High-carbohydrate diets and insulin-dependent diabetics

SIR,—Dr N M O'Mullane suggests (29 September, p 796) that the improved diabetic control we observed in insulin-requiring diabetics on high-carbohydrate diets (1 September, p 523) could be explained by a difference in dietary fibre between the experimental diet and the standard low-carbohydrate comparison diets.

Unfortunately Dr O'Mullane did not refer to the earlier publication (30 June, p 1755) in which we presented results of a similar dietary comparison in maturity-onset patients. We did, however, observe that the high-carbohydrate diets did indeed contain appreciably more dietary fibre than the low-carbohydrate diet. We agree that our own published work thus far provides no conclusive data concerning which aspect of the high-carbohydrate diets is responsible for the improved diabetic control. We did, however, quote various studies which suggest that the reduced glucose baseline on a high-carbohydrate diet is a consequence of digestive carbohydrate. Ongoing dietary studies in Oxford aim to determine the optimal proportion and nature of carbohydrate in the diabetic dietary prescription.

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Withdrawal of cyanocobalamin

SIR,—Professor D M Matthews and Dr J C Linnell (1 September, p 533) make valuable distinctions between the cobalamins. Not only is hydroxocobalamin at least as effective therapeutically in all circumstances as cyanocobalamin, but it is also safer in occasional patients with tobacco amylloba, Leber's optic atrophy, and tropical polyneuropathy. In addition, the basic NHS cost quoted by Glaxo is the same for both.

Surely there is a very strong case for asking for the complete withdrawal of cyanocobalamin from the market.

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The concept of disease

SIR,—In addition to the endless debate whether so-and-so is a disease, we read of diseases in their own right, diseases sui generis, disease entities, entities, specific entities, true entities, and real entities. And we may ask whether some entity is a disease, or some disease is an entity. That majority of doctors who—in the words of your leading

Endotoxins and gastroduodenal ulceration after burns

SIR,—Your leading article "Gastric and duodenal ulceration after burns" (1 September, p 512) exactly indicates that we still know little about how this occurs. In an attempt to identify some of the mediators involved in the pathogenetic mechanism of these multiple erosions, we would like to suggest a possible role of endotoxins.

In fact, experimental investigations have demonstrated that endotoxins of intestinal origin enter the circulation because the intestine is made more permeable by the release into the blood of a vasoactive substance from the burned skin. In addition, fatal endotoxaemia detected by the Limulus endotoxin assay developed in normal rabbits that received the venous blood from the burned area before it contained endotoxin; by contrast, absence of endotoxaemia and the survival of rabbits which did not harbour Gram-negative bacteria in the intestine was noted. Recent clinical observations have added credence to the importance of endotoxaemia in burned patients. Since the myriad of biological activities of endotoxins includes also a direct effect on the gastric mucosal circulation, permeability, and morphology, we—like others—believe that significant mucosal ischaemia can occur and may represent a mechanism in the development of gastric erosions in endotoxaemia after large burns.

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