hospices.\(^7\) Heroin is still used: its great solubility allows large doses to be given in small volumes, which is of great value in the patient who has lost much flesh. The long half life of methadone increases the risk of accumulation, but given night and morning it produces good background control.

If analgesics are ineffective, destruction of an appropriate neural pathway can provide quick relief of pain. The effect lasts for a variable period—often long enough in patients with malignant pain to cover all or most of their remaining life. The need for the procedure to be repeated makes this approach unsuitable for most patients with chronic benign pain.

Most pain-relief clinics start by reviewing, firstly, the diagnosis and then the drugs used, their dose, and their frequency. Often a psychiatric opinion follows, and, lastly, other treatment is considered. Among the specialised techniques available are destructive methods including coeliac plexus block for upper abdominal pain\(^8\); subarachnoid phenol for pain in limited spinal dermatome distribution or in perineal pain\(^9\); percutaneous cervical cordotomy\(^10\) in unilateral pain below the C5 dermatome; and pituitary injection of alcohol\(^11\) for cancer pain in any distribution. Benign pain is treated by drugs and non-destructive methods such as peripheral nerve stimulation\(^12\) \(^13\); dorsal column stimulation,\(^14\) useful in phantom limb pain or in arachnoiditis; and acupuncture,\(^15\) which is another simple stimulation technique with a low (but useful) success rate. Finally, there are biofeedback methods, relaxation techniques,\(^16\) operant conditioning,\(^17\) counselling, and other psychiatric methods.

When all these methods have been tried there will still be failures. Such patients should not be turned away without any explanation. Someone must be delegated to spend time talking to the patient before he or she leaves hospital. In the large pain-relief clinic at Walton Hospital, Liverpool, patients of this type are asked at the very least to write in once a year to report what has happened to them, to remind the clinic of their existence, and to ask if there is anything new for their pain. Getting patients to write in ensures two things. Firstly, they will not forget to write—while the hospital might—and, secondly, all patients who write in get an answer (and sometimes a recall). They feel that they are still connected to the hospital and can contact it if their condition deteriorates unexpectedly.

Many years ago, Szasz put the whole problem succinctly: "What is the physician's task in this situation? Whose pain should he control: The patient's? That of his relatives, tortured by the patient's complaints? Or his own, generated by his inability to help the patient?"

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After gastrectomy

In 1885 Billroth removed an antral tumour, closed the cut end of the stomach and the duodenum, and performed an anterior gastrojejunostomy.\(^1\) Since then all procedures that divert bile and pancreatic secretions across the gastric remnant tend to be labelled "Billroth II" to distinguish them from his earlier operation (1881) of gastroduodenostomy. If eponyms are to be used accurately, however, the operation, still widely practised, that dominated the surgery of duodenal ulcer from the late 1930s to the late 1960s—anastomosis of the full width of the open end of the stomach to the jejunum—should be termed the Kronlein procedure.\(^2\) The third name applied to the operation, Polya, is familiar in Britain because of Polya's persuasive writing\(^3\) more than 20 years after Kronlein's paper. Whether termed Billroth II, Kronlein, or Polya, these procedures are highly effective in curing duodenal ulcer. They do so by excluding the ulcer-bearing area, by removing the gastrin-secreting antrum (though its importance in the pathogenesis of duodenal ulcer in man is doubtful), by partially removing acid-secretory cells in the body of the stomach, and by continually bathing those that remain in alkaline secretions. The long-term price for this freedom from recurrence is a small gastric remnant without a sphincter to control its emptying; this may be associated with a complex of vasomotor and other symptoms, generally called "dumping of whose cause is still not entirely clear. A second drawback is the effect of bile on the stomach, which in around 2% of patients causes bilious vomiting. If this occurs it usually appears early, and in persistent cases can nearly always be cured either by transposing the inflow of bile further down the small gut or by a return to normal continuity with the stomach distanced from the duodenum by a jejunal interposition.

Of possibly greater importance is the prolonged contact of bile with the stomach stump, which leads in time almost invariably either to chronic superficial gastritis or to its successor atrophic gastritis.\(^4\) Gastritis merges into intestinal metaplasia, which some authorities\(^5\) believe is a precancerous lesion, though others doubt that the metaplasia is a predictor of cancer.\(^6\) Certainly in many series there has been an increased incidence of gastric cancer after gastrectomy (especially with gastrojejunostomy), including such surgical horror stories\(^7\) as the four patients with cancer and three with severe dysplasia out of 108 who had undergone routine gastroscopy more than 20 years previously. A further 12 patients were diagnosed as having moderate dysplasia and no fewer than 52 as having
slight dysplasia. More recently Skarstein and his colleagues carried out a similar cohort study of patients 12 years after surgery and found a high incidence of chronic superficial gastritis—though (perhaps because of shorter follow-up) the changes were for the most part mild and dysplasia was found in only 15% of biopsy specimens. This study also examined late deaths and the clinical state of the survivors. The death rate for patients treated surgically for peptic ulcer is 40%, greater than that of the general population. Whether this is the consequence of the diathesis, of surgery, or of risk factors such as tobacco and alcohol consumption remains uncertain. The health of the survivors is consistent in long-term follow-up studies: around 80% of patients are symptom-free—though some may have subclinical deficiency of iron or vitamin B12 and have some degree of anaemia—and 9% will have mild gastrointestinal symptoms, 7% moderate symptoms, and 4% severe problems. Since similar results are found at earlier follow-up assessments the functional results seem to be stable.

