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At first glance, the spleen would seem to be a rather useless organ. Its removal in the normal human being or animal is unattended with any noticeable clinical effects. Careful laboratory examinations under such circumstances reveal, however, that the spleen and the blood are closely related. The red cells become thinner and show Howell-Jolly bodies, the leukocytes and the platelets become greatly increased, and the output of urobilinogen in the feces becomes definitely diminished. From these observations it would appear that the spleen normally makes red cells thicker, has effects on the denucleation of normoblasts in the marrow, and in some measure controls the growth and/or delivery of granulocytes from the marrow to the blood. The old adage that the spleen is the “graveyard” of the red cell cannot now pass unchallenged, even though the output of bile pigment in the feces is definitely diminished after splenectomy. How the red cell normally becomes broken down still remains something of a mystery. The rather glib explanation that the reticulo-endothelial system destroys it fails to measure up to careful histologic studies, which show no hint of phagocytosis. Other explanations which invoke a rather mysterious “lysolecithin,” the high lecithin content of the thoracic duct, etc., likewise fail to satisfy. Perhaps the red cell after its many thousand passages through the miles of capillaries and sinusoids, gradually wears out, and becomes hemolyzed in the circulation. The spleen, by tending to make it thicker (i.e., spherocytic), probably helps to wear it out. This purely mechanical function of the spleen is far less intriguing than possible hormonal effects of that organ upon the bone marrow. These are concerned, not only with the red cells, but with the leukocytes and platelets as well.

Thus far, very little direct evidence for the normal existence of splenic hormones has been adduced, although in a recent article, Ungar reports the isolation from the spleen of guinea-pigs of a material in crystalline form which he calls “splenin.” This material was found to reduce the bleeding time, increase the capillary resistance, and inhibit the release of histamine from blood cells. The spleen, as an endocrine organ, is thought by Ungar to play a role in the control of protein metabolism and in certain reactions to stress. These observations require confirmation and amplification; e.g., effects on platelets were apparently not studied.

Normally, the splenic physiology is probably of minor importance, although it is concerned to some extent in the maturation and release of the various cells from the marrow to the blood. This implies the presence of one or several splenic hormones. Under certain abnormal conditions, however, an altered splenic physiology becomes all-important, so much so indeed that the continued presence of that organ may lead to serious disability and even death. This happens for example in idiopathic thrombocytopenic purpura, in many cases of hemolytic anemia, and in not a few of splenic neutropenia. As in many other pathologic states, the behavior of a disordered organ, in this case the spleen, tends to illuminate the rather more subtle activities of the normal spleen. Thus, in Werlhof’s disease, the extreme degree of platelet diminution may be due to the production in excess of a splenic
hormone which controls platelet growth and delivery, i.e., idiopathic thrombocyto- 
topenic purpura may be a form of "hypersplenism." The leukopenia and granulo-
ctopenia of many different kinds of splenomegaly (e.g., cirrhosis of the liver, 
Felty's syndrome, Boeck's sarcoid, malaria, kala-azar, and the more recently 
described "splenic neutropenia") may be due to the excessive production of another 
hormone controlling granulocytes. Certainly the marrow is crowded with granulo-
cytes and yet the circulating blood is greatly depleted of these cells. On the other 
hand, Doan and his group explain this state of affairs by "sequestration" and 
phagocytosis by the spleen. Anemia often occurs with various types of splenomeg-
aly, and certain cases of hemolytic anemia seem to be purely "hypersplenic" in 
type, associated as they are with leukopenia and thrombocytopenia, and respond-
ing dramatically to splenectomy. Abrami and collaborators have recently de-
scribed two types of splenic anemia: a hemolytic form, and one which is purely 
inhibitory in type. The effects of an abnormal spleen upon the marrow may thus 
be selective or "total"; if the latter, pancytopenia or panhematopenia ensues. 
These abnormal states of the spleen, particularly splenic neutropenia and pan-
cytopenia, and the great importance of splenectomy as a life-saving measure in 
these cases are just beginning to be appreciated. Many cases go about unrecognized 
for months or years, often because the enlarged spleen has not even been palpated. 
The pancytopenia is usually thought to be due to aplasia or hypoplasia of the 
marrow, whereas actually the marrow in these states is hyperplastic. Recognition 
of these cases is facilitated by careful studies and by correct interpretations 
of the clinical, blood, and bone-marrow pictures. It is hoped that continued study will 
bring these states of hypersplenism more forcibly to the attention of the medical 
profession.

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REFERENCES

1 SINGER, KARL, MILLER, E. B., AND DAMESHEK, WILLIAM: Hemolytic changes following splenec-
tomy in man, with particular reference to target cells, hemolytic index and lyssolecithin. Am. 
2 ———, AND WEISZ, LEO: The life span of the erythrocyte after splenectomy and the problems 
4 DAMESHEK, WILLIAM, AND MILLER, E. B.: The megakaryocytes in idiopathic thrombocytopenic 
5 DOAN, C. A., AND WRIGHT, CLAUDE-STARR: Primary congenital and secondary acquired splenic 
6 ABRAMI, P., DE GAUDART D'ALLAINES, F., AND DUGAS, J.: Du mécanisme de l'anémie au cours des 
anémies spléniqes de l'adulte. Splénomégalies hémolytiques et splénomégalies myélopréquiales. Le Sang 
16: 213, 1944.
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