reason in post-pneumonic cases relatively early decortication may not be quite so necessary.

2. The original workers considered that early decortication played its greatest part in the treatment of total collapse or apical collapse. Patients with partial empyema, if limited toward the base, do sufficiently well after simple drainage during this transition period between the acute and chronic stages.

SUMMARY

1. The fundamental principles of the treatment of acute empyema have been reviewed as they influence the causes of chronicity.

2. A brief review of the treatment of chronic empyema has been outlined.

3. Three important changes in the empyema problem due to the introduction of penicillin have been mentioned. (*a*) Acute post-pneumonic empyema has become a rare condition in civilian practice. (*b*) There is still danger of undue delay in instituting adequate surgical drainage. (*c*) Thorough decortication is now possible with a low mortality rate, at least, during a limited transition period.

REFERENCES

1. PAGET, S.: Surgery of the Chest, 1896.
2. FARQUHARSON, R. F.: *Canad. M. A. J.*, **53**: 199, 1945.
3. BLADES, B.: *Ann. Surg.*, **121**: 672, 1945.
4. GRAHAM, E. A. AND BELL, R. D.: *Am. J. Med. Sc.*, **156**: 839, 1918.
5. GRAHAM, E. A. AND BERK, M.: *Ann. Surg.*, **98**: 520, 1933.
6. HEDBLOM, C. A.: *J. Am. M. Ass.*, **81**: 999, 1923.
7. CLAGETT, O. T. AND SHEPHARD, V. D.: *J. Thoracic Surg.*, **12**: 464, 1943.
8. BURFORD, T. H., PARKER, E. F. AND SAMSON, P. C.: *Ann. Surg.*, **122**: 163, 1945.
9. EARLE, B. K. AND MEADE, R. H.: *Surg., Gyn. & Obst.*, **82**: 13, 1946.
10. SANGER, P. W.: *Surg., Gyn. & Obst.*, **82**: 71, 1946.

RÉSUMÉ

Revue des principes fondamentaux du traitement de l'empyème aigu et des causes de l'empyème chronique. Revue des techniques chirurgicales et discussion de celles-ci. Il ressort de l'expérience de la guerre que la méthode de Fowler-Delorme est remise à la page et que la décortication de la plèvre est possible et même indiquées si l'opération est faite assez tôt après le début de l'empyème. L'usage de la pénicilline a encore facilité les choses et il est rare de voir aujourd'hui les empyèmes aigus après une pneumonie. Le danger de retarder la décision de drainer est toujours aussi grand et si l'on choisit de faire le décortication il faut procéder assez tôt.

JEAN SAUCIER

The evaluation of penicillin in the treatment of patients with syphilis is still going on and must continue for several years more before concrete deductions may be made as to its actual value. Penicillin has the following advantages: it produces few reactions and it can be given in a short period of time. The need for hospitalization for a week or ten days and injections given at two or three hour intervals are its disadvantages. It may be that penicillin in beeswax and peanut oil will overcome these objections.—P. A. O'Leary and R. R. Kierland, *J. Am. M. Ass.*, **132**: 428, 1946.

SPLENOSIS

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SPLENECTOMY is the recognized treatment for an increasing number of diseases which formerly belonged in the province of medicine, as well as being imperative in rupture of the spleen. Curtis and Movitz[1] reported a series of 178 splenectomies covering a list of 21 diseases for which splenectomy was indicated. They found congenital hæmolytic icterus to be the disease in which it was most commonly indicated—53 cases or 29.7% in their series of 178 cases. The next most common indication was primary thrombocytopenic purpura, 33 cases or 18.5%.

