coexistence of these two defects. It is, of course, not possible on the data available to distinguish between a secondary or compensatory component of the metabolic acidosis and a primary component.

This method of plotting the acid-base disturbance shows that the simple estimation of bicarbonate concentration or alkali reserve in conditions such as these gives insufficient information for proper management. In the present case, the combination of a low bicarbonate concentration and overbreathing could easily have been misinterpreted as indicating a primary metabolic acidosis with acidaemia and have led to the administration of alkali with possibly harmful results.

Treatment

After the first few hours in hospital it was clear that the patient, in addition to having salicylate intoxication, was also in the early diuretic phase of acute renal failure probably due to tubular necrosis. The latter condition required water and electrolyte replenishment, which was given as described above. Attempts to collect full balance data were unfortunately frustrated by the diarrhoea and urinary incontinence. Correction of the acid-base disturbance would have required the administration of alkali to combat the metabolic acidosis, and the reduction of pulmonary ventilation to correct the respiratory alkalosis. Alkali is often recommended because, in addition to combating the metabolic acidosis, it hastens the excretion of salicylates. We withheld alkali in the early stages of treatment because there was an alkalaemia and because the severe renal damage would probably have prevented the rate of salicylate excretion from being significantly increased. It is of interest that in the most authoritative review of the subject Harvie and Singer (1955) doubt the value of alkali administration even in the presence of adequate renal function.

Freier *et al.* (1957) described the case of a patient with salicylate intoxication in whom the respiratory alkalosis was treated by the use of muscular relaxants and artificial respiration to decrease the ventilation. Although they did not estimate the *p*H or pCO_2 the severe tetany of this patient makes it very probable that there was in fact an alkalaemia. It might be inadvisable, however, to use this method simply on the evidence of a high pulmonary ventilation in the absence of good evidence of an alkalaemia. The Chart suggests that if we had acutely reduced the alveolar ventilation to restore the arterial pCO_2 to normal levels (40 mm. Hg) a mild alkalaemia (*p*H 7.46) would have been converted into a more severe acidaemia (*p*H about 7.26).

Fortunately, the clinical progress of our patient and the mild degree of alkalaemia did not demand any specific treatment for the respiratory alkalosis.

Summary

A case of acute renal failure probably due to tubular necrosis following salicylate poisoning is described. A review of the literature suggests that renal damage in salicylate poisoning may be commoner than is generally appreciated and may play a part in the acid-base disturbance. The acid-base disturbance and its bearing on treatment are discussed.

We thank Professor A. Kekwick, under whose care the patient was admitted, for his permission to report this case, and Dr. R. B. Singer for his helpful comments. Many of the investigations were performed by the staff of the routine laboratory of the Courtauld Institute.

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Medical Memorandum

Retroperitoneal Duodenal Rupture

Cohn *et al.* (1952), in a review of 25 cases, estimated that the mortality of retroperitoneal rupture of the duodenum due to blunt force is 20%. Johnson (1944), in a review of the world literature from 1916 to 1944, found only 52 bona-fide cases of retroperitoneal rupture, with a mortality of 50%. Schumacher (1910) and Guibé (1910) gave a mortality of over 90%. The mortality has been falling steadily, no doubt due to more recent methods. When it is considered that the lesion is sometimes discovered only at necropsy and that several series record that it has been missed at laparotomy, there need be no surprise that the mortality remains as it is.

It has been estimated that 10% of all gastro-intestinal ruptures are duodenal and that a quarter of those are retroperitoneal, 90% of the latter affecting the second and third part.

Aetiology

The second part of the duodenum is fixed relatively rigidly by its peritoneal attachments and the third part is vulnerable because it can be compressed against the lumbar spine. Another more theoretical factor in the mechanism of rupture is the blow-out effect: when the pylorus is closed and the duodeno-jejunal angle acute an air trap is formed. The causative forces are many—crush and blast injuries, kicks, traffic accidents, sudden deceleration causing steering-wheel impingement in the epigastrium, etc. The force may be minimal and the causative accident ignored because it was so trivial.

