Splenectomy in Treatment of Secondary Hypersplenism *

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Indications for splenectomy are being extended gradually to include certain selected cases of hypersplenic cytopenias secondary to other diseases. It is the purpose of this paper to report 118 such cases treated by splenectomy at the Baylor University Medical Center during the 10-year period ending January 1, 1962, and to review similar reports by other authors. These reports indicate that a more aggressive surgical attack is justified in the management of a considerable number of diseases previously consigned to medical treatment alone.

Hypersplenism

The term *Hypersplenism* has become generally accepted to designate an abnormal hematological state brought about by overactivity of the spleen. Essential clinical features, though subject to some variations are 1) splenomegaly (usually); 2) peripheral blood cytopenias; 3) normally cellular or hypercellular bone marrow; and 4) symptoms attending these abnormalities. The term apparently was fabricated by Chauffard,[4] in 1907, to place such diseases in a proper frame of reference. Hypersplenism was discussed by King,[20] in 1914, but was brought into focus first by Damashek,[7] in 1941, then by Doan,[9] in 1942, when these two authors initiated a controversy regarding the concepts of splenic hyperfunction. Evans,[13] in 1951, introduced an auto-immunologic concept. The present status of this complex subject is well summarized in a statement by Rambach and Alt: [22] "The problem of splenic hyperactivity, since the days of Gretzel in 1866 and Banti in 1898, has been plagued by uncertainty of meaning, doubt of etiology, and capriciousness of therapeutic results."

Nevertheless, important advances have been made, both in understanding of the hypersplenic states and in their treatment. Diagnostic measures and medical therapy have improved, especially in regard to the use of steroids, but in many cases, as stated by Doan,[10] in 1949, "quick and sure action by the surgeon of the team is mandatory, and is usually effective."

Concepts of Splenic Hyperfunction

Damashek [8] believes that the normal spleen exerts a mild inhibitory effect on the bone marrow's hematopoietic activity through a hormonal influence and that, in hypersplenism, the inhibition becomes pathologically severe. There is no proof of the existence of such a hormone, but clinical and experimental evidence seem to substantiate the theory. This evidence includes improvement of hematopoietic function following splenectomy in certain nonhemolytic anemias, aplastic anemias, and myelofibrosis. In splenic thrombocytopenia there is inhibition of maturation of megakaryocytes. In splenic neutropenia there is inhibition of delivery of granulocytes. In splenic pancytopenia with nonhemolytic anemia there is inhibition of production and/or delivery of red blood cells, leukocytes and platelets. In splenic pancytopenia there is inhibition of production of platelets

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and delivery of leukocytes, and excessive destruction of red blood cells.

In some cases of chronic thrombocytopenia, studies of platelet life by radioactive tagging indicate diminished production with normal platelet life span. Following removal of the normal spleen, animals show increased resistance to X-irradiation and nitrogen mustard, suggesting that the spleen contributes something to the toxicity of these myeloinhibitory agents. Other experiments suggest the existence of a splenic hormone affecting bone marrow function.

Doan[9] is credited with the theory that splenic hyperfunction is due mainly to sequestration and phagocytosis of blood cells in the spleen. In dramatic support of this theory is the observation at the operating table of immediate termination of a true hemoclastic crisis at the moment of ligation of the splenic pedicle. Also, it is shown that in hypersplenism the blood cell counts are lower in the splenic vein than in the splenic artery. These findings are not observed in subjects with normal blood.

When radioactive red cells are transfused into a patient with hemolytic anemia, surface counting may indicate that radioactivity has become concentrated in the spleen. In such cases, splenectomy is reported to produce uniformly good results, indicating that the red cells were destroyed in the spleen.

Plasma from patients with idiopathic thrombocytopenia produces a temporary thrombocytopenia when transfused into normal recipients. Destruction of platelets under these conditions requires the collaboration of an antibody and the spleen. The origin of this antibody is not splenic, since the antiplatelet factor can be demonstrated following splenectomy even in patients whose thrombocytopenia have been cured by splenectomy.

Crosby[6] reviews the arguments for the concepts of Dameshek and of Doan as set forth in the preceding paragraphs. In summary, Crosby states, "Evidence favoring the concept of hypersplenic sequestration seems solid indeed, but it does not exclude the possibility that other splenic mechanisms may also cause cytopenic diseases. Although the evidence in favor of inhibitory hypersplenism is fragile and loopholed, there is still reason to suspect the existence of splenic humoral factors; at least, there is room for argument."