The spleen is coming more and more into the field of the ductless glands. Hypersplenism or an increase in splenic secretions is being looked upon as a possible cause for the destruction of all or certain elements of the blood, such as that found in thrombocytopenic purpura where only the platelets of the blood are affected, and again in congenital hæmolytic icterus, where a marked destruction of the erythrocytes goes on, due to an increased fragility of the cells. The increased pigments in the blood and tissues from the broken-down erythrocytes which must be excreted by the liver results in a high incidence of gallstones of the bile pigment type. In a series of 118 cases reported by Pemberton[13] "conclusive evidence of disease of the gall bladder, with or without stones occurred as a secondary complication in 81 cases (68.6%)".

There is nothing more convincing in surgical therapy that the spleen is the causative factor in these two blood diseases than the dramatic and usually lasting improvement that follows removal of an enlarged and obviously diseased spleen. One boy from whom I removed the spleen at the age of 17, for congenital hæmolytic icterus of the familial type—his father and one sister had splenomegalia also — has been four years in the army during the recent war. It is now eighteen years after splenectomy, and there has been no recurrence of his jaundice or anæmia. There are, however, cases that recur after a period of cure or marked improvement following splenectomy. If recurrence occurs, McLaughlin, Sharpe and Cunningham[2] concluded that: (1) the original diagnosis was in-

correct: (2) overlooked splenic tissue had become active; or that (3) hæmolymph nodes had hypertrophied and assumed excessive hæmolytic activity.

Curtis and Movitz[1] believe the recurrence to be due to overlooked accessory splenic tissue. They report a case of recurrence in a patient in which an accessory spleen was observed but not removed at the time of splenectomy. They report another case of recurrence in which the patient died and the accessory spleen was found at necropsy. But their most convincing case was a case of recurrence relieved by the removal of an accessory spleen. The incidence of accessory spleens varies according to age. They are much more common in infancy and childhood and tend to decrease with advancing age, from some form of involutional atrophy. Adami and Nicholls[3] report an incidence of 11% in all their autopsies. The incidence is also greater in patients with splenic disease. Curtis and Movitz[1] report 56 cases, or an incidence of 31.4% of accessory spleens found in a series of 178 cases of splenic disease. Maingot[4] reported an incidence of 44.4% of accessory spleens found in a series of 18 cases where the spleen was removed for primary thrombocytopenic purpura. Vaughn[5] reported a 17% recurrence of primary thrombocytopenic purpura following a period of relief after splenectomy in a series of 303 cases. Curtis and White[6] think that this 17% recurrence corresponds closely enough with Adami and Nicholls 11% incidence of accessory spleens to attribute the recurrence to the accessory spleen.

True accessory spleens are only found in areas where splenic tissue developmentally arises, not on the parietal peritoneum or ventral mesogastrial layers, and are limited in number, there being rarely more than ten. There is, however, another condition in which multiple pieces of splenic tissue are found attached and growing throughout the whole peritoneal cavity, on parietal as well as visceral peritoneum, in the pelvis, on the diaphragm, omentum and dome of the liver. In one case reported by Shaw and Shafi[7] a portion of splenic tissue was found growing in the left thoracic cavity opposite the eighth dorsal vertebra, together with numerous other small splenic implants in the peritoneal cavity of the same patient (Fig. 1). Albrecht in 1896 (quoted by Curtis and Movitz[1]) was the first to report a case of this kind; there were as many as four hundred small accessory spleens in the patient, and a similar case by Orth was cited. Since then there have been 15 similar observations reported, according to Waugh,[12] 4 of which are in the American literature. Hamrick and Buch[8] give a synopsis of 13 of these cases and since then there have been two others reported, one by Waugh.

These multiple splenules are not considered in the same category as the usual accessory spleen. They have been found either during a subsequent laparotomy or at autopsy, in patients who have had a splenectomy for traumatic rupture or in patients who gave a history of an abdominal injury in the region of the spleen.