Associated injuries include damage to the liver, spleen, kidney, pancreas, stomach, intestines, omentum, and biliary passages. The lower ribs and transverse processes are often involved and there may be thoracic or more distant skeletal injuries. All these, especially the intraperitoneal lesions, serve to distract the surgeon's attention at the time of operation, and unless signs are fairly gross the duodenal injury may be overlooked.

Diagnosis

The mode of presentation is by no means constant. There is often a paucity of symptoms, and the early course may be benign and insidious. To await overt signs like generalized rigidity may mean the optimum time for intervention has passed. Shock is not usually present, and indeed until the duodenal leak in the retroperitoneum has reached significant proportions symptoms and signs are minimal.

The commonest symptom is abdominal pain, constant and of steadily increasing severity, usually in the right half of the abdomen and often going through to the back. Testicular pain has been described from irritation of the testicular nerves accompanying the spermatic arteries by the retroperitoneal exudate (Butler and Carlson, 1931). Vomiting may come late, and some cases have a minor haematemesis. A rise in temperature and pulse rate may not be evident early. Tenderness and guarding in the right hypochondrium are constant, and, later, rigidity and absent bowel sounds in a larger area indicate peritoneal involvement. Occasionally the crepitation of surgical emphysema in the abdominal or thoracic wall may be felt and pelvic crepitation on rectal examination has been described. There is no constant clinical picture, and only a vigilant awareness and a high "index of suspicion" of the condition enable an early diagnosis to be made. Ancillary investigations provide a useful guide, radiological examination being the most helpful. A plain *x*-ray film of the abdomen can be of the utmost value. This may reveal nothing abnormal, but more often shows free air in the retroperitoneum which may outline the right kidney or present a characteristic "bubble" effect. The retroperitoneal exudate usually obscures the right psoas shadow.

The white-cell count is moderately raised in the early stages and the serum amylase may be elevated from coincident pancreatic damage or even from the retroperitoneal duodenal collection.

TREATMENT

It is imperative to repair the duodenal tear as quickly as possible, and once the diagnosis has been established or even suspected the patient should be prepared for surgery after any blood loss or fluid and electrolytic imbalance has been corrected. Naso-gastric suction by a Ryle or other tube is mandatory. Thorough exploration of the abdomen is necessary, remembering that multiple lesions may be present and that intraperitoneal signs of duodenal rupture may be few or absent. The commonest are: a variable amount of serosanguineous free fluid; haematoma formation in the retroperitoneum or mesentery of the transverse colon or small bowel, sometimes tinged by bile; crepitation in the retroperitoneum or collection of air bubbles on the serosal aspect of the colon; and fat necrosis of retroperitoneal tissues. Any other lesions in the abdomen should be first searched for and remedied, and then the second part of the duodenum "Kocherized" by incising the peritoneum along its lateral avascular border and turning the bowel medially. If the region of the ampulla is involved the common duct should be identified, as it may need to be reimplanted (Welch, 1951).

The local treatment of the perforation will be to close it, if at all possible, without narrowing the lumen grossly. If stricture seems likely from this procedure the following measures have been adopted: (1) complete division of the duodenum and an end-to-end anastomosis; (2) complete division of the duodenum, closure of both ends, and gastrojejunostomy; and (3) closure of the distal duodenal stump and duodenojejunostomy. Drainage of the retroperitoneal area through a stab wound in the flank is the next most important step, as the retroperitoneum is peculiarly liable to prolonged phlegmonous suppuration—even with this precaution. Parenteral fluids, gastric suction, and antibiotics are continued until intestinal function is firmly established. In the convalescent phase a barium-meal examination is advisable to exclude stricture formation at the site of repair.

Complications include peritonitis, paralytic ileus, retroperitoneal cellulitis, external duodenal fistula, local or subphrenic abscess, stenosis of duodenum or bile ducts, duodenocolic fistula, pulmonary collapse or infection, and wound infection and dehiscence.