Neither of these concepts explains adequately some of the manifestations of the hypersplenic states. More and more attention is being given to auto-immunologic factors influencing these manifestations. In many patients there may be demonstrated a complex and variable antigen-antibody reaction associated with destruction of forming and circulating blood cells and platelets. Lymphocytes and plasma cells are principally involved in antibody formation and both of these cell types are abundant in the spleen.

During the past 15 years, corticosteroid therapy has been used with almost uniformly beneficial, and sometimes curative, results in hypersplenism. This is a strong indication that auto-immune factors are involved, since corticosteroids are known to inhibit the antigen-antibody reaction and also to have a lytic effect on lymphocytes and plasma cells, the antibody carriers. In 1951, Evans[13] emphasized this role of an immune mechanism as the etiological factor in certain cases of acquired hemolytic anemia and thrombocytopenic purpura occurring together or separately (Evans syndrome). Rambach and Alt,[22] in 1962, published an article entitled "A Revaluation of Hypersplenism." They contend that an auto-immune concept will explain all types of hypersplenism.

It seems obvious that the term *hypersplenism* has a broad and obscure meaning, but is useful as proposed by Chauffard, in 1907, to place such diseases in a proper frame of reference. Etiology of the hypersplenic states is also obscure, but opinion

seems to favor the sequestration and phagocytosis concept of Doan to account for most of the manifestations. It is probable, however, that other factors are effective in many cases.

Classification

Hypersplenism is classified as *primary* if no known disease involves the spleen directly, and *secondary* if caused by or associated with another pathologic state.

The designation of *primary hypersplenism* is customarily applied to three well recognized conditions: 1) idiopathic thrombocytopenic purpura, a hemorrhagic disease of unknown cause with extreme platelet reduction, responding permanently to splenectomy; 2) congenital hemolytic anemia, with an inherited defect of red cell formation characterized by spherocytosis and cured by splenectomy; and 3) primary splenic neutropenia or pancytopenia, with significant reduction in circulating neutrophilic leukocytes, without or with reduction in red blood cells and platelets, and responding to splenectomy.

The term *secondary hypersplenism* is applied to a variety of syndromes in which there is either a direct tissue involvement of the spleen by a disease process, or the functions of the spleen are altered to produce cytopenias. Prominent among those diseases which may directly involve the spleen are the leukemias, Hodgkin's disease, lymphoma, lymphosarcoma, sarcoidosis, metastatic carcinoma, benign tumors, chronic and acute infections, and chemical poisoning. Diseases indirectly producing splenic hyperfunction include acquired hemolytic anemia, Felty's syndrome, Von Gierke's disease, Gaucher's disease, collagen diseases, and cirrhosis of the liver with congestive splenomegaly. When hypersplenism develops during the course of any of these diseases, splenectomy has usually been of benefit in correcting or improving the cytopenic state, although of no significant effect upon the underlying disease.

In another group, including aplastic and hypoplastic anemia, Mediterranean anemia, sickle cell anemia, and myelofibrosis without myeloid metaplasia, all criteria for a diagnosis of hypersplenism are not present, but there is apparently a related splenic factor in production of anemia since splenectomy is beneficial in many cases (Bouroncle and Doan [2]).

Another of the many manifestations of hypersplenism challenging interpretation is acute thrombocytopenic purpura occurring shortly after a viral infection such as measles or chickenpox in children. Is this idiopathic thrombocytopenic purpura or secondary hypersplenism due to an autoimmunological reaction? Steroid therapy should always be given a trial in these cases, since many will be cured by it. Others, however, will not respond satisfactorily and splenectomy will be necessary.

Failure of splenectomy to cure a patient with an apparent primary hypersplenic state may be due to diagnostic or technical errors or some associated disease producing secondary hypersplenism may have been overlooked. At operation, a spleen may be found unexpectedly involved with Hodgkin's disease or lymphosarcoma. A fragment of the spleen (splenosis) or an accessory spleen may be retained, resulting in persistence or recurrence of symptoms.