How much might have been done to reduce this moderate and perhaps reasonably acceptable morbidity? Some of the patients will have been ill selected and merely persist in their preoperative state; but selection is difficult, as every surgical gastroenterologist knows. Careful studies of dietary intake have shown that some of the poor results in terms of weight loss and anaemia are due to dietary factors and can be reversed. Armed with such knowledge surgeons might now hope to achieve better results with partial gastrectomy; in practice the decline in incidence of ulcer, the use of H2-antagonists such as cimetidine, and—above all—the development of vagotomy have made the question largely hypothetical.

The crucial problem now is whether malignant change in the stomach is really going to affect 5%, or more of the survivors of the gastrectomy bonanza of the 1950s and 1960s. Assuredly there is no ground for complacency. The only hope of curing this lesion lies in detecting it at an early phase of mucosal infiltration. The poor correlation of gastritis with symptoms makes routine endoscopy essential for patients who have survived 20 years; and that requires suitable populations, good records, and a determination to maintain follow-up. Can we meet such requirements, and if so can we afford to take the necessary next step of attempting to carry out endoscopy on what will shortly be many thousands of patients?

Surgeons, like cooks, know they cannot make omelettes without breaking eggs. Most of their time they are worried about early catastrophe—death and immediate serious complications. The low mortality and morbidity associated with much modern surgery should now be changing attitudes: firstly, surgeons should reset their time scales to think forward to much longer-term effects; and, secondly, they will need to devise avoiding actions, either by developing operations that should be less harmful in the long run or by making sure that long-term surveillance is adequate. The history of partial gastrectomy for duodenal ulcer is rapidly emerging as a model for good surgical behaviour on both counts.

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Adult polycystic disease of the kidneys

Inherited autosomal dominant adult polycystic disease of the kidneys is characterised by the development of multiple renal cysts. It frequently leads to end-stage kidney failure in the fourth decade and is the most common inherited kidney disease. Dalgaard estimated the morbidity risk up to the age of 80 as amounting to 80 to 90 cases per 100 000 population; the condition accounts for 7% of patients with end-stage renal disease. Whereas adult polycystic disease of the kidneys may remain symptomless, its presentations are many and varied. They include high blood pressure, recurrent urinary tract infections, pain and a mass in one or both loins, renal colic due to the passage of clots or stones, symptoms of kidney failure, and subarachnoid haemorrhage from a ruptured berry aneurysm—an abnormality commonly associated with adult polycystic disease of the kidneys.

Identification of the disease depends on excretion urography and ultrasound examination. Neither of these are sensitive, and cysts measuring less than 1-5 cm diameter may not be detected. Even with the use of computed tomography cysts measuring less than 0.5 cm diameter may be missed. A clean bill of health should not, therefore, be given to relatives of affected persons much before the age of 30—which is, unfortunately, well into the reproductive period. This makes for difficulties with genetic counselling and in choosing suitable live kidney donors from among the relatives of patients with kidney failure due to adult polycystic disease of the kidneys. More sensitive methods for early diagnosis are urgently required.

Milotinovic and his colleagues in Seattle recently performed renal biopsies on 16 asymptomatic relatives of patients with adult polycystic disease of the kidneys who belonged to five different families. Fourteen of the 16 had normal excretion urograms at the time of the biopsy, and in four adult polycystic disease of the kidneys had "developed" three years later. In three of these four people the initial biopsy specimen had shown dilatation of the distal and collecting tubules and splitting of glomerular and tubular basement membranes; these histological features may prove to be the earliest markers of the disease in carriers of adult polycystic disease of the kidneys. The Seattle group did not suggest renal biopsy as a routine method for the early detection of carriers because the histological changes are not uniformly distributed. Their observations may, however, throw some new light on the pathogenesis of the disease.

Past theories have included intratubular renal infection accompanied by tubular obstruction and non-union of the branches of the ureteric bud with the nephrogenic blastema, neither of which has been verified. Microdissection has shown that cysts communicate with the drainage system and no