CASE REPORT

A 19-year-old soldier was admitted to a British military hospital in Germany on September 28, 1955. That afternoon, whilst manœuvring lorries in an enclosed space, he had been crushed between the back of one and the radiator of another. He had immediately experienced severe pain in the left loin and left side of the abdomen, which had continued unabated. He had not vomited. When seen he was groaning in pain. The pulse was 88, temperature normal, and B.P. 110/80. Tenderness and guarding were present mainly over the left side of the abdomen, and a large haematoma filled the left loin. An *x*-ray film showed fractures of the transverse processes of L 1–4 on the left side. Rectal examination was negative and normal urine had been passed since the accident. Bowel sounds were absent. His symptoms were thought to be due to his spinal injuries, and 100 mg. of pethidine was given. Two hours later the pain was still severe and there were generalized guarding and release tenderness, with rigidity maximal on the left side. An *x*-ray film of the chest was taken to show the diaphragms for the presence of free gas under the right cupola. This was absent, but the presence on the wet film of free gas around the right kidney, giving a characteristic "pneumogram," was overlooked. This was the most important single finding, as the other signs were not at all indicative of a right-sided lesion. A tentative diagnosis of retroperitoneal rupture of the *left* side of the colon was made and preparations for laparotomy were instituted after gastric suction and intravenous saline were set up.

Operation.—Under general anaesthesia a left paramedian para-umbilical incision was made. A small amount of bloody free fluid was present, but the left side of the colon was normal. A large retroperitoneal haematoma was visible, and palpable, around the right kidney and infiltrating the mesocolon and small bowel mesentery. The most striking feature was the presence of numerous beaded bubbles of air trapped between the serosal and muscular coats of the right half of the transverse colon, and there was a crepitant collection of air around the right kidney. For better exposure a transverse extension was made across the right rectus above the umbilicus, converting the wound to a T shape. The small bowel was intact and no other lesion was seen, so the right colon was reflected medially by incising the parietal peritoneum on its lateral aspect. Bile mixed with the blood was found in the haematoma and only then did the true diagnosis come to mind. On reflecting the second part of the duodenum medially, the bile was seen to be coming from a tear at the junction of the second and third parts of the duodenum which involved two-thirds of its circumference. The margins of the tear were swollen and oedematous. A repair was effected at right angles to the bowel axis with two layers of catgut, the first embracing all coats and the second in Lembert fashion. Owing to the size of the tear and the oedema around it the resultant repair caused some misgivings concerning the adequacy of the duodenal lumen. However, no trouble occurred postoperatively. A large drain was led down to the area retroperitoneally via a stab incision in the right flank, and the abdomen was closed in layers.

Post-operative Progress.—Bowel function was finally established on the sixth day, when gastric suction and intravenous alimentation were discontinued. Progress was good until the 14th day, when he coughed up some blood and had signs of a right lower lobe collapse, no doubt due to a pulmonary embolus, though there was never any calf tenderness. Pulmonary signs rapidly abated, but pyrexia continued and large amounts of pus were intermittently discharged from the drainage track in the right loin. The pus grew *E. coli*, and a course of chlortetracycline, to which it was sensitive, was given. A month after operation a tender indurated mass could be felt in the right iliac fossa, extending up towards the right kidney. The drainage track was dilated, some pus evacuated, and a rubber drain reinserted. However, the mass grew larger, and it was obvious this residual abscess would have to be evacuated more directly. On the 42nd day a gridiron type of incision was made under general anaesthesia in the right iliac fossa, and, keeping extraperitoneal, a loculated phlegmonous mass was found on the posterior abdominal wall. The original track was dilated and some pus evacuated from the perinephric region and drainage instituted from either end of the gridiron incision.

In two days he was afebrile, though the drains discharged for a further three weeks, and he rapidly gained weight and strength. Ten weeks after operation a barium-meal examination showed no hold-up at the site of repair, which could barely be detected. Eleven weeks after operation he returned to his unit on light duties. His original T-shaped laparotomy wound had healed excellently and there was no sign of weakness in it. Fourteen months later he was very well and had no digestive or abdominal symptoms (postal communication).

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