Review of Literature

Since 1866, when Billroth [1] removed a spleen for *leukosarcoma,* the operation has been done sporadically in leukemias and lymphomas, usually for massive splenomegaly with pain and obstructive symptoms. In 1928, Mayo [21] published a review of 500 splenectomies done between 1904 and 1928. In reference to 54 of those done for *splenomyelogenous leukemia, lymphatic splenomegaly* and *Hodgkin's disease,* he stated that the operations prolonged life,

TABLE 1. *Results of Splenectomy for Secondary Hypersplenism by Various Authors*

Author	Case Material	Splenectomies	Results
Bouroncle & Doan[2]	Myelofibrosis	24	Favorable in 14 "Able to go back to usual activities, requiring none or fewer transfusions."
Chatterjea[3]	Thalassemia	36	2 deaths. All surviving operation improved.
Cole, *et al.*[5]	Various types	51	Good 11; fair 8; poor 18; deaths 8.
Doan, *et al.*[12]	Secondary thrombocytopenic purpura	59	Good, without recurrence, 84%.
Ferris, *et al.*[14]	Various types	44	Excellent 14; good 27; failure 17; death 1.
Heaton, *et al.*[15]	Hypoplastic anemia	12	Improved 6; failure 3; deaths 3.
Hilkovitz & Martin[16]	Sickle cell anemia	2	Improved 2.
Jandl[18]	Infections	4	One case each of miliary tuberculosis, infectious hepatitis, psittacosis, infectious mononucleosis, recovered from anemia.
Kimbrell[19]	Sarcoidosis of spleen	7	All good results.
Rousselot, *et al.*[23]	Hodgkin's disease	14	Improved 9, "Resolution of anemia or purpura." 2 alive 7½ and 11½ years.
Sarles & Levin[24]	Lupus erythematosus	3	"Splenectomy life saving in all."
Scott, *et al.*[25]	Acquired aplastic anemia	15	Transfusion requirements substantially reduced in 5.
Schrijver & Verdonk[26]	Hamartoma of spleen	1	"Anemia cured by splenectomy."
Sedgwick & Hume[27]	Various types	136	Mortality 9.5%. "Immediate clinical response satisfactory in all surviving patients, but hematological improvement less predictable."
Strawitz, *et al.*[28]	Lymphoma and leukemia	36	Mortality 8%. "Favorable hematological response in 80%."
Walter & Chaffin[29]	Various types in children	15	Good 4; fair 2; poor 8; deaths 1.
Wolff, *et al.*[30]	Thalassemia	18	"All benefitted initially, requiring fewer transfusions."

but were palliative and there was "no great change in the blood picture."

In 1914, King[20] stated significantly, "If it can be shown that important clinical symptoms consistently point to a hyperfunction of the spleen, and that these symptoms disappear or are strikingly mitigated when the spleen is removed from the body, an important step will have been taken toward defining changes in function of the spleen." It remained for Doan,[11] however, to focus attention on the value of splenectomy in treating secondary as well as primary hypersplenism. In a review of 828 cases with hypersplenic cytopenic syndromes published in 1958, and representing 25 years of experience, he stated among other conclusions, "Splenectomy in our hands has given: (a) more prompt remissions, (b) more permanent recoveries, with (c) fewer complications, and (d) less minor sequelae than any other treatment yet suggested and tried. (Corticosteroid treatment should be reserved for selected patients.)"

Although splenectomy for primary hypersplenism has been an accepted procedure for many years, there has been a reluctance on the part of many to extend the indication for the operation to include those diseases which often show evidence of *secondary hypersplenism.* There is now a definite trend, however, toward recognition of the value of splenectomy in secondary hypersplenism as evidenced by numerous reports which have been published in recent years (Table 1). Generally, improvement has resulted from splenectomy.

Splenectomy for Secondary Hypersplenism

A study has been made of 118 cases of various types of secondary hypersplenism and related anemias for which splenectomy has been done at Baylor University Medical Center in Dallas, Texas, during the

10-year period ending January 1, 1962. Most of these cases were referred for surgery by Dr. Joseph M. Hill, Director of the Wadley Blood and Research Center, where many have been under treatment for periods up to several years, before and after operation. Thus, the opportunity has been afforded for an almost complete follow up and evaluation of results obtained by combined medical and surgical management in this series. There were nine deaths occurring at operation or within the following few days, an operative mortality of 7.5 per cent. Since splenectomy was done only for treatment of the associated cytopenic states, and did not affect significantly the underlying serious disease processes, unless confined to the spleen only, mortality within the ensuing years was high. A high mortality rate is expected in many of these serious diseases, but splenectomy has undoubtedly prolonged life in many instances, and has facilitated the medical care. Forty-four patients are known to be living, surviving one and one-half years to almost eleven years since operation.

