differences between group 1 and groups 2 and 3 were highly significant for both P_{CO_2} and lactate.

Furthermore, the differences were apparent in the first hour of perfusion and certainly reflected the degree of lactic acidosis which the kidney had developed before cooling.

The position of any given kidney in relation to the regression line of perfusate pH against lactate is a precise indication of that kidney's chance of functioning immediately and of having reasonable function three months after transplantation.

It was of particular interest to note that the perfusion characteristics of group 3 kidneys closely paralleled those of group 2 in terms of pH, P_{CO_2}, and lactate.

There was also a high degree of correlation between the severity of lactic acidosis and the accumulation of LDH in the perfusate. Since LDH is present in large quantities in the cytoplasm of renal tubular cells, a rise in LDH should indicate the extent of renal tubular injury. LDH accumulated progressively throughout perfusion and there was overlap between the groups. The release of enzymes by injured cells does not follow a uniform pattern and when low may reflect patchy areas of injury or poorly distributed perfusate.

The injury process shown by this study appears to have occurred most often in the agonal period since there was no significant difference in warm ischaemic interval between the groups. Neither was there any significant difference in ice storage between the groups. Paradoxically, group 1 kidneys had the longest total storage times; this was because prolonged periods of perfusion were regarded as safe by virtue of a stable pH.

The antemortem injury was usually due to a prolonged agonal period with either hypotension or a long interval between ventilatory and circulatory arrest. The ventilatory support which all the group 1 donors received appears to have reduced significantly the risk of injury. The injury process was usually subtle and apparently not significant from donor history. Most of the kidneys washed out rapidly and uniformly, confirmed by their subsequent lack of resistance to perfusion. It was only by perfusing the kidney and correlating changes in pH and lactate and P_{CO_2} that differences between them could be distinguished. It may be that continuous perfusion will have its greatest application as a method of assessing kidneys before transplantation.

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MEDICAL MEMORANDA

Iatrogenic Rupture of the Spleen

A. J. MEARNS

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The hazards of pleural biopsy and aspiration are well known, but there is little documentation of splenic injury after these procedures. Shepherd (1960, 1968), in an extensive review of the literature of trauma to the spleen, makes no reference to this type of injury. In a series of 308 splenectomies (Slate *et al.*, 1969) 108 were performed as a result of iatrogenic injury, but none were the result of chest aspiration or pleural biopsy.

Textbooks recognize the risk of splenic injury during such procedures (Davis, 1967), and although serious damage is rare the spleen may sustain many minor subcapsular injuries.

Three cases were recorded during a period of seven years in the Liverpool Regional Thoracic Surgical Centre and they illustrate the presentation of such injury and the difficulties of its diagnosis.

Hammersmith Hospital, London W12 OHS
A. J. MEARNS, F.R.C.S., Registrar in Cardiothoracic Surgery

Case Histories

CASE 1

A 50-year-old woman complained of weight loss, left chest pain, and occasional haemoptysis for 11 months. On admission to hospital signs of left lower lobe pneumonia were present. After seven days' treatment bronchoscopy and pleural biopsy were performed. The bronchoscopy showed a stenosis of the left lower lobe bronchus, and a biopsy specimen was taken. A pleural biopsy was attempted through the seventh intercostal space in the posterior axillary line.

That evening she complained of pain in the left chest and shoulder. The abdomen was soft and no tenderness was elicited. The blood pressure was 110/70 mm/Hg. A few hours later she was pale, sweating, and had abdominal pain. The abdomen was distended and bowel sounds were absent. The blood pressure was 70/0 mm/Hg. Blood and plasma transfusions were started, and the blood pressure rose to 110/70 mm/Hg; three more bottles of blood were given during the night.

At laparotomy there was 750 ml of free blood in the peritoneal cavity, and a bleeding spleen was removed. Multiple secondary deposits in the liver were noted. Biopsy of the bronchus and liver deposits showed oat cell carcinoma, and the patient died of carcinomatosis three months later. Examination of the spleen showed a subcapsular haematoma with rupture near the upper pole on its diaphragmatic aspect and a 1·4-cm track passing into the pulp at this site.

CASE 2

A 33-year-old woman was admitted to hospital with tiredness, ankle oedema, and haemoptysis. There were clinical signs of rheumatic mitral stenosis. She was taking 0·25 mg digoxin twice a day, 40 mg frusemide (Lasix) daily, and warfarin.

A chest radiograph showed signs of pulmonary venous hyper-

tension and atrial enlargement. E.C.G. showed right ventricular hypertrophy and atrial fibrillation. The haemoglobin was 13 g/100 ml, and the blood pressure was 120/80 mm/Hg. Mitral valvotomy was performed, and the wound was closed with an under-water seal drain, which was removed on the first postoperative day.

On the fourth postoperative day a left pleural effusion was aspirated through the left seventh intercostal space in the posterior axillary line and 250 ml of blood-stained fluid was obtained. On the seventh post operative day the patient collapsed with pain in the abdomen, left shoulder, and left hypochondrium. The blood pressure was 70/= mm Hg and the haemoglobin was 10 g/100 ml. The pulse was 120/min in atrial fibrillation. The serum amylase was 100 Somogyi units/100 ml. There was tenderness and guarding throughout the abdomen, and bowel sounds were absent.