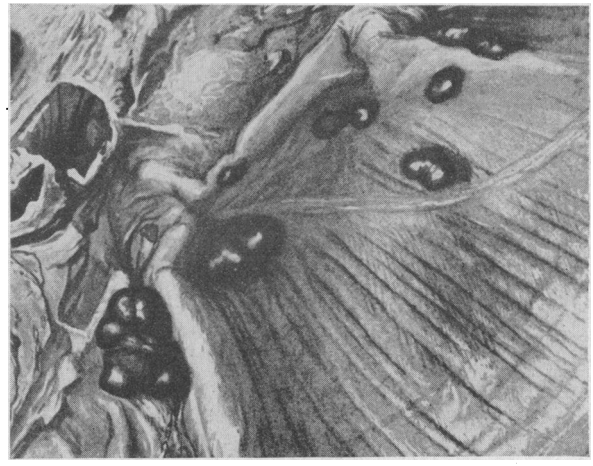

Fig. 1.—Part of left vault of diaphragm showing a number of splenic transplants. Natural size.
(From Shaw and Shafi's article.)

It has been shown experimentally that splenic tissue may be implanted in the peritoneum and readily survives transplantation. Von Stubenrauch (quoted by Buchbinder and Lipkoff[9]) removed the spleen in several dogs, crushed the pulp and "seeded" these throughout the peritoneal cavity. On sacrificing the dogs from one to three months later, he was able to demonstrate the same picture he described in man. Marine and Manley[10] in 1920 and Perla[11] in 1936 showed the readiness with which autoplastic splenic transplants grow in rabbits and albino rats although their transplants were into the abdominal wall and not the peritoneum. Buchbinder and Lipkoff[9] think Foltin in 1911 to be the first to suggest that these multiple splenules were "seeded" throughout the peritoneal cavity in man by the blood which carried splenic pulp cells to all areas of the abdominal cavity at the time of the ruptured spleen.

Buchbinder and Lipkoff originated the term "splenosis".

So far I have been unable to find any case reported in which widely disseminated splenic nodules were found following splenectomy for non-traumatic disease of the spleen. Shaw and Shafi express the opinion that the removal of diseased spleens apparently is rarely, if ever, followed by implants, in spite of occasional tearing and its associated hæmorrhage during splenectomy. This same opinion is held by several other authors including Waugh[12] and Hamrick and Bush[8] who also make the statement that "as yet no symptoms have been known to result from these splenic implants". The following case would appear to be the first recorded case in which autoplastic transplants of splenic tissue occurred in the abdomen following splenectomy for a non-traumatic condition and also in which these transplants have caused a recurrence of the disease for which splenectomy had been performed, namely congenital hæmolytic icterus.

Case Report

Mrs. H. This patient, a married woman of 23 years, came under my care in September, 1940. There was nothing of significance in the family history other than that her mother and one sister had gallstones. This girl had never been strong, had "weak spells" and her complexion was always sallow and muddy. At the age of 18 years, her doctor told her she had gallstones, and her history for the five years preceding admission to hospital was a clear cut history of dyspepsia due to cholecystitis. On admission she was five months' pregnant, deeply jaundiced, the jaundice having had its onset ten days previously following a severe attack of upper abdominal pain. The laboratory data were as follows: red blood cells 3,110,000; white blood cells 15,000; Hg. 53%; icterus index 41; van den Bergh, delayed direct reaction 7.2; the urine showed bile 3 plus and bile salts 3 plus; temperature, pulse, and respiration were normal.

She complained of pain under the right costal margin and was extremely tender to touch in this area. The diagnosis of acute cholecystitis was obvious. After vitamin K, transfusions of blood, glucose and saline therapy, she was operated upon under spinal anæsthesia. At operation the gall bladder was found to be acutely inflamed and full of stones. The gall bladder and contents were removed. Five black, sharp, jaggy stones were found in the common bile duct, proximal to a stricture in the common bile duct, situated between the entrance of the cystic duct and the bifurcation of the hepatic duct. These stones were removed, the stricture was dilated and a "T" tube drainage established with the lower limb of the tube through the stricture. Her convalescence was very stormy; drainage was poor through the "T" tube. The jaundice cleared slowly. However, she carried on and on December 1, 1940 was delivered of a normal, healthy child, three months after cholecystectomy. She gained strength very slowly. The jaundice never completely cleared and in February, 1941, it began to deepen and continued to do so until April, at which time she was re-admitted to hospital, deeply jaundiced. The laboratory findings were: red blood cells 3,310,000; white blood cells 15,650; Hg. 54%; icterus index 50; van den Bergh, qualitative prompt direct; quantitative 7.2 unit.

