Pancreatic Society of Great Britain and Ireland

Abstracts of papers presented at the Inaugural Meeting

The Inaugural Meeting of the Pancreatic Society of Great Britain and Ireland was held on 16th June 1975 at the Royal College of Surgeons of England. Sir Rodney Smith KBE PRCS *was elected the first President of the society. Other officers elected at the meeting included Mr Michael Knight (Secretary/Treasurer), Professor Henry Howat, Professor Patrick Collins, Mr Peter Bevan, Dr Martin Sarner, and Mr John Trapnell (Committee). Distinguished guests present at the meeting who received Honorary Membership of the society included Dr Kenneth Warren (USA), Dr Solly Marks (South Africa), Professor Maurice Mercadier (France), Professor Walter Hess (Switzerland), and Professor Waller Ballinger (USA).*

The declared aim of the new society is the study of diseases of the pancreas, and scientific meetings will be held annually at the centre of the elected President. The next meeting of the Society will be in Birmingham on 12th July 1976 with Mr Peter Bevan in the chair.

All enquiries concerning the society (and requests for reprints) should be addressed to Mr Michael Knight MS FRCS, *Royal Hampshire County Hospital, Romsey Road, Winchester, Hants.*

ARTERIAL HYPOXIA IN ACUTE PANCREATITIS

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Some 15–20% of patients with acute pancreatitis develop a basal pleural effusion, usually on the left side, but a much higher proportion develop a surprising degree of arterial hypoxia, frequently without overt signs of respiratory embarrassment. Of 84 patients studied by serial arterial blood gas monitoring, 45% developed arterial Po_2 levels below 8 kPa (60 mm Hg) at some time during the first week of illness. Most patients had a satisfactory level of arterial Po_2 on admission, but a steady fall usually occurred within the first few days in hospital. 'Trough' levels tended to occur from the 2nd to 4th day after admission, and such arterial hypoxia may represent a life-threatening aspect of the disease in itself[1].

Fifty-seven patients (80%) developed arterial Po_2 levels below 9.3 kPa (70 mm Hg) and all 6 deaths occurred in this group. It is therefore possible that the degree of arterial hypoxia is of prognostic importance. All the above measurements were made breathing air. However, humidified oxygen was later provided for all older patients (over 60 years) and for the younger patients developing arterial hypoxia (Po_2 <9.3 kPa (70 mm Hg)). This reversed the arterial hypoxia in all patients in this series and the introduction of supplementary therapy has been associated with a downward trend in the mortality rate for the high-risk older patients, which was 27% in the early part of the study, with no supplementary oxygen, but only 12% in the group provided with humidified oxygen.

Preliminary studies into the mechanism of this arterial hypoxia were carried out by means of much more elaborate respiratory function tests on 16 of the 84 patients. Daily chest X-rays were performed and revealed abnormalities in 7 (43%) of this small group, the abnormalities being pleural effusions and atelectasis with or without diaphragmatic elevation. Gas exchange studies were also performed on 13 of this subgroup and a significant ab-

normality detected in 12. The abnormality reflected a right-to-left shunt of between 5 and 15% of the total cardiac output (normal maximum shunt 0–5%). Concentrations of fibrin degradation products in the blood have also been measured in this group, and 15 of the 16 patients had abnormal levels (normal 0–5 mg/l).

[1] Ranson, J H C, Roses, D F, and Fink, S D (1973) *Annals of Surgery, 178,* 75.

OBSERVATIONS ON ACUTE PANCREATITIS

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Bristol was one of several centres invited by the Medical Research Council to participate in a double-blind trial of glucagon and aprotinin (Trasylol) in the treatment of acute pancreatitis. Thirty-seven patients have been admitted to the trial in the 6 months since its inception in November 1974, which is about twice the anticipated number. Their clinical and biochemical progress has been closely monitored by the same observer, but it is not yet known whether they have received glucagon, aprotinin, or placebo.

Thirty-five patients were admitted to the trial on the basis of a serum amylase estimation in excess of 2000 IU Phadebas (corresponding to 1000 Somogyi units). Two patients with a value below this level but with unequivocal evidence of pancreatitis at laparotomy were admitted. One patient with a high serum amylase level was subsequently withdrawn from the trial after laparotomy had shown biliary peritonitis but a normal pancreas.

Six patients were judged on admission to to have a severe attack with evidence of shock, and 1 of these died. Two other deaths have occurred from the disease in the series to date, an overall mortality of 8%. One or more complications occurred in 35%, and 3 patients had an early relapse after discharge. Gallstones have been particularly prevalent, occurring in 63% of the patients so far studied. The widely varying clinical profile of acute pancreatitis and the low overall mortality to date emphasize the need for a large number of patients to be studied if the objectives of the trial are to be attained.

EFFECT OF GLUCAGON ON PAIN IN ACUTE PANCREATITIS

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Patients with acute pancreatitis, as indicated by a consistent clinical picture with a raised serum amylase (> 1000 Somogyi units) within 72 h of the onset of the attack, were treated with intravenous glucagon. On diagnosis the patients allotted a score to the severity of their pain based on the following analogue:

10/10	Unbearable agony
8/10	Severe but not unbearable pain
6/10	Pain necessitating staying in bed and preventing sleep
4/10	Pain necessitating reduction of normal activities but not mobility or sleep—'aspirin level'
2/10	Pain not requiring reduction in normal activities
0/10	Pain-free

After administration of glucagon the pain was assessed by direct questioning at 4 h using the same analogue.

Thirty patients were admitted to the trial. Twenty-one achieved complete relief of pain. A further 6 patients achieved a reduction in the intensity of their pain by 50% or more. Formal analgesia was found necessary in only 8 cases.

These results are significant when the aetiology of the pancreatitis in the patients admitted to the trial is considered. Complete pain relief was achieved in 17 out of 20 cases secondary to biliary tract disease but in only 2 out of 6 cases secondary to chronic alcoholism. This suggests that glucagon is less effective in relieving the pain in alcoholic pancreatitis.