It must be emphasized that all patients subjected to splenectomy had been studied by a competent hemotologist. Diagnostic studies routinely included bone marrow examination, antibody tests, and other indicated procedures. Splenectomy was done only when medical management was found to be inadequate or when continuation of intensive steroid therapy was considered hazardous.

Analysis of Cases

Results of splenectomy in this series of cases are discussed in the following paragraphs and summarized in Table 2. There was immediate good response in the blood picture in 88 per cent of patients surviving operation. Late results are classified as follows:

Excellent. Return to normal blood counts without transfusions.

Good. Return to near normal blood counts with rare transfusions or none.

Poor. Improved, but requiring transfusions.

Failure. No improvement or complete loss of immediate improvement.

Chronic lymphatic leukemia is the most favorable group. There was 27 cases with two operative deaths. All had anemia and 25 thrombocytopenia. Six died between one and 12 months after operation; seven between one and two years; three between three and four years. Nine are known to be alive from one and one-half years to six

TABLE 2. *Analysis of Cases*

		Deceased					Results				
	No.	Op.	1–12 Mo.	1–2 Yr.	Longer	Living	Exc.	Good	Poor	Failure	Lost
Leukemia, lymphocytic	27	2	6	7	3	9	10	12	2	1	
Leukemia, myeloid	16	2	11	3				6	7	1	
Leukemia, monocytic	1		1						1		
Lymphoma, malignant	3					3	2	1			
Lymphosarcoma	1			1				1			
Hodgkin's disease	5		1	1	3			4	1		
Carcinoma, metastatic	2	1				1	1				
Gaucher's disease	2					2	2				
Von Gierke's disease	1					1	1				
Felty's syndrome	4				2	2	2	2			
Collagen diseases	2		1		1		1		1		
Cirrhosis of liver	4			2		2	2	2			
Thrombocytopenia	5					4	4				1
Pancytopenia	5		1			3	4				1
Acquired hemolytic anemia	19	4	3	1		8	5	5	2		3
Aplastic and hypoplastic anemias	19		9	1		7	2	10	5		2
Thalassemia	2				1	1		2			
Totals	118	9	33	16	10	43	36	45	19	2	7

years after operation. Results are considered excellent in ten, good in twelve, poor in two, and failure in one.

Case Report

E. C., white woman, age 69 years. Diagnosis of chronic lymphatic leukemia made in January 1954 when under treatment for virus pneumonia. Medical management was successful until June 1956 when there was a rise in lymphocyte count and radioactive phosphorus was given. In January 1956 hemoglobin was 11 Gm., white blood cells 18,000, lymphocytes 60 per cent, platelets 414,000. In November purpura appeared, hemoglobin was 9.0 Gm. and platelets 19,500. Two blood transfusions were given and ACTH started. On January 7, 1957, she was admitted to the hospital with hemoglobin 13 Gm., but no platelets and a generalized purpuric eruption. On January 11, 1957, splenectomy was done without incident and intensive steroid therapy continued before and after surgery. On January 12, 1957, hemoglobin was 12 Gm. and platelets 110,000. The purpuric eruption promptly disappeared and she rapidly returned to normal activity as a housewife. Since January 1961 she has taken chlorambucil 2.0 mg. daily for control of the white blood cell count, and has required no transfusions. On October 15, 1962, hemoglobin was 12 Gm., platelets 310,000, white blood cells 18,000, lymphocytes 46 per cent.

Chronic and subacute myelogenous leukemia is a less favorable group. There were 16 cases (three subacute) with two operative deaths. Eleven died between one and 12 months, and the remaining three within two years after operation. Of the 14 surviving operation, six received good results; seven, poor; and one failure.

Chronic aleukemic monocytic leukemia was represented by one case which was first diagnosed as hypoplastic anemia with thrombocytopenia of 18 months duration. After operation, the pathologic studies of the enlarged spleen, lymph nodes and biopsy of the liver showed monocytic leukemia. He survived for six weeks with a moderately improved blood picture.

Malignant lymphoma. All three cases had splenomegaly, hypersplenism and disseminated disease. One had anemia, one anemia and thrombocytopenia, and one pancytopenia. Two had excellent results and are living one and one-half years and two years after splenectomy with normal blood counts. The third is living three and one-half years after splenectomy with normal blood counts, but with a recent pathological fracture of a vertebra. All are receiving chemotherapy.

Lymphosarcoma with splenomegaly, hypersplenism, and disseminated disease was found in one patient. The result from splenectomy has been good. She is alive two years after operation, normally active, but with moderate anemia and thrombocytopenia. Chemotherapy is being continued.

