lying sense of unease throughout the debate. This no doubt reflected the Council's concern about the impact of this legislation on the future of medical practice. Though it was not the Government's intention, this Act for reforming industrial relations could prove a watershed in the affairs of many professions.

Growth Hormone and Diabetes

Glucose homeostasis is partly governed by the hormonal effects of insulin and growth hormone. Insulin is thought to promote the entry of glucose into muscle and fat, and it also facilitates glycogen synthesis in liver and muscle. Growth hormone probably regulates the utilization of intracellular glucose and by this means alters sensitivity to insulin.

In 1968 J. Bornstein and his colleagues from Melbourne described two polypeptides prepared from growth hormone by hydrolysis which by their specific actions on certain enzymes of the glycolytic pathway appeared capable of accounting for both the early hypoglycaemic and the later hyperglycaemic actions of growth hormone. The first of these fractions, Bornstein named acceleratory polypeptide growth hormone (A.C.G.) and the second inhibitory polypeptide growth hormone (I.N.G.) All the known actions of growth hormone on carbohydrate and fat metabolism may be accounted for by the action of these two polypeptides. I.N.G. inhibits both glycogenesis and fat synthesis and accelerates breakdown of fat. A.C.G. causes hyperglycaemia and reverses the inhibitions produced by I.N.G.

When the acceleratory polypeptide was given to five diabetic patients, from whom insulin treatment had been withdrawn there was a fall in blood sugar, sustained up to 100 min. This effect of A.C.G. may be due to its own intrinsic action or to an increase in insulin sensitivity. To elucidate this further A.C.G. was given with a small amount of insulin (1-4-1-6 units) to fasting normal subjects, and there was a more profound and prolonged fall in blood sugar than when the same amount of insulin was given alone. Since these experiments were done on fasting persons, growth hormone levels and hence I.N.G. levels would be increased, and thus the effect of the acceleratory polypeptide in reversing I.N.G. inhibition might be more readily seen. This increased insulin sensitivity produced by A.C.G. raises the possibility of its therapeutic role in treating patients with insulin-resistant diabetes—for example, patients with resistant ketoadicosis.

Since the inhibitory polypeptide inhibits glycogenesis, it is important to find out if it is in the plasma of diabetic patients. In the latest communication from the department of biochemistry at Monash University, Professor Bornstein and his colleagues report at page 203 of the B.M.J. this week that they estimated I.N.G. activity in extracts of the plasma of nine juvenile-onset diabetics, 26 maturity-onset diabetics, 3 patients with pancreatic diabetes, and 4 hypophysectomized diabetics, and compared the results with those obtained from 16 normal persons. Since I.N.G. is thought to act by inhibiting triosephosphate dehydrogenase, the results were expressed as percentage inhibition of this enzyme.

Plasma extracts from both juvenile- and maturity-onset diabetics produced a greater inhibition than extracts of normal plasma. The greatest inhibition was seen in plasma from patients on insulin therapy and was less in those treated with tolbutamide. Plasma extracts of hypophysectomized diabetics, on the other hand, had virtually no inhibitory activity at all. That this effect was due to I.N.G. as previously described was suggested by three pieces of evidence. Firstly, the inhibitory fraction prepared from these plasma samples corresponded chromatographically to I.N.G. prepared by hydrolysis of human growth hormone. Secondly, the same enzymes were inhibited by the plasma extracts and by prepared I.N.G. Thirdly, A.C.G. produced partial reversal of the inhibitory effect of the plasma extracts, as would be expected if I.N.G. was the active component of the plasma extracts. When I.N.G. levels were compared during glucose tolerance tests in four normal persons and four patients with maturity-onset diabetes, administration of glucose produced a fall in the inhibitory polypeptide in the normal persons, whereas in the diabetics the concentration of the inhibitory material rose. This suggests that the polypeptide may have some role in controlling the level of blood glucose in man.

The final proof that this inhibitory polypeptide extracted from human plasma is identical to the inhibitory polypeptide derived from human growth hormone must be a comparison of their chemical structures, but the results cited above would suggest that the two are functionally very similar. It is tempting to attribute to this substance a causal role in the genesis of some forms of diabetes. A polypeptide produced in the pituitary would appear to fit this particular bill very well.

Acid Tests for Peptic Ulcer

Gastric acid acts physiologically as a barrier to bacteria entering the small intestine. It also activates protein digestion in the stomach. In man acid secretion appears to be of little importance, for achlorhydric patients come to no clinical harm from lack of acid. All the same, much medical energy has been expanded on testing gastric acid when investigating gastric function, largely as a result of the "no acid, no ulcer" postulate first expounded in 1910. It is now becoming clear that other features of the gastric mucosa, such as epithelial turnover and loss, may be more important in the pathogenesis of gastric lesions. However, tests of gastric acid are still of important clinical value, and their place has recently been assessed in a critical review of the world literature.

Attempts to measure the amount of gastric secretion were virtually useless until the introduction of the maximal stimulus to acid secretion in 1953. Tubeless tests are unreliable. It is important that the gastric tube is properly placed in the stomach, if possible under fluoroscopic control. Aspiration by hand is the most reliable method of obtaining gastric juice, but continuous low pressure suction, when supervised by an experienced operator, also works satisfactorily.

