

and we would be glad to hear from any centres that still have questions on these or any other matters. Equally, we would welcome reports from any centres that have found ways of dealing with any particular reporting problems that may have arisen as a result of the act.

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1 Brinsden P. A tax on infertility? *BMJ* 1991;303:309. (3 August.)

"Brittle" diabetes

SIR,—Dr Stephanie Amiel's editorial on brittle diabetes was a timely and very useful review of a difficult and poorly defined area.¹ We would, however, like to make some comments about a common misconception and a long standing myth about this condition, which are mentioned in her discussion.

The more important misconception, which is probably widely held, is that defects such as impaired subcutaneous insulin absorption or exaggerated counterregulatory hormone responses have actually been shown in a significant number of patients. We do not dispute that these mechanisms provide a possible and plausible basis for the unpredictable metabolic control of brittle diabetes. Apart from a very few exceptional cases, however,^{2,6} there is little or no direct evidence of genuine pathophysiological defects, whereas deliberate and often well concealed interference by the patients with their treatment has been detected in many cases. The reference which Dr Amiel cites to support a specific defect in insulin absorption merely shows that in some patients brittle diabetes could be better controlled for short periods by continuous intravenous insulin infusion than by subcutaneous insulin. Further experience showed intravenous insulin to be unhelpful, at least in part because of proved sabotage by the patients.⁸ The only formal studies of insulin absorption and action in patients with brittle diabetes and those with apparently high insulin requirements did not show any defect in either absorption or insulin action.^{9,10}

Dr Amiel also discussed exaggerated counterregulatory responses to hypoglycaemia and psychological stress as possible causes of brittleness, or at least of glycaemic swings. We are aware of no evidence that either of these factors produce the very large and rapid glycaemic rises seen in brittle diabetes. In particular, the psychological studies which claim to show such an effect have been poorly controlled or not controlled at all, or are not reproducible.¹¹ Psychological stress is undoubtedly related to brittleness, but the only established link is in patients who manipulate their treatment to escape from psychosocial difficulties at home or work, not because of rebellious hormones. The most likely cause of rapidly developing hyperglycaemia and ketosis in these patients remains insulin deficiency due to the patient interrupting insulin administration.

A role for metabolic or hormonal defects in brittleness may eventually emerge, particularly in the case of recurrent hypoglycaemia. For the time being, however, we believe that it is best for those looking after patients with recurrent ketoacidosis to concentrate on helping them to live more comfortably with their diabetes, rather than using pathophysiological mechanisms as a scapegoat.

The less important "myth" is that the term brittle was first used by Woodyatt in the 1934 edition of *Cecil's Textbook of Medicine*. Our own attempts to find and verify this reference have been unsuccessful, but Dr Birger Thorsteinsson of the Steno Hospital, Copenhagen, has managed to locate the chapter and we are grateful to him for sending it to us. It does not in fact contain any reference to brittle diabetes. This self replicating

bibliographic virus has attacked our own publications on brittle diabetes, and those of several authorities in the field,^{12,13} as well as Dr Amiel's review. The debate on the causes of brittleness will no doubt continue, but perhaps we can at least find out who really did coin this much abused term.

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Eating disorders in insulin dependent diabetes mellitus

SIR,—Dr Christopher G Fairburn and colleagues, unexpectedly, found no evidence of an increased incidence of eating disorder in their diabetic population and made plausible suggestions as to the reason for the discrepancy between the results of their study and earlier studies.¹ I note, however, that four female patients (nearly 7%) declined to take part. If these women declined because of a self diagnosed eating disorder would the conclusions of the study have been different? Were the authors able to ascertain reasons for these patients' refusal to participate?

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- 1 Fairburn CG, Peveler RC, Davies B, Mann JI, Mayou RA. Eating disorders in young adults with insulin dependent diabetes mellitus: a controlled study. *BMJ* 1991;303:17-20. (6 July.)

SIR,—In their study on eating disorders in young diabetic patients Dr C G Fairburn and colleagues concluded that there is no evidence that clinical eating disorders are more prevalent in young women with diabetes than in non-diabetic women.¹

On the basis of our experience with anorectic

patients we would like to comment on some of the points raised by the authors. Firstly, 10% of the diabetic women in their study chose not to participate or could not be traced. These non-participants may include women with eating disorders as people with anorexia do not generally cooperate and tend to be secretive about their behaviour. Secondly, the authors did not mention whether the eating attitude test was adapted for use in diabetic patients because some of the items in the test are unsuitable for use in diabetic populations.² Thirdly, the fact that no subject with anorexia was identified in the diabetic sample may be attributable to the relatively high mean age (21) of the diabetic women. In adolescents with diabetes we could expect a higher prevalence of eating disorders.

The high proportion of the diabetic women who omitted or underused insulin to influence weight emphasises the "dangerous weapon" given to diabetic patients—that is, insulin.

The question whether anorexia nervosa and bulimia nervosa are more prevalent in diabetic young women is secondary to the possible hazardous effects of an eating disorder on the control of the diabetes. We believe that every physician should be familiar with the possible coexistence of diabetes mellitus and eating disorders. Moreover, eating disorders should be considered whenever satisfactory glycaemic control proves unexpectedly difficult to achieve.^{3,4}

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- 1 Fairburn CG, Peveler RC, Davies B, Mann JI, Mayou RA. Eating disorders in young adults with insulin dependent diabetes mellitus: a controlled study. *BMJ* 1991;303:17-20. (6 July.)
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SIR,—Dr Christopher G Fairburn and colleagues found that eating disorders are no more common in diabetic than non-diabetic women.¹ This conclusion conflicts with that of several other studies, including our own, which looked at 147 young women (97% of a cohort attending a diabetic clinic in Edinburgh).² The authors suggest three reasons for the discrepancies.

Firstly, they carried out full interviews. We accept the inherent merit of doing this. We interviewed only those with high scores to ensure honest reporting. On this basis our data may have given an underestimate of the prevalence of eating disorders.

Secondly, they comment that in studies from tertiary referral centres patients would be pre-selected. In Edinburgh, as in their study from Oxford, all young diabetic women attend the local diabetic clinic.

Thirdly, they suggest that control groups in other studies were inadequate. Ours comprised non-diabetic siblings or friends of the same sex of similar age and social class. The slight differences we found in weight did not account for the differences in test scores. Dr Fairburn and colleagues do not state the weight of their subjects. We found no difference in scores obtained with the eating attitude test or eating disorder inventory (corrected for questions related to diabetes) between our male diabetic patients and controls. By contrast, 20% of our female diabetic patients compared with 10% of the controls had a high score in the eating attitude test. Significant differences were also found with several subscores of the eating disorder inventory. Peveler and Fairburn criticised our control group on the grounds that none of the subjects had a high score