The polyneuropathy associated with insulinoma tends to show distinctive features; it usually makes its appearance after a protracted period of coma, it is predominantly motor in character, and wasting of the small muscles of the hand is a common feature.¹⁻³ The polyneuropathy which may follow insulin shock therapy is, on the other hand, predominantly sensory in character and objective evidence of sensory loss is often absent,⁴ a clinical picture much more reminiscent of diabetic neuropathy.

In the two patients described the diabetes was of late onset, insulin requirement diminished quite rapidly once treatment had been commenced, and hypoglycaemic episodes were frequent but mild. The neuropathy was entirely sensory, was confined to the lower limbs, was purely subjective in character, and was relieved by withdrawal of insulin. The clinical picture was consistent with a diagnosis of diabetic neuropathy,⁷ but the abolition of symptoms on withdrawal of insulin would suggest that excessive administration of insulin may have been a primary factor in causation.

In Case 1 there was no recurrence of neuropathy on tolbutamide therapy. Nevertheless it is conceivable that instances of hypoglycaemic neuropathy might occur in patients taking tolbutamide, for Marks *et al.*^{8,9} have shown that tolbutamide may precipitate neurological symptoms in the presence of a low blood-sugar level.

Since these two patients were observed in a small general practice over a period of seven years hypoglycaemic neuropathy in diabetic patients may be of fairly common occurrence. It might be rewarding to review all treated diabetic patients who suffer from peripheral neuropathy.—I am, etc.,

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Neurogenic Intermittent Claudication

SIR,—Neurogenic intermittent claudication is quite often associated with lumbar spondylosis.¹⁻⁴ In this condition the volume of the spinal canal is diminished, and it is tempting to infer that relative radicular ischaemia on exercise is related to nerve-root compression. Blau and Logue⁵ record that claudication was relieved after a decompressive operation in all their treated patients, whether or not the accompanying disk protrusion was completely removed. Ehni has pointed out that the condition of lumbar spondylosis is little known and has been variously reported under several headings, among which he includes Blau and Logue's paper. It is typified by encroachment by bulging disks, hypertrophied facets and laminae, and redundant ligamenta subflava on a developmentally small spinal canal.

In Dr. J. G. Evans's paper (17 October, p. 985) it was noted at operation on the second patient that the lumbar roots bulged through the incised dura, implying that they were compressed abnormally. I wonder if this is another example of lumbar spondylosis.—I am, etc.,

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Resuscitation of the Newborn

SIR,—Some readers of the *B.M.J.* have asked for details concerning the blind method of endotracheal intubation described in my earlier letter (15 August, p. 446).

The principle of this method is that a tube held in front of the finger, which is partially occupying the pharynx, should tend to enter the larynx, if it is pushed in that direction. There is not much room inside a baby's laryngopharynx, and once the pharynx is partially occupied by the finger the same finger can manipulate the tube into the glottis. The operator needs some practice to do this quickly and efficiently, but this is easy to achieve. The wearing of a well-fitting glove does not interfere. As for the tube, I have preferred to use a gavage catheter. It is firm, yet has a blunt point, is supple and is unlikely to damage the vocal cords. An endotracheal tube is stiff and curved, but has a sharp point and therefore is best left alone. Another suitable substitute is the end of a polyethylene tube used in blood-transfusions. The end is thickened and the tube itself has a tendency to form a natural curve, which makes it easy to pass.

Once intubated, secretions are sucked out. When blowing in, it is not essential to have a stethoscope to confirm that the tube is in. If it is in the trachea the whole abdomen and chest bulge out and one may be able to feel a thrill due to air entry, with the hand placed on either side of the chest. If the oesophagus has been entered the left side of the abdomen moves, there is a rattling noise, and there is a lack of resistance. The same is observed if the tube has slipped out during the procedure.

A refinement would be to give the air or oxygen under controlled pressure. One may use a bottle with a central straight tube dipping in water, an oxygen inlet tube also dipping, and an outlet tube. The central tube serves as a pressure gauge.

Sometimes the heart beat is absent when intubation is started. Cardiac massage with the thumb was effective in some babies, but in one there was no spontaneous respiration even after 1½ hours and removal of the tube caused death in a few minutes. In most of the cases resuscitated in this hospital intragastric oxygen was already being given before intubation without avail. Significant amounts of air do not leak out of the glottis during the procedure. Most of these cases were babies who had not taken a first breath, but even in them the expansion was good.

There were no failures, though the success with the first attempt largely depends on the previous experience of the person doing the intubation. Smaller babies are more difficult. The method is, I think, worthy of trial in smaller institutions, where other facilities are not readily available.—I am, etc.,

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Code for Drugs

SIR,—Drs. F. C. Clouting and J. Wightman (24 October, p. 1073) will no doubt be interested to learn that slow but definite progress is being made with the problems of tablet identification. This, although considered to be impracticable, has been in fact Association policy since the Annual Representative Meeting of 1959.

I should perhaps refer them to my original paper on this subject¹ and also paragraphs 143 of the 1961² and 160 of the 1962 Annual Report of Council³ in which the results are shown of the discussions which took place between the B.M.A., the Association of the British Pharmaceutical Industry, and the Pharmaceutical Society. I have recently asked for the whole question of tablet identification to be reopened in the light of the following developments:

(a) Two American publications,^{4,5} which show conclusively the difficulty of identifying accurately tablets (and in particular white unmarked ones) as things are at present and the considerable improvement of the accuracy of identification by marking tablets.

(b) That as Drs. Clouting and Wightman have indicated it is possible to mark sugar-coated tablets—a fact which I pointed out in 1960, but at that time it was considered to be too expensive. However, since at least three firms are now marking sugar-coated tablets it cannot be too expensive at the present time.

(c) With the advent of spray-coatings of tablets (with methyl cellulose) it is possible to mark the core, as with an uncoated tablet, and this marking will show through the spray coating.

(d) It is felt in many quarters that the British Pharmaceutical Industry might well be more willing to undertake this on behalf of the profession.

I understand that the Joint Subcommittee of the Association, A.B.P.I., and Pharmaceutical Society on tablet identification is to be reconvened very shortly, when they will have before them a communication from me on the above lines.—I am, etc.,

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Addiction to Dextromoramide

SIR,—I have recently had a case of addiction to dextromoramide which resembles that reported by Dr. B. A. M. Thompson (31 October, p. 1136).