It was obvious that the stricture had recurred. A second operation was performed at which the stricture was excised and the gap bridged with a rubber tube. Convalescence was satisfactory, the jaundice disappeared slowly, but she remained pale and sallow. Following this she led a fairly normal life for about one and one-half years, then began to have "weak spells" again. The jaundice returned, unaccompanied by pain or indigestion and gradually deepened until October, 1942. She was re-admitted to hospital one and one-half years after the second operation. Her blood studies showed: red blood cells 3,215,000; white blood cells 31,950; Hg. 73%; icterus index 30.

On this occasion she was found to have a greatly enlarged spleen and the diagnosis of congenital hæmolytic icterus was made. After being given vitamins, blood and parenteral fluids, a splenectomy was done on October 16. The spleen was greatly enlarged weighing 479 gm. and was found to be attached by dense adhesions to the stomach, diaphragm, colon and parietal peritoneum. The operation was difficult and very bloody due to tearing of the capsule.

Her convalescence was rapid and the jaundice cleared very quickly. Her colour was better than it had ever been in her life and she enjoyed better health than ever before, until September, 1945, when she had a few crampy pains in the upper abdomen followed by a slight tinge of jaundice, which soon cleared after she passed the rubber catheter by bowel four and one-half years after it had been inserted. She was then as well as formerly until early 1946, when she became pregnant and her former attacks of fatigue reappeared, her complexion became sallow and the jaundice soon reappeared. She was re-admitted to hospital in July, 1946, eight months' pregnant, showing: red blood cells 2,900,000; white blood cells 32,450; Hg. 54%; icterus index 41.

She was suffering a great deal of abdominal pain which was thought to be caused by the increasing size of the uterus stretching an abdomen which was limited in size by numerous adhesions. After blood transfusions, vitamins and fluids, a Cæsarean section was done on July 5. When the abdomen was opened, numerous spleen-like masses were found scattered throughout the whole abdomen. Their true significance was not realized at the time, although I made the remark that they looked like small spleens. Six of these tumours were removed and sent to the pathologist and he confirmed the diagnosis of splenic tissue. No attempt was made to count them, but there must have been over one hundred. They were in the pelvis, on the parietal peritoneum, the surface of the uterus, and very numerous on the omentum, ranging in size from a pea to that of a plum, and the colour of a normal spleen.

Convalescence was slow, but she gradually improved and left the hospital on July 15. She returned in September for a check-up. She then looked well, her complexion was clear, although still slightly anæmic: Hg. 72. Otherwise her blood picture and laboratory tests were normal.

Summary and Conclusions

A case of splenosis is reported, in which "seeding" occurred after splenectomy for non-traumatic reasons.

The case is the first to be reported in which the splenic transplants showed function.

The surgical technique for splenectomy for either traumatic or non-traumatic reasons should be designed to completely remove all blood from the abdominal cavity and the abdominal wall incision should also be protected from splenic blood to prevent implants.

A recurrence of congenital hæmolytic icterus following splenectomy may be caused by splenosis.

I wish to acknowledge the splendid assistance given me by our pathologist, Dr. J. B. McKay, in the laboratory tests necessary to diagnose the several phases of this case.

