It is concluded that carotid sinus epilepsy is one example of a wider pathological phenomenon whereby the stimulation of certain receptors which ordinarily give rise to cardio-inhibitory reaction may instead engender epileptic activity.

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TREATMENT OF ACUTE HAEMORRHAGIC PANCREATITIS WITH CORTISONE

BY

J. G. BROCKIS, Ch.M., F.R.C.S.
Senior Registrar

AND

E. T. JONES, B.Sc., M.B., B.Ch.
House-Surgeon

Cardiff Royal Infirmary

Amongst surgical emergencies, acute haemorrhagic pancreatitis remains therapeutically one of the most difficult. The mortality from this condition, including the many mild cases, is still in the region of 33% (Paxton and Payne, 1948). It is now generally agreed that, when a correct diagnosis can be made, it is better to avoid operation in the acute phase of the illness and to institute measures to combat dehydration and circulatory failure. These take the form of correction of protein, fluid, and electrolyte imbalance by transfusion. Gastric suction is also necessary when there is paralytic ileus. Analgesics are given to relieve the intense pain, together with antibiotics to combat infection and propantheline to diminish the amount of pancreatic secretion (Comfort, 1955). In spite of these measures a number of patients (about 11% according to Paxton and Payne) pass into a state of peripheral circulatory failure from which they do not recover.

With this in mind it was decided to try the effect of cortisone in preventing or treating this circulatory collapse. Only one case so treated appears to have been reported previously (Eskwith *et al.*, 1955), and the authors considered their result sufficiently striking to justify further trials. The following two cases are recorded as it seems that this method of combating the severe effects of haemorrhagic pancreatitis is promising.

Case 1

A married woman aged 64 was admitted to Cardiff Royal Infirmary on September 14, 1955, having seven days previously experienced severe mid-abdominal pain and vomiting. Two hours before her admission a particularly severe attack of upper abdominal pain occurred which was constant and did not radiate. She felt faint and lost consciousness for a short period. During the previous five years there had been at least five attacks of abdominal pain, each coming on suddenly with retching and vomiting; none was associated with jaundice.

The patient was found to be an obese woman, cold, clammy, and slightly cyanosed, and retching continuously. Pulse rate, 120 per minute; blood pressure, 230/130 mm. Hg. Tongue dry and coated. Lung fields clear and heart normal. There was gross abdominal distension, with maximal tenderness in the left hypochondrium. Both flanks were dull to percussion. Urine contained sugar, acetone bodies, and a trace of albumin. Serum amylase, 1,600+ Sömogyi units per 100 ml. A diagnosis of acute haemorrhagic pancreatitis was made.

She was immediately given pethidine, 100 mg., and atropine, 0.65 mg., followed by intravenous transfusion of plasma and normal saline containing aureomycin. By the following morning she was very much worse and desperately ill. Blood pressure had dropped to 100/70 mm.; pulse rate was 140, and respiration rate was 40 per minute. Cortisone, 150 mg. twice daily for three days, was prescribed, together with propantheline, 15 mg. twice daily. Intravenous administration of fluids containing aureomycin was continued. Staining in the left flank and around the umbilicus was then noted, and this persisted for three days. On September 16 there was some improvement, the pulse rate having fallen to 120 per minute, though the blood pressure was still low—140/70 mm. The treatment outlined above was continued. At this stage the serum electrolyte values were: Na 130, K 8.2, Cl 95 mEq per litre. Blood sugar, 130 mg. per 100 ml. The raised serum potassium level caused some anxiety, but, as the patient appeared to be better, no treatment for this was instituted. There was further improvement by the 17th, the pulse rate having fallen to 100 per minute, but a tinge of cyanosis still remained about the lips and the respiratory rate was

FIG. 1.—Case 1: Showing the dosage of cortisone and the effect on the course of the illness.

30 per minute. Both lung bases showed signs of congestion. Intravenous transfusion of fluids was discontinued, as water could now be taken by mouth. Progress was maintained on September 19 and the cortisone dosage was reduced. The propantheline was stopped altogether owing to severe dryness of the mouth. At this stage the serum electrolytes were: Na 135, K 7.2, Cl 101 mEq per litre. On September 21 the dose of cortisone was reduced to

75 mg. twice daily; the patient felt well, her only complaint being a dull ache in the left hypochondrium, and the blood pressure was normal. A week later a mass the size of a grapefruit, which was enlarging slowly, was palpable in the left lumbar region. This was incised and a drain inserted. A large collection of brownish fluid was evacuated together with portions of omentum showing fat necrosis. The wound did not heal, but continued to discharge a watery fluid. The patient became very apathetic when complete cortisone withdrawal was attempted, and further doses had to be given as there was also a relapse in her general condition with more abdominal pain. The second course of cortisone was discontinued after gradual reduction in dosage over 22 days. During the next six months there was continuous discharge from the sinus, but this gradually lessened and finally ceased. She still suffers from nausea, anorexia, and occasional abdominal pain.

