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.[1]

Schiller *et al*[1] in their study of incontinent diabetics attempted to improve on previous incidental reports, one of which found abnormal rectal sensation and normal sphincter pressures,[2] and another which found low or low-normal sphincter pressures.[3] 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[4] 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[5] 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

this study is that the tests used did not pick up any latent abnormality of anorectal function in the continent diabetics.

In these studies the only objective finding was that of reduced internal sphincter pressure in the anal canal measured by manometer. Conscious appreciation of rectal distension is a highly unreliable test.[6] In our study, we found that thresholds of rectal distension could not detect differences between normal subjects, diabetics, or patients with faecal incontinence. None of the above papers have helped in defining the pathogenesis of faecal incontinence in diabetic subjects. They suggest that the cause is either internal anal sphincter dysfunction, external anal sphincter dysfunction or decreased awareness to rectal distension, or perhaps combinations of the above. In our study we have used precise, repeatable[7] and objective measurement of sensory and motor function of anorectal physiology, which has delineated previously unknown asymptomatic abnormalities in the pelvic floor of patients with diabetic neuropathy. This combined with the fact that the diabetic population are at risk of faecal incontinence[1] and that idiopathic faecal incontinence is commonly associated with pelvic floor neuropathy suggests that progression of pelvic floor neuropathy in diabetic subjects may lead to faecal incontinence. We still maintain, however, that this view will only be confirmed by a further study of a group of incontinent diabetics using the same techniques.

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Are individuals born as twins at a higher risk of developing Crohn's disease?

SIR,—In a recent survey of the Swedish twin registry including 25 000 pairs of twins, Tysk *et al*[1] found that the prevalence of ulcerative colitis among twins was almost similar to its prevalence in the general population in Stockholm (74×10^5 *v* 78×10^5, respectively). The prevalence of Crohn's disease among twins, however, was almost twice the prevalence of this disease in the general population in the same area (106×10^5 *v* 54×10^5, respectively). Unfortunately, the above mentioned authors did not relate to the difference in the prevalence of both diseases in twins, nor did they suggest any explanation for the increased incidence of Crohn's disease in twins.

It is anticipated that the prevalence of ulcerative colitis and Crohn's disease would be higher in a population of twins than in the general population because of the genetic aetiologic factor in both diseases, particularly in Crohn's disease. When one twin is affected with Crohn's disease, the other twin is at increased risk of being affected, especially if both are monozygotic. When the prevalence of Crohn's disease in the twin population is calculated, however, even if both twins are affected, the prevalence is 88×10^5 – much higher than in the general population.

Another method of determining whether twins are at increased risk of Crohn's disease is to calculate how many patients affected with the disease have twins. We are currently completing a study of the familial incidence of Crohn's disease among Jewish patients in Israel. In a group of 154 patients, four were found to have an unidentical twin who was not affected. In this group, one in 39 Crohn's patients has a twin as opposed to the incidence of twins in the general population, which is approximately one in 89.[2]

It, seems, therefore, that there is an increased risk for an individual born as a twin to be affected with Crohn's disease. Additional data are needed to confirm this observation. A better understanding of this tendency may help in the understanding of the aetiology and transmission of the disease.

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