With a presumptive diagnosis of mesenteric embolus a mid-line laparotomy was made showing a large amount of free blood and a large perisplenic haematoma. There was no evidence of mesenteric embolus, and a splenectomy was performed.

The patient was well four years later. The spleen was normal with a subcapsular haematoma and embedded in clot. There were two small punctate marks on it, presumably due to needle puncture.

CASE 3

A 66-year-old woman was referred to the surgical unit for investigation of a persistent pleural effusion, which had been aspirated on two previous occasions. The first aspiration had produced straw-coloured fluid and the second blood-stained fluid. Neither specimen showed malignant cells. A Mantoux test was negative, and bronchoscopy showed no abnormality. Left pleural biopsy retrieved pleura with some evidence of recent inflammation. The haemoglobin was 12·5 g/100 ml. Six weeks later she complained of progressive tiredness, pain in her left shoulder and chest which was worse on inspiration. There was tenderness and fullness of the left hypochondrium. Her temperature was 100·6°F (38·1°C) and her haemoglobin was 8·9 g/100 ml.

Chest aspiration in the seventh left intercostal space in the mid-axillary line produced blood, the haemoglobin content of which was 7·4 g/100 ml. A chest radiograph and screening of the diaphragm showed a fixed raised left hemidiaphragm and a small left pleural effusion. It was a hypochromic anaemia which responded to iron given by mouth. Diagnosis of a primary pleural tumour involving the diaphragm was considered. At thoracotomy there were recent friable inflammatory adhesions found. The upper surface of the diaphragm was normal, with a mass beneath. An incision made through the diaphragm entered a large perisplenic haematoma walled off from the rest of the peritoneal cavity by omentum, stomach, and transverse colon.

The spleen was removed with a large amount of clot and altered blood. The spleen was histologically normal with a large subcapsular haematoma extending over the whole convex aspect of the surface and an enveloping perisplenic haematoma. The patient was well 15 months later.

Comment

The clinical problems after splenic rupture fall into three groups characterized by the delay in presentation and type of symptoms. Group 1, the acute ruptured spleen, is the commonest presentation after damage to both capsule and pulp, resulting in intraperitoneal haemorrhage with attendant symptoms of hypotension and peritoneal irritation. Group 2, the delayed ruptured spleen, differs from group 1 in that the initial injury results in subcapsular haematoma only, with rupture following in days or even weeks depending on the integrity of the capsule (Fulty and Altemeier, 1955). Group 3, the occult ruptured spleen, is both rare and difficult to diagnose. The subcapsular haematoma after an injury is walled off by a low-grade inflammatory response involving the omentum, stomach, and transverse colon, thus containing any perisplenic haemorrhage.

Case 1 follows the pattern of acute ruptured spleen, closely related in time to the injury. The main features of this syndrome are well described elsewhere.

Case 2 was a testing case of differential diagnosis. Incidence of embolus after valvotomy has been reported to be as high as 30% (Storm and Hansen, 1955). Even in the absence of calcification of the valve or clot in the atrium, the incidence of systemic emboli was found to be 3% (Coulshed *et al.*, 1970). Emboli occasionally occur even in the presence of anticoagulant cover (Ricordeau and Balansa, 1957; Smith *et al.*, 1965). The major procedure planned was removal of a presumed embolus from the superior mensenteric artery. The correct diagnosis became apparent only at laparotomy.

Case 3 is a good example of the misdiagnosis of the occult ruptured spleen syndrome (Drapanas, 1969) in which the correct diagnosis has seldom been made preoperatively (Foley *et al.*, 1969). As in this case the most common misdiagnosis was that of neoplasm (Drapanas, 1969; Foley *et al.*, 1969). In retrospect it is difficult to say which procedure caused splenic damage as two aspirations and a pleural biopsy had been performed. The chest aspiration which produced blood with a high haemoglobin content had probably penetrated the diaphragm, aspirating blood from the subphrenic space, and it was unlikely to have been the cause of the injury. There was no evidence to suggest this was the rupture of a diseased spleen—that is, the rupture of sclerotic vessels due to minor trauma (Coleman, 1939; Jones, 1944). No other pathological conditions known to predispose to splenic rupture were present. The spleen is rendered more vulnerable to injury during aspiration or pleural biopsy when there is a possibility of left lower lobe collapse, as in these three cases. In similar circumstances the aspirating needle should be inserted with care, and not, as sometimes happens, too low down.

An interesting aspect of Case 3 was the use of the transthoracic approach to the lesion. The diaphragmatic incision produces immediate identification of the disorder, and the pitfall of the apparent malignant mass in the left upper quadrant, encountered in the abdominal approach, is avoided (Lorimer, 1964).

With the increasing use of selective angiography of the branches of the aorta (Drapanas, 1969; Foley *et al.*, 1969) the diagnosis of the occult ruptured spleen will undoubtedly be easier in the future.

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