Surgery for Duodenal Ulcer

Sir,—Your leading article on results of vagotomy (15 August, p. 358) offers little advance on the conclusions of six years ago.1 The problem here is one of my own cases of peptic ulcer may therefore be of interest.

Since 1967 I have treated, by pyloroplasty alone, 55 patients suffering from duodenal ulcer, entering into the stomach (38 duodenal, 17 gastric). Thirty-seven patients have been followed for more than one year and 15 of these for more than two years. None now suffers any dyspepsia; nor have there been any side effects of the pyloroplasty. A second pyloroplasty was necessary in five patients before the ulcer healed. The first operation did not produce a viable, incompetent pylorus, but after the second and effective pyloroplasty the ulcers healed and remain so 20 months (one duodenal), one year (one duodenal, one gastric), and less than one year (one duodenal, three gastric) 181.

In seven patients eating excessive fat caused symptoms which are fully controlled by low-insulin intake.

Cancer2 showed that benign gastric ulcer healed following pyloroplasty alone "because a wide, incompetent pylorus does not permit a narrow, forceful regurgitation of duodenal contents into the stomach. Caryer's words—narrow, forceful—led me to apply his conclusions to duodenal ulcer as well. Farris and Smith3 accepted that cure of anatomical obstruction at the pylorus was the most important factor in cure of gastric ulcer but added vagotomy to pyloroplasty for fear that "duodenal ulcer diathesis might recur. Their fears appear to be unfounded. Majowicz and Zacek4 in 1928 considered the failure of cure by pyloroplasty of any peptic ulcer was due to incomplete abolition of sphincter activity at the pylorus, or to postoperative narrowing of the pyloroplasty. Their views were neglected perhaps because although we can assess the time the stomach takes to empty of barium we cannot see whether it is "swishing like a bath of water through a relaxed, wide-open pylorus, or is spurring as a narrow, forceful "jet" through a restricted pylorus (restricted by spasm, muscle hypertrophy, inflammation and fibrosis, or even pyloric mucous diaphragm). Yet, it is possible, also, that it was possible to "measure" them, however inaccurately, acid levels attained spurious importance.

The concept underlying use of pyloroplasty alone is a simple one. If the pylorus does not relax fully the flow becomes narrow and forceful; acid gastric contents are squirted as a "jet" to impinge upon duodenal mucosa, possibly persistently on bile duct area, before mingling with and being neutralized by duodenal contents. If the pylorus does not close completely during duodenal cap systole pyloric reflex occurs. Resistance to flow of gastric contents through the pylorus into the duodenum may stimulate the antrum and cause a rise in gastric acid secretion.4 Anything which mimics a "jet" itself (for example, by interfering with gastric motility), or which alters pH, can modify the ulcerating effect. Pyloroplasty prevents the formation of a "jet".

Pyloroplasty must be adequate to prevent reflux from duodenal cap systole and to decompress the antrum. It is then followed by healing of duodenal and high and low gastric ulcers, high acid secretion levels may become normal, and there are no side effects of the operation. I have not found any kind of vagotomy or gastrectomy necessary to cure any chronic peptic ulcer during the last two-and-a-half years. I have also used effective pyloroplasty alone successfully to cure cases of failed vagotomy and pyloroplasty and failed vagotomy and gastrectomy. —I am, etc.,

Peter ChilDS.

Plymouth General Hospital, Plymouth, Devon.

REFERENCES


Pseudo-obstruction of the Large Bowel

Sir,—I am surprised that Dr. Barbara F. Smith (20 June, p. 732) and Mr. J. A. C. Neely (27 June, p. 793) have objected to the use by Mr. P. K. Caves and Dr. H. A. Crockett (6 June, p. 383) of the term "pseudo-obstruction". Dr. Smith's objection is that "pseudo-obstruction" was used to describe another condition of apparent obstruction by Nash and his colleagues in 1960. I would direct attention to the paper by Dudley et al. in 19587 to describe the condition reviewed by Mr. Caves and Dr. Crockett.