Hodgkin's disease. There were five cases, all with anemia and thrombocytopenia. One survived only two months after splenectomy, but had a striking rise of platelets from 50,000 to 600,000 within one week. Hemoglobin rose from 7.0 to 13 Gm. within one month on steroid and iron therapy. Anemia and thrombocytopenia recurred and the patient expired with a cerebral hemorrhage. The four other patients lived from two years to three and one-half years, having obtained good results from splenectomy, but eventually succumbing to their disease.

Metastatic carcinoma. One patient had radical mastectomy for carcinoma of the breast in June 1954. One year later treatment was given for bone metastases, first with x-ray irradiation, then with radioactive phosphorus. Anemia and thrombocytopenia developed and splenectomy was done on March 23, 1957. She expired 24 hours after operation. Autopsy showed that the liver and bone marrow, as well as the spleen, were extensively involved with carcinoma.

A second patient had splenectomy for splenomegaly and pancytopenia of unknown origin. At operation the left kidney was found, unexpectedly, to be enlarged. The spleen showed metastatic adenocarcinoma and, when nephrectomy was done one week later, the kidney contained a

primary adenocarcinoma of the same type. The result of splenectomy is considered excellent, since the patient is still living nearly two years after the operations and has normal blood counts, but evidence of metastatic malignancy in lungs and pleura.

Gaucher's disease (glycogen storage disturbance of the liver) was found in two children, age two years and six years, each with splenomegaly and thrombocytopenic purpura. One also had anemia. Both are alive and well three and five years after splenectomy.

Von Gierke's disease (lipodystrophy) was found in a six-year-old boy with splenomegaly, anemia, and thrombocytopenia. He is alive and well four years after splenectomy.

Felty's syndrome (rheumatoid arthritis, splenomegaly, and neutropenia). All four patients had pancytopenia. Two had excellent results from splenectomy and are living four years and four months, and four years and eight months. Two had excellent immediate results, but had recurrence of anemia and thrombocytopenia shortly before death, three years and three months, and three years and four months after splenectomy.

Collagen disease. One patient had disseminated lupus erythematosus with thrombocytopenia, and another had periarteritis nodosa with anemia and thrombocytopenia. The patient with lupus erythematosus had an excellent result, with normal blood counts, until a terminal infection two years and seven months after splenectomy. The patient with peri-arteritis nodosa had an immediate good response, but died after two months.

Cirrhosis of the liver with congestive splenomegaly and hypersplenism. Of the four cases, two had thrombocytopenia. Both of these have maintained excellent results and are alive three years and five years after splenectomy. The other two cases had pancytopenia. One had a good result for a year, then died from bleeding esophageal varices six months later after an unsuccessful portalcaval shunt. The second case with pancytopenia had a good immediate result, but was lost to follow up four months after splenectomy. Sedgwick and Hume [27] reported 36 patients with cirrhosis, congestive splenomegaly and hypersplenism. In eight of these, portalsystemic shunt was done at the time of splenectomy.

Secondary thrombocytopenia of probable toxic or viral etiology. Two patients were examples of fulminating thrombotic thrombocytopenic purpura, an almost inevitably fatal disease. Both patients were treated with massive dosages of steroids without effect, but made dramatic recoveries after splenectomy. Both are alive without remissions, 11 years and five years, respectively, after operation. Hill and Loeb [17] have reported these two cases, together with a third who died before splenectomy could be undertaken.

Two patients had thrombocytopenic purpura, one after infectious hepatitis, the other after exposure to fumes of a toxic solvent. Both had excellent response to splenectomy and are living two years with normal blood counts. One patient developed thrombocytopenic purpura after recovery from homologous serum jaundice. Her immediate result from splenectomy was excellent, but she was lost to follow up after one month.

Secondary pancytopenia of probable toxic or viral etiology. Four patients had excellent results from splenectomy and are living from two years and five months to six years and five months, without remission. A fifth patient had a good response maintained for two years then was lost to follow up.

