benefit of workers in other parts of the world. In the UK in 1960-5, according to Ashley,[10] evidence suggested that "some 15% of males and 18% of females are subjected to appendicectomy." What are the proportions nowadays? What is the present situation in, say, the Hebrides compared with that in London? In 1942 operation frequency in pre-OCTU student candidates was *eight* times higher than that in non-student young men.[11] Does a like disparity still prevail? Furthermore, in immigrant populations in London such as Caribbeans and Asians, what are the respective frequencies of operations among them? In the USA appendicitis incidence "has decreased 40% in 20 years, perhaps more"[12]; but from what rate to what rate?

We live in a time when the armamentarium for disease detection is far advanced and magnificent. It is therefore ironic that elementary information is almost completely lacking on a disease's precise frequency, the index of which, in a community, is more readily ascertainable than the making of an anthropometric measurement.

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Guar crispbread in the diabetic diet

SIR,—We are naturally interested in the preliminary report by Drs M Cohen and M I R Martin (3 March, p 616) of their study on the addition for three months of either a gel-forming fibre or bran or an unspecified placebo to the diet of obese "maturity-type-onset" diabetic outpatients, and thoroughly agree with their emphasis on the important need for long-term studies of dietary fibre supplements in diabetics. Hence we have already submitted for publication results on the first small group of diabetics taking a guar-supplemented diet continuously for six months, happily without obvious mineral deficiency.

We would like to make two main comments concerning their negative finding with guar gum. Firstly, as recently emphasised in correspondence elsewhere,[1] gel-forming fibre is likely to be markedly effective in reducing postprandial blood glucose concentrations only when it is intimately mixed with the carbohydrate portion of the food—for example, guar gum in the bread mix before cooking or extra pectin mixed into marmalade.[2 3] We are uncertain how the guar gum was added to the diet in their study. Gel-forming fibre must be *in* the food, not on it, to be fully effective.

Secondly, short-term studies[2-4] of gel-forming fibre have all dealt with the reduction in postprandial (or postloading) glucose levels. The principal postulated action of guar is to effect a reduced rate of absorption of nutrient from the small bowel without causing malabsorption. Apart from the effect on satiety,[5] there is no a priori reason to expect from this a weight change or alteration in overnight fasting glucose concentrations. If they occur, they are likely to be secondary consequences of the main action. This is also why we have looked particularly at urinary glucose values, which are so susceptible to alterations in postprandial blood glucose levels.

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Heart rate in diabetes mellitus

SIR,—There is great attraction in simplifying measurements of autonomic function. Dr G Sundkvist and others (7 April, p 924) propose a new test of heart rate variation—the "E:I ratio," which they say "seems to be as accurate as traditional tests for autonomic function and easier to perform." Although they may be right in this claim, the interpretation of the results that they have actually presented must be viewed with some caution.

They have shown clearly that the R-R interval is shortened in their diabetics with sensory neuropathy, and that R-R interval variation is also reduced. They have also shown that the E:I ratio is lower in the same subjects. But what evidence have they that the E:I ratio in these subjects is not just a function of a faster resting heart rate? None of them had any clinical symptoms of autonomic neuropathy, and although diabetics as a group have a faster heart rate than normal subjects its relation to autonomic neuropathy is obscure.

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**We sent a copy of this letter to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—Dr Ewing has raised the question whether the reason for the observed abnormalities in E:I ratio (1·10 or below) among our diabetics with sensory neuropathy is due to the increase of the resting heart rate. Earlier studies,[1] however, have shown that the heart normally is inhibited by vagal tone, and defective nervous function gives rise to tachycardia that is easily demonstrated by atropin experiments. The influence of deep breathing on heart rate (beat-to-beat variation) has also been proved to be dependent on normal vagal function.[2] Thus deficient beat-to-beat variation indicates vagal dysfunction and the same mechanism explains the increased heart rate in such patients. It has also been stated[3] that slight impairment in beat-to-beat variation could be the earliest manifestation of neuropathy.

Our study clearly demonstrated that estimation of the E:I ratio was equivalent to the method used in these previous studies.[1-3] Therefore our patients probably have vagal neuropathy, which could explain their increased heart rate at rest. In diabetics with abnormal E:I values correlation analysis did not show a significant r value (0·19) between the R-R intervals at rest and E:I ratios. Furthermore, no significant correlation was found in a recent study[4] between changes in heart rate in response to a single deep breath and mean resting supine heart rate. These results seem to contradict the idea that the increased heart rate gives the abnormal ratio.

In conclusion, our belief is that the abnormal E:I ratios are manifestations of early autonomic neuropathy in the investigated patients. But the final answer to the controversy will, of course, come during the next years when the patients are followed up.

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Heart rate variation in tetraplegic patients

SIR,—Having read the article written by Dr G Sundkvist and others (7 April, p 924) and being very interested in the analyses of heart rate variations as an easy method for the evaluation of autonomic nervous disturbances, we would like to add our experience.

Recently Neubauer and Gundersen[1] have proved that in patients with multiple sclerosis, in whom the autonomic nervous system can be involved, the mean square successive difference (MSSD) and the usual standard deviation (SD) of the R-R successive intervals in the ECG show a reduction comparable to that observed in long-term diabetics with autonomic abnormalities. We are now studying the autonomic disturbances in a group of 15 tetraplegic patients admitted to our intensive care unit because of severe respiratory failure. Ages range from 19 to 45 years, seven patients being women and eight men. The underlying diseases are a traumatic injury of the spinal cord in 12 patients, a cervical tumour in one, Heine-Medine disease in one, and cervical myelopathy in the last. The level of tetraplegia is between C4 and C6. In all these patients, as in 14 members of the medical staff (seven women and seven men) with ages ranging from 19 to 35 years, an ECG was obtained during a period of three minutes with a paper speed of 50 mm per second, measuring the lengths in millimetres of at least 150 consecutive R-R intervals. The statistical analyses of these two groups show an MSSD and SD of 1·36 and 1·77 respectively for the control group and 0·40 and 0·60 for the group of patients. This means a statistically significant difference at the level of $P<0·00001$ when a comparison of MSSD and SD for both groups is made. We consider the measure-