Mr. Neely states that the ileus should be used rather than obstruction, but the word ileus comes from the Greek 7eluo which means "intestinal obstruction." He would qualify the ileus as "paralytic," yet refers to a condition with features not of paralysis but of disorganized or ineffective function (the presence of bowel sounds associated with colic). He believes that the obstruction is not "pseudo" but is real, yet agrees that the tube is not blocked, but has an inefficient propulsive mechanism, and so appears with most features of blockage or organic obstruction.

To me, the term "pseudo-obstruction" describes the occurrence distinctly and effectively. If adopted universally it would make for clarity in terminology, and even improvement in its understanding.—I am, etc.,

F. O. STEPHENS.

Sydney Hospital, N.S.W., Australia.

REFERENCE


Polypuria after Cardiac Surgery

Sir,—We were interested to read the report by Dr. R. O. Robinson and Mr. K. M. Pagel (22 August) of a patient with polypuria after cardiac surgery. Two patients in the Liverpool Regional Cardiothoracic Centre undergoing open heart surgery developed polypuria postoperatively, and in one there was a dramatic response to pitressin.

A 34-year-old housewife with a 27-year history of a cardiac murmur presented with an 18-month history of dyspnoea on exertion and haemoptysis. Physical examination revealed an atrial septal defect and the E.C.G. showed a right bundle branch block. At cardiac catheterization a large atrial septal defect was found with a 1:5-1:1 shunt. At operation a 15 sq. cm. atrial septal defect was repaired using a Dacron patch on full dopamine for investigation of breathlessness with a perfusion time of 45 minutes. The urine output was adequate, glycosuria was present, but blood sugar was normal. The next day she was vague and had polyuria, but there were no other abnormal neurological features. A brisk diuresis started with a urine flow of 9 ml/hour for eight hours and then rose to normal. Twelve hours later it again increased to 8 ml/min. and, after 11 hours she had a major generalised convulsion while intubated; the tube was curarized, intubated, and put on a ventilator. The urine flow then returned to normal. A cardiac arrest developed from unknown causes and following successful resuscitation she was found to have bilateral pyramidal damage and was comatose. Her cardiac, renal, and cerebral function did not alter during the next 10 days, when she succumbed to an overwhelming bronchopneumonia. Necropsy revealed petechial haemorrhages throughout the cerebral white matter but no softening or oedema.

A 39-year-old housewife, who had had a mitral valvotomy 14 years previously, was admitted to hospital on 10 April 1922 for investigation of breathlessness. Physical examination revealed a pure mitral stenosis and valvotomy was recommended. A left-sided peripheral pulse had complicated the original operation and a sinus had drained intermittently since.

An attempt to perform a mitral valvotomy might have been attempted on 12 April with a good fortune on the patient to cut to cardiopulmonary bypass and the mitral valve split to 3-5 cm. with a Tubb's dilator. There was sub-eclampsia, elevation of atrial pressure, and the aorta was grossly dilated during the period of left atriotomy. She did not regain consciousness after the operation and was ventilated. Neurological examination revealed bilateral pyramidal damage. She required an isoprenaline infusion for bradydysrhythmias and hypotension. In the 12-hour period postoperatively she passed 1-6 l. of urine in excess of input. The blood urea, calcium, potassium, and magnesium were all normal. The blood sugar, initially 325 mg./100 ml. fell to 124 mg./100 ml. within 6 hours. The following day she became hypothermic and the diuresis persisted, urine S.G. 1000. Ten units of insulin and 150 mg. of hydrocortisone were given subcutaneously and the urine flow fell from 7-0 ml/min. to 0-75 ml/min. The urine S.G. fell to 10.

Fifteen hours later, with a normal urine output, the ventilator failed. When she was put back on another machine the tidal volume was smaller, she showed a hypotension, a tachycardia, and the urine flow ceased. She was found to be acidic, hypoxic, and hypercapneic. Restoration of the cardiac ventilatory function did not improve either cardiac or renal function and she died 48 hours after surgery. Necropsy was not performed.

In the second case diffuse cerebral damage was recognized before the onset of polypuria, and hypovolaemia, suggesting a disturbance of hypophysal