Acquired hemolytic anemia with normal or hypercellular bone marrow, enlargement of the spleen, and a positive Coombs' test for antibodies indicating an auto-immunological factor. There were 19 such cases in this series. Associated disease processes included arteriosclerotic heart disease, hepa-

titis, gallbladder disease, duodenal ulcer, chronic and acute bacterial infections, viral infections, and drug reactions. Several patients were critically ill when splenectomy was done as a life-saving effort after steroid therapy and blood transfusions had failed to arrest the hemolytic process. There were four operative deaths, two occurring on the operating table, one four days, and one ten days after surgery. Results were excellent in five patients, although one died three months after operation, of congestive heart failure. The other four are living after one to five years and have shown no remissions. Good results were obtained also in five patients. One died after three months, of congestive heart failure, and another after one year, of liver failure. Three are living after one year and eight months to five years and ten months, without remissions. Two had poor results, although one is living one year and nine months requiring steroid therapy and blood transfusions. One died of hepatitis one month after splenectomy. Three were lost to follow up after one to six months.

Related Anemias

Aplastic and hypoplastic anemias are characterized by acellular or hypocellular bone marrow and, consequently, are not examples of hypersplenism according to definition. Improvement in many cases after splenectomy, however, suggests a relative splenic factor. Doan,[10] Heaton,[15] and others recommend splenectomy in selected cases on the basis of possible improvement and prolongation of life in diseases which otherwise are almost inevitably fatal.

In this series there were nineteen cases of aplastic or hypoplastic anemia. There were no operative deaths. Seven patients are living from one year and six months to three years and nine months. Two show excellent results, requiring no transfusions. Four have good results, but require medical therapy. One has a poor response although requiring fewer transfusions than were necessary before operation. Nine died within one year after operation, six having temporary good results and three poor results. One died one year and four months after operation, having had a poor response. One had an excellent response for one year and six months, but was then lost to follow up and another, with poor response, was lost to follow up after two months.

Thalassemia (*Mediterranean anemia*) is a form of hereditary hemolytic anemia characterized by a familial and racial incidence, typical facial and skeletal changes, and enlargement of the spleen. The bone marrow is hyperplastic and the peripheral blood contains large numbers of erythroblasts and abnormal erythrocytes, especially leptocytes. Splenectomy in this disease, as well as in sickle cell anemia, will often relieve the effects of excessive red blood cell destruction (Wolff *et al.*[30] and Hilkovitz and Martin[16]).

In this series, good results were obtained in both cases. One is living for five years and four months on medical therapy and an occasional transfusion. The other died after four years and four months, having been moderately improved until the occurrence of a terminal infection.

Summary

Hypersplenism is an abnormal hematologic state brought about by overactivity of the spleen. It is characterized by various forms of cytopenia, classified as primary if no known disease involves the spleen directly, and as secondary if caused by or associated with another pathologic state. The etiology of splenic hyperfunction is uncertain. Doan's theory of sequestration and phagocytosis of blood cells and platelets by the spleen has been accepted widely. An auto-immunologic factor is apparent in many cases.

Splenectomy has long been the treatment of choice for primary hypersplenic states, but in recent years, indications for splenectomy have been extended cautiously to in-

clude many cases of secondary hypersplenism.

Splenic hyperfunction may result from direct involvement of the spleen by leukemia, lymphoma, lymphosarcoma, Hodgkin's disease, sarcoidosis, benign tumors, metastatic carcinoma, chronic and acute infections, and chemical poisoning. Diseases producing splenic hyperfunction indirectly include acquired hemolytic anemia, Felty's syndrome, Von Gierke's disease, Gaucher's disease, collagen diseases, and cirrhosis of the liver with congestive splenomegaly.

In a third group of diseases the importance of the splenic factor is equivocal. This group includes aplastic and hypoplastic anemias, thalassemia, sickle cell anemia, and myelofibrosis without myeloid metaplasia. However, in many instances there apparently is a relative state of hypersplenism and splenectomy is often beneficial.

Our experience with splenectomy in treatment of secondary hypersplenism and related anemias includes 118 cases. The operative mortality was 7.5 per cent. Among those patients surviving operation, there was immediate improvement of the cytopenic state in 88 per cent. This improvement was maintained in varying degree among those who have died and those who are still alive. Forty-nine (44.5%) died within two years after operation. Ten (9.0%) died between two years and seven years after operation. Forty-three (40%) are living, one for eleven years. Seven (6.3%) are lost to follow up.

Late results are tabulated as follows:

Excellent (return to normal blood counts without transfusions) in 36 (33%).

Good (return to near normal blood counts with rare transfusions or none) in 45 (40.9%).

Poor (improved, but requiring transfusions) in 19 (17.2%).

Failure (no improvement or complete loss of immediate improvement) in two (1.8%).

Results in this and in other published series seem to justify the conclusion that splenectomy is a valuable adjunct to medical therapy in many carefully evaluated and selected cases of secondary hypersplenism and related anemias.

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