CONCLUSION

The results show a negligible difference between the trial and control groups in 32 patients. There is no reason to suppose that a larger sample will reveal a significantly greater difference. This means that the addition of 20 mg. of oxethazaine does not improve the capacity of an aluminium–magnesium hydroxide mixture to relieve the pain associated with a duodenal ulcer: mucaine cannot be recommended as an advance in the symptomatic treatment of this condition.

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Diabetic Coma Without Ketoacidosis in a Patient With Acute Pancreatitis

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The association of acute pancreatitis with diabetes mellitus has been commented upon many times since Harley (1862) and Atkinson (1895) described cases of acute pancreatitis with glycosuria. Schumaker (1940) reviewed 700 cases of acute pancreatitis and found glycosuria in 14%.

Diabetic acidosis with acute pancreatitis has been described on several occasions; Hughes (1961) found 57 cases in the literature and added one of his own. Diabetic coma in association with pancreatitis is also now a recognized clinical syndrome (Tully and Lowenthal, 1958).

Lucas *et al.* (1963) described two cases of coma due to uncontrolled diabetes mellitus with blood-sugar concentrations greater than 1,000 mg./100 ml., but without ketoacidosis. In neither of these patients was there any suggestion of acute pancreatitis, and both recovered. Sament and Schwartz (1957) and Iyengar (1961) have reported similar cases. The patient whose case is described below presented in hyperglycaemic coma without ketoacidosis and was found to have acute pancreatitis.

CASE REPORT

A 59-year-old woman who had been found unconscious in bed was admitted in coma to a medical ward in Aberdeen Royal Infirmary. One month before admission she had an attack of "influenza" from which she never fully recovered, continuing to complain of tiredness and malaise. Two days before admission she was confused and incontinent of urine. She had never complained of abdominal pain, there was no family history of diabetes mellitus, and, so far as is known, she had not been taking drugs.

The patient was comatose and did not respond to painful stimuli; there were no signs of loss of weight or oedema and no clinical evidence of dehydration—the tongue and mucous membranes were moist. Her temperature was 97.2° F. (36.2° C.), the pulse rate was 110, and the blood-pressure 110/70 mm. Hg. Respiration was shallow and rapid. The limbs were flaccid, with symmetrically diminished deep tendon reflexes; plantar reflexes were flexor. The right pupil was slightly larger than the left, but both reacted to light. No abnormality was found in chest and abdomen.

The urine contained sugar + + + + but no acetone. The blood sugar was 994 mg./100 ml. Serum electrolytes were: sodium 148, potassium 3.5, chloride 99, and bicarbonate 26.5 mEq/l., and the serum urea was 76 mg./100 ml. The electrocardiogram showed diffuse myocardial ischaemia.

Intravenous normal saline was given along with large doses of soluble insulin, both intravenously and intramuscularly. In six hours the blood sugar fell to 200 mg., but there was no improvement in the patient's general condition. Her urine now contained sugar + + + but no acetone. On 5 litres of intravenous fluid she maintained a good urine output. The blood-pressure and body temperature slowly fell, and she died 16 hours after admission.

At necropsy serosangineous fluid was found in the peritoneal cavity, and acute haemorrhagic inflammation of the whole of the pancreas. There were gross fatty changes in the liver. Histological examination also showed congestion of the lipid deprivation in the adrenals. There were some areas of fibrosis in the myocardium but no evidence of infarction. No abnormality was found in the brain or meninges.

DISCUSSION

It is well recognized that abdominal pain in patients with diabetic acidosis may be associated with pancreatitis (Warfield, 1927; Joslin, 1928; Hughes, 1961). It has been suggested that diabetic acidosis can precipitate pancreatitis rather than vice versa (Tully and Lowenthal, 1958), but the occurrence of severe acute pancreatitis and diabetic coma with no evidence of ketoacidosis has not, so far as can be ascertained, been previously reported. Lucas *et al.* (1963) attributed the coma in their patients to hyperosmolality and dehydration. In the present case the serum osmolality was not estimated, but there was no marked hypernatraemia and no clinical evidence of dehydration; this, of course, does not exclude cellular dehydration. With standard treatment the blood sugar fell more rapidly than is usual in diabetic coma. Tully and Lowenthal (1958) maintain that this feature, along with failure to respond clinically, should suggest acute pancreatitis.

The hypothermia was a terminal feature and developed only eight hours after admission, when the blood-sugar level had fallen to less than 200 mg.

The exact cause of death in this patient is not certain, but it is likely that the most important factor was severe acute pancreatitis. There is no evidence for or against the possibility that she had diabetes before developing pancreatitis.

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