necropsy the lesions described by Banti are found, but the literature teems with variations from this general type, showing that Banti's disease has most indeterminate outlines. Clinically this is so, and it seems well agreed by everybody, even by Banti himself, that a diagnosis is impossible on the basis of the pathological findings alone.

Moschcowitz's conception of Banti's disease is based on an analysis of published work. He is led to conclude that—(1) All the evidence thus far submitted gives us no right to believe that splenic anæmia and Banti's disease are not identical. (2) There is no reason for differentiating Banti's disease from other splenomegalies associated with anæmia on the ground that in Banti's disease no etiology can be determined. A nosologic distinction based on whether a disease has
a known or unknown etiology has no raison d'être in clinical medicine. Rather, he believes, we should regard Banti's disease as merely a nosologic and clinical entity, which may be due to both known and unknown causes. The known causes are syphilis, alcohol, malaria, trypanosomiasis, persistence of umbilical vein, etc. In all these maladies a splenomegaly, an indurative splenitis, with eventual atrophy of the Malpighian follicles, and an anemia are the predominant features. It may be argued that in the splenomegalies associated with alcohol, malaria, syphilis, etc., the causes of the disease and some of the clinical phenomena differ from those which Banti described. In reply, Moschcowitz answers—(1) Banti's disease has no typical course and diagnostic symptoms; indeed, if the specifications which Banti predicted are strictly adhered to as criteria for making a diagnosis, we would find that Banti's disease is extraordinarily rare. (2) A clinical diagnosis of Banti's disease has been made by most able clinicians in cases that eventually proved to be cirrhosis of the liver, malaria, syphilitic splenomegaly, etc. Moschcowitz cites many instances of this in his communication. Again, it may be argued that the pathology of Banti's disease is quite different from that of the splenomegalies of known origin. But the pathology of Banti's disease is by no means a specific one. The fact that many observers hold that Banti's disease and cirrhosis of the liver are identical shows that this contention is not unjustified. (3) Banti's explanation of the cirrhosis of the liver as secondary to a splenotoxin is wrong. Further, he does not believe that patients who show at necropsy no cirrhosis of the liver should be regarded as affected with a different malady or that such cases should be regarded as instances of Banti's disease in the first stage. It is more logical and would simplify matters considerably to regard these variations as phenomena of one and the same disease; that a fibro-genetic toxin, probably of intestinal origin, attacks the organs draining the portal area, causing primarily a fibrosis of the spleen, and, if the toxin is sufficiently intense or the patient lives a sufficiently long time, causing a cirrhosis of the liver as well. The common association of sclerotic vascular changes in the mesenteric vessels in Banti's disease are most readily explainable on the same grounds.

Finally, in view of the fact that Banti has failed to make out a case for the disease called by his name and has not shown it to be a distinct entity, the term "Banti complex" should be substituted for the term "Banti's disease."

J. E.