The dosage of cortisone and its effect on the course of the illness are shown in Fig. 1.

Case 2

A married woman aged 74 was admitted to Cardiff Royal Infirmary on September 14, 1955, complaining of severe upper abdominal pain which had started 24 hours previously and radiated down both sides to the iliac fossae and to the back, with nausea and vomiting. The patient perspired freely and was very restless. During the preceding two years there had been several minor attacks of abdominal pain. Cholecystectomy had been performed eight years before for cholelithiasis. She was obviously in severe pain. Blood pressure, 170/90 mm. Hg; pulse rate 90 per minute. Heart sounds faint, but no murmurs heard. Lung fields clear to percussion and auscultation. Abdomen generally tender, with guarding in upper part, and bowel sounds absent.

The following morning the condition was little changed, though the temperature had risen to 102° F. (38.9° C.). Serum amylase, 800 Sömogyi units per 100 ml.; serum electrolytes—Na 130 and K 7.2 mEq per litre; blood sugar, 130 mg. per 100 ml. Urine contained traces of sugar and acetone. Discoloration of the skin in the left loin appearing 36 hours after admission confirmed the impression that the patient was suffering from acute pancreatitis.

Therapy started 18 hours after admission with 300 mg. cortisone and 1 g. aureomycin daily. Within 48 hours of starting treatment there was marked symptomatic improvement; the patient was sitting up in bed free from pain, and, though the abdomen was still tender, there was no abdominal rigidity. Cortisone, 150 mg. twice daily, was given for three days, and then the dose was slowly reduced over ten days and the treatment discontinued on the fourteenth day after admission. Aureomycin was stopped on the eighth day of the illness. The temperature range and the dosage of cortisone are shown in Fig. 2.

FIG. 2.—Case 2: Showing the dosage of cortisone with the range of temperature during treatment.

Convalescence was complicated by the development of an abscess in the left buttock, which responded to simple treatment. The patient was sent home feeling well after 35 days in hospital.

Discussion

The use of cortisone in acute pancreatitis was tried on a patient who appeared to be dying and had developed severe peripheral circulatory failure in spite of plasma and saline transfusion. The rapid improvement in the general condition and the relief of pain which began after the administration of cortisone were impressive. It is, of course, difficult to be certain to what extent other factors have contributed to this recovery, as, for obvious reasons, the patient also received intravenous fluids, propantheline, and antibiotics. An indication that the cortisone had aided recovery was, however, given by the relapse which occurred when the drug was stopped on the twentieth day of the illness. As soon as cortisone was again taken, the patient improved. That this also occurred in the case of Eskwith *et al.* is of some interest. Some authorities maintain that in the presence of infection cortisone is dangerous, but Kinsell (1954) stated that he had been using the drug in combination with an antibiotic in cases of generalized peritonitis and pyogenic meningitis with good results over a period of four years. He claimed that the non-specific antitoxic and antishock effects of cortisone tided the patient over until antibiotics had a chance to take effect. It is probable that the cause of collapse in acute pancreatitis is to be found in toxic products liberated by enzyme action rather than by infection, for it has not proved possible to culture organisms from the blood stream in patients suffering from this disease (Dragstedt, 1943). On the other hand, it has proved equally difficult to isolate the toxic products of autodigestion, which are thought to cause the circulatory failure. Plasma and plasma protein fractions have been said to be specific antidotes to this circulatory collapse, but the work of Elliott (1955) and of Pollock (1956) suggests that the value of plasma in this condition may be a non-specific one in providing additional circulating fluid and protein.

The second patient did not have such severe pancreatitis as the first, and, although the response to cortisone seemed to be equally dramatic, other factors may have been important in her rapid recovery.

The place of cortisone probably lies in the control of the severe attack of the disease where there is evidence of profound circulatory disturbance. Its administration should be stopped at the earliest possible moment, as an infection may spread rapidly in a patient under its influence.

Summary

Two patients suffering from pancreatitis of some severity were treated with cortisone. The results suggest that further investigation into this line of therapy is justified, and that the place of the drug may be in the control of the early stages of the fulminating disease.

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