How useful are gastric acid tests in practice? In an interesting paper at page 196 of the B.M.J. this week Dr. J. H. Baron and Mr. J. Alexander Williams have asked British gastroenterologists (both physicians and surgeons) which tests they used and in what clinical circumstances. The replies indicate that even
among the experts there appears to be some misuse of tests of gastric function. For instance, though there would seem to be no clinical rationale for performing acid tests on patients with dyspepsia who have not had a gastroscopy or barium meal, 14% of the gastroenterologists did so. A much higher proportion (80%) did acid tests on patients with a convincing history of peptic ulcer but who had a normal barium meal. This is reasonable, for in a third to half of patients with a duodenal ulcer secretion of acid is above the normal range, and it is thus possible to diagnose duodenal ulceration in the absence of x-ray changes. This is of particular importance if the patient's symptoms are sufficiently severe to warrant consideration of surgical operation.

Two-thirds of British gastroenterologists measured the secretion of gastric acid in patients with gastric ulcers. This is surprising, for, in Baron and Williams's view, the results of tests of acid secretion do not indicate the clinical course or whether or not surgical treatment is required. The only clinical information of value to come from acid tests for gastric ulcer is if complete achlorhydria is found. This would indicate that the ulcer is a carcinoma. However, as complete achlorhydria is found in only one in five patients with carcinomatous ulcer, the chance of making the correct diagnosis by this means is too small to justify the time, labour, and discomfort to the patient. It is now relatively simple to obtain gastric biopsy specimens under direct vision by using a fibroscope with biopsy attachment. This would seem to be a better investigation for gastric ulcers.

In the rare condition of intractable peptic ulceration due to hypersecretion of gastrin—the Zollinger-Ellison syndrome—tests of gastric acid are helpful. In the typical case the basal acid output is high and is little increased by stimulation. In some cases the results of acid tests are not clear-cut, and it seems likely that estimation of gastric activity in the blood will ultimately replace tests of gastric function for this condition.

Are tests of gastric acid necessary to decide whether or not a patient with a duodenal ulcer needs surgical treatment? In Baron and Williams's opinion the answer is No. However, half to two-thirds of British gastroenterologists were influenced in deciding about surgery, at least sometimes, by acid secretion values. Despite the apparent practice of many gastroenterologists, probably most physicians and surgeons in Britain would agree with the view that the decision whether to operate should be taken on clinical criteria alone.

Tests of gastric function are informative to the surgeon if the patient develops symptoms after gastric surgery. In patients who have had a vagotomy an insulin test is useful in deciding whether or not the vagotomy is complete. There is also evidence that gastric acid tests give helpful information about patients with dyspepsia after partial gastrectomy, and three-quarters of the gastroenterological surgeons, but surprisingly only one-quarter of the gastroenterological physicians, used them in this situation. There seems to be no necessity to do tests of gastric function in asymptomatic patients after partial gastrectomy. After vagotomy an insulin test assesses the skill of the surgeon and gives an indication of the prognosis.

It should be stressed that gastric acid tests are of value only if done carefully by an experienced team. For peptic ulceration they are clinically indicated in three circumstances: x-ray-negative dyspepsia, or when the diagnosis of duodenal ulceration is in doubt; recurrent dyspepsia after partial gastrectomy or vagotomy; and if a diagnosis of Zollinger-Ellison syndrome is being considered. It would appear that tests of gastric acid secretion are being misused even by gastroenterologists.

1 Schwartz, K., Beiträge zur klinischen Chirurgie, 1910, 67, 96.

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**Termination of Life**

So confused have the arguments for and against euthanasia often been that the B.M.A. report issued this week is wise to start with some definitions. Brought into use in the seventeenth century, according to the *Oxford English Dictionary*, the word means simply "a quiet and easy death." Later it was extended to include the means of procuring it or the action of inducing it. In its present sense, it means the deliberate termination of life of a person suffering from a distressing and irremediable disease. Thus from denoting an aspiration that every doctor would wish for his patient, an easy passage from this life, the term has come to mean something that is wholly contrary to the ethics and traditions of medicine—namely, the deliberate causing of a person's death, in fact the killing of his patient if it is a doctor who carries out this act.

The last Bill to come before Parliament achieved a second reading in the House of Lords in 1969, when it was defeated by 61 votes to 40. That it attracted so many supporters as that is a matter for some surprise, for it enabled both physicians and nurses to end their patients' lives under certain conditions. One of these was that the patient must make a written declaration requesting euthanasia "If I should at any time suffer from a serious physical illness or impairment reasonably thought in my case to be incurable and expected to cause me severe distress or render me incapable of rational existence." If the patient became incapable of giving directions, euthanasia was to be carried out at the discretion of the physician in charge of the patient.

Some declaration of this kind must be at the centre of any legislation enabling euthanasia to be carried out. Yet it is hard to see how it can possibly safeguard the patient's true interests. His mental state when committing himself so firmly to the possibility of his own extinction must be difficult for his doctor to ascertain, to say the least. Certainly there are robust rationalists who can weigh up the meaning of alternative possibilities in a calm and collected way, and who can contemplate having a preference for death in certain circumstances. But the experience of many doctors is that such people are remarkably few and that the full resources of medicine will allow a peaceful end to almost all the patients who need them. Moreover, the will to live when directly challenged is apt to be found much stronger than it is often credited with being in rational discussion. Even so, the existence of legal euthanasia might tragically undermine the patient's endurance by making him feel some obligation—even if not reminded of it by a hint from an impatient relative—to sign away his life.

The suicide of a patient when he is under medical care may be more of a reproach for the inadequate treatment he has received than a consequence of any clear assessment of his real prospects. So too a decision to ask for euthanasia might commonly be based on serious misunderstandings. In fact the whole movement itself is open to the same suspicion. Do those who advocate euthanasia and vote for it really understand...