REFERENCES

1. CURTIS, G. M. AND MOVITZ, D.: *Ann. Surg.*, **123**: 276, 1946.
2. MCLAUGHLIN, C. W. JR., SHARPE, J. C. AND CUNNINGHAM, R.: *Internat. Clin.*, **4**: 108, 1941.
3. ADAMI, J. G. AND NICHOLLS, A. G.: Principles of Pathology, London, 1911.
4. MAINGOT, R.: Abdominal Operations, Vol. 1, Appleton Century Co., 1940.
5. VAUGHIN, J. M.: *Brit. M. J.*, **2**: 842, 1937.
6. CURTIS, G. M. AND WHITE, P. L.: *Trans. West Surg. Ass.*, **46**: 364, 1937.
7. SHAW, A. F. B. AND SHAFI, A.: *J. Exper. Path. & Bact.*, **45**: 215, 1937.
8. HAMRICK, R. A. AND BUSH, J. D.: *Ann. Surg.*, **115**: 84, 1942.
9. BUCHBINDER, J. H. AND LIPKOFF, C. J.: *Surgery*, **6**: 927, 1939.
10. MARINE, D. AND MANLEY, O. T.: *J. Exper. M.*, **32**: 113, 1920.
11. PERLA, D.: *Ann. J. Path.*, **12**: 665, 1936.
12. WAUGH, L.: *New England M. J.*, **234**: 621, 1946.
13. FEMBERTON, J. DEJ.: *Ann. Surg.*, **94**: 755, 1931.

NEW FACTORS IN THE ETIOLOGY OF OBLITERATIVE ARTERIAL DISEASE*

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THIS brief report has been prepared in an attempt to shed some further light on the etiology of obliterative arterial lesions of the legs. The subject material is taken from 25 cases recently studied in the Vascular Service of the Royal Victoria Hospital and certain conclusions are drawn from the clinical and laboratory investigations, and also from the fact that in every case a specimen of artery for pathological examination was obtained from one of the leg arteries.

The diagnosis of obliterative arterial disease of the lower limb is not too difficult to make providing one keeps the condition in mind and spends a few minutes in the examination of the feet and legs. A true history of intermittent claudication or ischæmic neuritis, and the absence of pulsations in the dorsalis pedis, posterior tibial and popliteal arteries are easily determined. The presence of ischæmic changes in the foot with blanching on elevation and rubor on dependency will corroborate the findings. However, the determination of the type of obliterative disease is a much more difficult matter.

In following the present day literature on this subject there appear to be only two causes of chronic ischæmia of the extremities. These are Buerger's disease and arteriosclerosis. The differential diagnosis between Buerger's and an early case of arteriosclerosis has been most difficult to make but, in brief, it appears that in the past the diagnosis of Buerger's disease was mainly made by excluding any evidence of arteriosclerosis in the patient. This diagnosis was aided by such additional evidence as the youth of the patient, his being of the Jewish race, with a history of heavy smoking, and perhaps of a migrating phlebitis. Using such criteria therefore, the majority of cases of obliterative vascular disease up to the age of fifty were diagnosed as Buerger's. Such was the situation preoperatively in 19 of our 25 cases.

It is the purpose of this paper to present some evidence that the picture is not quite so simple as this; that Buerger's disease is relatively rare; and to describe a group comprising the majority of our cases, showing a picture which is neither Buerger's disease nor arteriosclerosis as we now define it.

METHODS AND RESULTS

In the cases studied, an investigative routine was carried out and consisted of a hæmogram, electrocardiogram and cardiac examination, blood Wassermann, x-ray of the arteries of the pelvis for calcification, examination of the fundi for evidence of arteriosclerosis, oscillometric readings of the leg arteries and skin temperature readings before and after lumbar sympathetic procaine block. More recently we have used the new drug tetra ethyl ammonium chloride to check the results of the sympathetic block. This drug causes a paralysis of the autonomic nervous system at the ganglionic synapses for varying periods of time depending on whether it is given intravenously or intramuscularly. The results in skin temperature readings have closely paralleled those following lumbar sympathetic block.

All cases had pathological examination of a leg artery taken either from an amputation specimen or from biopsy of the dorsalis pedis artery at the time of sympathectomy.

Six of the 25 cases showed evidence of arteriosclerosis in the fundi or on x-ray of the

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