Correspondence

Pelvic floor neuropathy

Sir,—I read with great interest the recent article by Rogers et al (Gut 1988; 29: 756-61). Their study focused on subclinical sensorimotor neuropathy in continent diabetics and included speculation concerning the pathogenesis of faecal incontinence in diabetics. The article ended with the statement that the authors’ hypotheses ... will only be confirmed by a further study of ... incontinent diabetic patients ...

Indeed, there is already a body of data which characterises diabetics with faecal incontinence and I am surprised that these earlier studies were not appropriately discussed. Schiller et al (N Engl J Med 1982; 307: 1666-71), cited by the authors only for the 20% prevalence of faecal incontinence found in their survey of diabetic outpatients, found that incontinence was frequently associated with decreased basal anal sphincter pressures, inability to retain an infused volume of saline into the rectum and diminished external sphincter pressures, findings which were not present in continent diabetics. These findings are similar to the studies of Rogers et al. Subsequently, A K Tunuguntla and I reported that thresholds of conscious rectal sensation were frequently impaired in diabetics with faecal incontinence and external sphincter function was also impaired, in contrast to preservation of normal sensorimotor anorectal function in continent diabetics (N Engl J Med 1984; 310: 1282-7). Subsequently, we compared anorectal function in diabetic and idiopathic faecal incontinence (Gastroenterology 1984; 86: 1285). Both groups exhibited similar abnormalities of anal sphincter function characterised by impaired continence at rest and with sphincter contraction, and raised thresholds of phasic external sphincter contraction; in contrast, only diabetics had impaired rectal sensation which was not related to rectal tone or compliance.

These previous studies do not detract from the fine studies of Rogers et al which confirm and extend previous studies done in continent diabetics. I believe that your readership would have benefited from the citation and discussion of these earlier reports in order to place the current studies and this important clinical problem in a broader and more accurate perspective.

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Reply

Sir,—We were interested in the letter from Dr Wald commenting on our paper.

The discussion section of our paper was not intended for a speculative review on the likely pathogenesis of incontinence in patients with diabetes mellitus, but rather to comment on our current data. Our study was of continent diabetic subjects with proven peripheral neuropathy. The aim was to determine if subclinical abnormalities of sensorimotor function exist in the pelvic floor, in light of the high prevalence of faecal incontinence that occurs in diabetic subjects.

Schiller et al in their study of incontinent diabetics attempted to improve on previous incidental reports, one of which found abnormal rectal sensation and normal sphincter pressures, and another which found low or low-normal sphincter pressures. They found low basal or resting pressure in the anal canals of those diabetic patients with incontinence but normal voluntary contraction pressures. All but one of the incontinent diabetics had diarrhoea and 75% reported that the onset of diarrhoea coincided with incontinence. In contrast the continent diabetics studied had normal sphincter pressures and no history of diarrhoea yet this group had a similar incidence of autonomic neuropathy and steatorrhoea.

The conclusion made of the incontinence being related to abnormal internal anal sphincter weakness is not entirely convincing, as diarrhoea may equally have been the cause.

Wald and Tunuguntla cited abnormal rectal sensation and impaired function of the external anal sphincter function (or both) as the cause of incontinence in diabetic patients. Although they found a significantly increased threshold to conscious (subjective) rectal sensation to balloon distension compared with normal subjects, continent diabetics and patients with IFI, in the incontinent diabetics they found no difference between the groups in the (objective) threshold of internal sphincter relaxation to rectal distension. Their finding of an abnormality in the external sphincter relates to absence or delay in the phasic activity as a response to balloon distension. This is a qualitative finding and they apparently did not make a quantitative assessment of anal canal function by manometric measurement of sphincter pressures. We agree with their statement ‘Thus, it is not possible to compare our patients with those in other reports’.

In a later study by Tunuguntla and Wald on incontinent diabetics, normal subjects, continent diabetics, and patients with IFI, the most surprising finding was that there were no differences in voluntary contraction pressures of the external sphincter between the groups. Another interesting finding of
Pelvic floor neuropathy.

A Wald

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