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Realism and Reluctance

The Council at its recent meeting decided unanimously not to seek exclusion of the profession from the Industrial Relations Bill (see *Supplement*, p. 17). This decision taken, the Council now has to obtain amendments which are seen as essential if doctors are to have the full protection of the Bill's provisions.

The matters to be settled include clarification of the definition of "worker" to eliminate any doubt about the position of general practitioners, as it would be an impossible situation if some N.H.S. doctors were in the Bill and others outside it. The definition of an industrial dispute must allow for the doctors' and dentists' negotiating machinery in the N.H.S.,[1] and an amendment will also be required to allow N.H.S. doctors to take advantage of the Bill's provisions on enforcing collective bargaining agreements. Both these issues arise from the Department of Health's responsibility for negotiating N.H.S. terms and conditions of employment without being the doctors' legal employer. The Association also wishes to ensure that all doctors would be represented by one "bargaining" agent, with N.H.S. doctors regarded as a single bargaining unit. Recognition of a sole bargaining agent for doctors would strengthen their negotiating position and protect those doctors who are in a minority in a particular work situation. The Council also wants provision in the Bill to allow a ballot of the doctors concerned where they are a minority group in a threatened industrial dispute.

The Government's proposal[2] to amend the Bill in a way which would allow professional organizations to register without disturbing their existing constitutional status is an acknowledgement that the Bill as drafted did not meet the special position of the professions. The B.M.A.'s involvement in negotiations on terms and conditions of employment for doctors has evolved as part of its broader responsibilities for maintaining the honour and interests of the medical profession. It is this involvement and the doctor's prime responsibility to his patient which adds urgency to the Association's concern about the effects of this legislation.

Until the wording of the Government's amendment is seen it is impossible to know if it will safeguard the profession's position. However, the Department of Employment has made it clear that the special register would not be for a profession as such—an elusive entity to define—but "for organizations that are not eligible for registration as trade unions, but that could justifiably be granted advantages given to registered unions by the Bill."[2] The amendment should be helpful to the B.M.A., but even more so to the Royal College of Nursing and the Royal College of Midwives, both of which are chartered bodies with charitable status, and whose future looked in peril as a consequence of the legislation.

Though the B.M.A. should now be able to register under the Act without any radical change of its Articles of Association some doubts were raised in Council on the wisdom of doing so in a special section. The argument voiced was that the B.M.A. should face facts, join up as a union, and act like one. By doing so it would merely be giving formal expression to what it already does. In addition it would attract support from doctors who see nothing wrong with union status. This might be true, but the great majority of doctors still find trade unionism an uncomfortable bed fellow for a professional man. If the B.M.A. became a full-blown trade union, undertaking the necessary sweeping constitutional changes and relegating its scientific and educational activities to a back seat, it would undoubtedly alienate many members. Moreover, the Opposition have threatened to repeal the Act on returning to power. Should the Act's advantages be removed in the future then the Association could find itself left with the worst of all worlds. Registration in a special niche in the Act should leave future options open. If the Act becomes a permanent part of the political landscape recognition as a special group would probably make it easier to achieve amendments on the profession's behalf in the future. If the Act was repealed then the B.M.A. would be able to continue much as before.

Considering the complexity of the legislation, the uncertainty about its interpretations, and its implications for doctors the unanimity among the different branches of the profession in the Council debate was notable. Mr. Walpole Lewin suggested that the decision of the Central Committee for Hospital Medical Services not to seek exclusion was taken with realism though reluctance. The same may well be said of the Council. The Government's intention to include doctors was realistically accepted, but there was an under-

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.

[1] *British Medical Journal*, 1971, 1, 126.
[2] *British Medical Journal Supplement*, 1971, 1, 14.

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[1] 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 glycolysis and fat synthesis and accelerates breakdown of fat. A.C.G. causes hypoglycaemia and reverses the inhibitions produced by I.N.G.

When the acceleratory polypeptide was given to five diabetic patients[2] 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[3] 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 ketoacidosis.

Since the inhibitory polypeptide inhibits glycolysis, 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.

[1] Bornstein, J., Krahl, M. E., Marshall, L. B., Gould, M. K., and Armstrong, J. McD., *Biochimica et Biophysica Acta*, 1968, 156, 31.
[2] Armstrong, J. McD., Bornstein, J., Ng, F. M., and Taft, H. P., *British Medical Journal*, 1969, 2, 157.
[3] Bornstein, J., Armstrong, J. McD., Ng, F. M., and Taft, H. P., *British Medical Journal*, 1969, 3, 451.

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.[1] It is now becoming clear that other features of the gastric mucosa, such as epithelial turnover and loss,[2] 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.[3]

Attempts to measure the amount of gastric secretion were virtually useless until the introduction of the maximal stimulus to acid secretion in 1953.[4] 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