Great expectations from proximal vagotomy

Progress in the surgical treatment of duodenal ulcer has been punctuated by excitements and disappointments. Almost every new procedure has aroused great expectations, with very favourable results reported in selected series of patients; but it is rare for the optimism to be maintained.

In recent years a new star has appeared: proximal gastric vagotomy. Considerable claims have been made, and are being made, for the superiority of this operation—but older surgeons, who have lived through several changes of fashion, will require convincing evidence before they abandon any procedure which in their own hands yields good results. In 1968 the Leeds trial1 of the then conventional methods of surgical treatment showed that very careful objective assessment was required before any definite conclusions could be reached about the results of surgery for duodenal ulcer. The reporting of the results obtained by using a particular operation in selected patients may produce both biased and erroneous ideas of the value of that procedure.

Three criteria may be used to judge the results of peptic ulcer surgery: firstly, the risk to life of effective surgical treatment; secondly, the incidence of residual gastrointestinal symptoms; and, thirdly, the risk of late nutritional deficiencies. The risk of effective surgical treatment includes not only the risk of the primary operation but also the recurrent ulcer rate. If a substantial number of patients require a second operation to control their ulcer diathesis then the morbidity and mortality rate of the second procedure has to be included in the reckoning.

The immediate risk of proximal vagotomy is low (0·3% in a collected series8 of over 5000 operations from 40 centres). One or two instances of necrosis of the lesser curve of the stomach have been reported, and this seems to be a rare but definite complication peculiar to this operation. The incidence of recurrent ulceration is, however, more difficult to assess. In most series it is low8·4 (less than 5%) but there have been two small series5·6 with recurrent ulcer rates of over 20%. Recurrent ulceration is almost certainly related to the effectiveness with which acid secretion is controlled. After proximal gastric vagotomy, secretion tests show an initial reduction of acid comparable with other types of vagotomy, but over the first year after operation a distinct rise is usually observed in basal, maximal, and insulin-stimulated secretion. Recently the results have been published7 of acid secretion tests in 21 patients measured at least five years after proximal vagotomy: basal secretion was still reduced by 79% and peak acid responses to pentagastrin by 48%. It seems, then, that after the slight rebound which occurs within the first year the operation produces a stable reduction of acid secretion by the stomach.

The incidence of postoperative gastrointestinal disturbance is minimal. Many of the common disturbances seen after gastric surgery such as dumping and bile vomiting are almost certainly related to the bypass, destruction, or resection of the normal mechanisms of gastric emptying. As the disturbance of gastric emptying is made minimal in proximal gastric vagotomy such symptoms should be uncommon. This is confirmed by most of the published series. Particularly important are the results of a controlled trial reported4 from Belfast, in which proximal vagotomy in 50 patients produced statistically better symptomatic results than selective vagotomy and gastroenterostomy in a comparable group of 50 patients.
Management of acute pancreatitis

Acute pancreatitis is a relatively uncommon disease in Britain, affecting about 50 persons per million of the population annually.1 The clinical course is notoriously unpredictable; both the variability in the criteria for diagnosis and the difficulty in establishing the severity of the disease have made it hard to conduct satisfactory clinical trials of management and to compare data from different units. Furthermore, while biliary tract disease and to a less extent alcohol are implicated in many cases, the aetiology remains not clear in at least one-third of the patients in Britain. Perhaps it is not surprising that mortality figures3 range from 6% to 30%.4

The basis of the management of a patient with acute pancreatitis remains adequate analgesia, nasogastric suction, and prompt and complete replacement of fluid and electrolytes. In severe pancreatitis the plasma volume may be depleted by 30–40%.5 The aim should be to achieve a minimum urinary flow of 30 ml an hour.6 Some patients require replacement of calcium and magnesium ions, and vitamin replacement is probably useful in alcoholics. Blood and albumin are necessary in seriously ill individuals. The benefits of anticholinergic and antibiotic agents are less well established. Trappell7 has questioned the value of routine antibiotic treatment, claiming that it does not prevent pancreatic abscesses, presumably the reason for initiating the treatment. Nasogastric suction is said to serve two purposes—to manage intestinal ileus and to reduce pancreatic stimulation—but the success of the latter aim is uncertain. It would be comforting to believe that anticholinergic therapy was more effective in reducing pancreatic secretion. Unfortunately this is not so, and the use of anticholinergic agents is as controversial in acute pancreatitis as in so many other gastrointestinal disorders. What is certain is the potential hazard of ileus, urinary retention, and tachycardia.8 The more frequent use of peritoneal dialysis, recognition of diffuse intravascular coagulation, and increased awareness of the syndrome of respiratory insufficiency requiring the early use of mechanical ventilation9 have added to effective medical care.

Treatment has been enhanced and at the same time complicated by the introduction of three allegedly specific regimens. We just do not know if they are effective. The oldest of these is the proteolytic enzyme inhibitor aprotinin (Trasylol), and there continues to be a lively debate over its therapeutic benefit. The majority opinion has been that it is ineffective,10 but a recent controlled trial11 on 105 patients gave promising results when large doses were used. Glucagon suppresses pancreatic secretion and increases splanchnic blood flow. Uncontrolled observations suggested that glucagon therapy was safe and induced relief of symptoms in pancreatitis.12 A study13 in rats failed to show any benefit from its use, but the difficulty of making analogies between animal studies of pancreatitis and human disease is well recognised. There is an urgent need for properly conducted clinical trials, and we have been told that the answer may well be forthcoming.

The third regimen to excite interest is that of glucose and insulin infusion. The rationale behind this treatment is the concept that in acute pancreatitis activation of a hormone-sensitive lipase present in abdominal adipose cells causes fat necrosis with ensuing abdominal pain and tenderness. Hallberg and Theve14 introduced treatment with 20 units of soluble insulin per litre of 5–10% glucose and 80 mmol sodium infused at a rate of 300–400 ml during the first hour with a total of 2 litres over 24 hours. The authors claimed rapid clinical improvement. Svensson’s small double-blind controlled trial15 of insulin and glucose showed that there was benefit during the initial 4–15 hours of treatment but that thereafter the patients were not at an advantage. This is another drug combination which requires evaluation by a larger study.

The role of early operation in acute pancreatitis is also controversial. In Britain most surgeons favour a nonoperative approach unless the diagnosis is uncertain, when all would agree that a laparotomy is mandatory. None the less claims have been made for early surgical intervention and drainage of the pancreatic bed or removal of gallstones and biliary tract exploration. Major pancreatic resections have been attempted, and even total pancreatectomy has been advocated for fulminant haemorrhagic pancreatitis: but these are hazardous procedures in critically ill patients and not surprisingly carry a high mortality rate. Recent studies suggest that early operative management has no advantages in acute pancreatitis.16

3 Olen, H, American Journal of Digestive Disease, 1974, 19, 1077.
9 Parkash, O, Digestion, 1972, 6, 215.
13 Wellbourn, R B, and Cox, A G, British Medical Journal, 1974, 1, 244.