Dr. G. A. Sutherland: For a long time the chief interest of splenic enlargement was its diagnostic value in various affections. The spleen was known to become enlarged in connexion with certain local or constitutional diseases, but was not regarded as the primary seat of disease. In his work on "Pediatrics," published in 1906, Dr. Rotch, of Boston, says, "there are no primary diseases of the spleen." If this were so, then an enlargement of that organ cannot be regarded as a possible indication for treatment directed to it. If the term "primary" be emphasized, we may have a difficulty in disproving his statement when interpreted literally but not clinically. In a literal sense it may be said that there are no primary diseases of the appendix, and that all are secondary to some form of infection. In a clinical sense, however, we speak of appendicitis as a primary disease of the appendix, and justify this by the fact that all symptoms of the disease disappear when the appendix has been removed. In the same way, if we find that an enlargement of the spleen is accompanied by certain definite signs of disease, and, if by excision of the spleen these signs are made to disappear and the patient is restored to health, then I take it we are entitled to speak of this condition as a primary disease of the spleen.

At the present time, instead of there being no recognized primary diseases of the spleen it would appear that the number of them is increasing, as judged by the results of splenectomy for the relief or cure of various clinical phenomena. Here, as in other cases, surgical procedures have gone ahead of pathological knowledge, for it is not yet known what the exact nature of the splenic disease has been. This ignorance has naturally hampered the physician in his attempt to find remedial measures. Whatever the disease, it has been clearly established that removal of the spleen means the relief of all symptoms and the cure of the patient.

My own interest in enlargement of the spleen in childhood has
centred chiefly in the condition of "splenic anæmia" or "primary splenomegaly," as it occurs after the age of infancy. The type of disease is the same as that met with in adult life. The clinical phenomena are sufficiently definite to make the disease easily recognizable after the first stages have passed. The first point I have to note is that in my two cases the splenic anæmia was a family affection. A sister of one patient had died at the age of 13 years from splenic anæmia. An uncle of the other patient had suffered for years from splenic anæmia. These facts suggest that in some cases at least there is a defect somewhere in the developmental processes in certain members of these families, which shows itself more especially in connexion with the splenic area.

In both of my cases the progress of the patients was steadily down-hill. There was an increasing degree of anæmia, lethargy and debility. It became clear that in all probability the patients would die if the condition were not relieved. But this is by no means always the case. We may find in the same family members who have grown up to adult life with a condition of splenic anæmia, varying from time to time in the intensity of the symptoms, while others have died young of progressive disease. Some writers lay stress on the prolonged course of the disease, others emphasize the fact that death usually occurs in from six months to four years from the onset of symptoms. This fact points to the conclusion that the severity of the disease depends on the extent and character of the disturbance of splenic function which is present.

Opinions differ as to the nature of the disease or disturbance in the spleen which produces the symptoms of splenic anæmia. Some hold that the spleen is destroying the blood cells by means of some toxin. Others believe that there is an inhibition of blood formation in the spleen. My own experience of splenectomy in such cases points strongly to there being an excessive destruction of blood cells going on in the spleen, and consequent anæmia. In one case, three days after operation the red cells had increased by 2,200,000, and the hæmoglobin by 36 per cent. In the other case, four days after operation the red cells had increased by 2,200,000, and the hæmoglobin by 22 per cent. At the same time the general appearance of the patients was greatly improved, and the other symptoms of the disease were vanishing rapidly. It is difficult to believe that such rapid improvement could have taken place if there was any chronic infection in the system or in
the spleen, or if the spleen had been inhibiting blood formation. It appears to me that the above facts are most easily explained by the assumption that the spleen had been over-active in the destruction of blood cells, while the blood-forming organs had been working at high pressure in order to counteract the loss of cells. Probably it is only in early life when the blood-forming organs are so active that these sudden and striking results will be obtained.

If there is a process of blood destruction going on, what brings this about? Pathological investigators are agreed as to a general hyperplasia of the splenic tissues, but have not been able to determine any other definite change. When one examines the spleen after splenectomy in these cases one striking feature is the amount of blood contained in the organ. When the divided blood-vessels of the pedicle are freed the blood rushes out, and on squeezing the organ it is found to collapse like a sponge, the great size having been due to the contained blood. The splenic vessels are large, and this has usually been ascribed to the call of the spleen for more blood. On the other hand, it is possible that there may be primarily a pathological condition of the splenic vessels, a vasomotor disorder, leading to hyperæmia of the spleen. We find in this affection that there is a marked tendency to gastric hæmorrhage, often at a comparatively early stage, and quite out of proportion to the frequency of hæmorrhages in other parts. This gastric hæmorrhage is usually ascribed to mechanical displacement of vessels, but it is not found in other forms of splenic enlargement with anything like the same frequency. On the other hand, if the size of the splenic artery is pathologically increased the branch to the stomach wall will also probably be similarly affected, and a tendency to gastric hæmorrhage will naturally follow. Even after the removal of the spleen for splenic anæmia gastric hæmorrhage may supervene at a considerable interval of time, as published cases have shown.

An excessive blood supply to an organ may disturb its natural functions. We see this in the case of the thyroid gland in Graves's disease. The natural functions may be stimulated to excess, or they may become perverted. The destruction of red blood cells is usually regarded as one of the functions of the spleen. If this function be stimulated to excess by a superabundant blood supply, the result may be that the balance in the body between blood formation and blood destruction is seriously disturbed, and that the latter predominates. The spleen may be said to be inebriated with the exuberance of its own
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blood supply. If we assume that there is not necessarily any disease in the spleen in this affection, but only a disturbance in certain of its functions from hyperaemia, we shall be able to understand the varying conditions present in family cases. The congenital defect may be referred to the vascular supply of the spleen, and, according to the degree of that defect, we may find some cases with no symptoms, others with only mild symptoms, and others with progressive symptoms leading to death from excessive blood destruction.

The question of the ultimate result of splenectomy for splenic anaemia is a very important one. In the two cases of splenic anaemia under my care, on whom Mr. Burghard performed the operation of splenectomy, the result has been a complete cure; in the one case seven years have elapsed since the operation, and the patient is now aged 20 years; in the other three years have elapsed, and the patient is now aged 9 years. There has been no recurrence of the symptoms, no evidence of any injury from absence of the spleen, and the blood counts have continued normal in all respects. I cannot recall in my experience of surgical proceedings any results which have been so striking and so permanently beneficial as those from splenectomy in splenic anaemia.

Dr. Poynton said that he realized the importance and difficulty of the subject under discussion, and the widespread desire there must be among many of the Fellows to add their part to the discussion. He would accordingly limit his remarks to the group of cases included under *family acholuric jaundice*. He had four families under observation, including twelve individuals, and among them were some of the earliest examples shown in this country, and consequently cases which had been under observation for some years. With but little exception his remarks would deal with clinical points. In passing, he would remark that in three of the four families Wassermann's test had been undertaken, and in each instance had been negative; the fourth group had not been investigated. In all the families the hereditary tendency was apparent, and amongst them the similarity in the appearances of a father and son and a father and daughter were most striking. He believed that he was among the first to point out in this country that the condition of yellowness might alternate with that of anaemia, and that an enlarged spleen with an anaemia so little tinged with a yellow colour as to be easily overlooked might occur in some members of these