claim, which will leave the diabetic with a heavy and unnecessary liability.—I am, etc.,

GEORGE R. W. N. LUNTZ,

Chairman,
Medical Advisory Committee,
British Diabetic Association.

London, W.C.1.

Glucose and Insulin After Diet and Tolazamide Treatment

SIR,—Dr. J. R. Turtle's study of glucose and insulin secretory response patterns following diet and tolazamide therapy in diabetes (12 September, p. 606) confirms our own findings^{1,2} in respect of the effects of long-term acetohexamide in non-obese, maturity-onset diabetes—namely, that after three months of treatment with acetohexamide (0.5g. b.d.) the effectiveness of endogenous insulin is enhanced.

Our patients differed from Dr. Turtle's in that their diabetes was more severe in terms of blood glucose levels. During treatment only 13 of our patients achieved a reduction in the area under the blood glucose curve of 16% or more of that at the initial test, before treatment, and the mean reduction after three months' treatment was by 40%. Also, in contrast to his patients, the mean area under the insulin curve at the time of our initial tests was significantly less than in our non-obese, non-diabetic controls. After two months' treatment the mean area under our glucose curves had fallen by 35%, but the mean area under the insulin curves had risen by 46%. After three months' treatment, however, although the mean area under the glucose curves was reduced further, by 40% of the initial value, the mean area under the insulin curves had declined, though it was still 31% above the initial value.

The differences between the results of our two studies appear to be due largely to differences in the severity of the diabetes. It is known that the insulin response to glucose in diabetics declines as blood glucose levels rise,³ which may explain the differences in the initial insulin responses with respect to controls in our two series. During the first month or two of treatment in our patients insulin output increased as glucose tolerance improved, but by three months sustained or progressive improvement in glucose tolerance was accompanied by a decline in insulin output towards, or even below initial levels. Thus at this time, in both Dr. Turtle's and our patients, there appeared to be less antagonism to insulin. Dr. Turtle has also shown that with diet alone though relative insulin deficiency persisted insulin effectiveness increased.

It appears, therefore, that reduction in blood glucose levels, whether by diet, sulphonylureas, or indeed by exogenous insulin, is a common factor, possibly allowing initially for improved β -cell function. Prolonged hyperglycaemia may cause islet cell degeneration, and persistent diabetes in cats⁴ and the insulin secretory mechanism in man becomes exhausted in the face of prolonged hyperglycaemia.⁵ It is reasonable to suppose that recovery of islet cell function might take place when long-standing hyperglycaemia is reduced by any means. The subsequent reduction of insulin antagonism may possibly be a direct consequence

of the initial rise in circulating insulin, as it is known that diminished insulin sensitivity in alloxan-diabetic animals may be counteracted by insulin.⁶—I am, etc.,

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Diabetic Ketoacidosis

SIR,—I welcome your leading article (10 October, p. 63) in which, in the last sentence, you stress that admission to hospital of children with diabetic ketoacidosis should be promptly sought. I have for some time been concerned at the delay over the admission to hospital of children with this condition. Having correctly diagnosed diabetes mellitus, the doctor has referred the child to the outpatient clinic, which results in a delay of a week or two.

Recently we lost a four-year-old child due to the sudden onset of cerebral oedema four hours after admission to hospital and the start of treatment. The danger of cerebral oedema, particularly in young patients, was pointed out again in a recent paper.¹ Though Clements² et al. have provided good evidence that this is due to a disturbance of the glucose-polyol metabolic pathway in the brain cells, the best method of treatment remains in doubt. Consequently, prevention of the advancement of the hyperglycaemia is essential. It may be that doctors are not differentiating clearly between the insulin-dependent diabetes of children and maturity onset diabetes, in which the rapid development of ketoacidosis is less likely.

It is important to appreciate that children and young adults are particularly at hazard from acute onset ketoacidosis and from cerebral oedema.—I am, etc.,

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SIR,—We think that Dr. P. Z. Zimmet and his colleagues (12 September, p. 610) do us less than justice when they cite only our paper¹ as an example of "over-correction" of diabetic acidosis with bicarbonate.

In fact, we commented there on the significance of the metabolic alkalosis encoun-

tered during treatment and then stated "from the experience gained in treating these patients, we now feel that if the blood pH is dangerously low (below 7.2) then a dose of 200 mEq bicarbonate should be given immediately intravenously and followed by insulin plus standard intravenous fluid replacement. If the pH fails to rise to 7.2, then further 100 mEq aliquots of bicarbonate should be given until that level is obtained. With this regimen no significant metabolic alkalosis should develop."

This advice closely parallels that offered by Dr. Zimmet and colleagues as their "more rational approach."

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REFERENCE

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Folate and Vitamin B₁₂ in Epilepsy

SIR,—May we comment on some of the issues raised by Dr. C. Neubauer's paper (27 June, p. 759), the accompanying leading article (p. 744), and subsequent correspondence.

Dr. Neubauer's uncontrolled observations are consistent with the hypothesis that drug-induced disturbance of folic acid and vitamin B₁₂ metabolism is one of the factors that may contribute to the development of psychiatric complications in some epileptic patients.^{1,2} This in no way undermines the importance of other well-recognized causes of mental illness in epileptic patients.

There is now evidence of a significant association between a variety of psychiatric diagnoses in epileptic patients and disturbed folate metabolism as measured in serum, red cells, and C.S.F.^{3,4} Though, as commented by Dr. R. P. Snaith (18 July, p. 165), no specific diagnosis is incriminated there is a tendency for the lowest levels to be found in patients with intellectual deterioration. It is relevant to point out that recently three separate inborn errors of folate metabolism have been described, all associated with mental retardation.⁵ Dr. N. S. Gordon (25 July, p. 226) refers to Jensen and Olesen's⁶ report of "normal" whole blood folate levels despite the fact that 91% of their intellectually deteriorated epileptic patients had subnormal serum folate levels. In fact, no control values were included, and examination of their data reveals very low whole blood folate levels.⁴

Though there are conflicting reports of the effects of treatment with folic acid alone we would like to emphasize the importance of treatment with both vitamins.² Why the vitamin B₁₂ is necessary is not clear, especially as serum vitamin B₁₂ levels are usually normal, but it may be related to the fall in serum vitamin B₁₂ levels that occurs in the majority of patients during treatment with folic acid.^{1,2}

With regard to the effect of the vitamins on seizure control, although we would agree with Dr. Gordon that the evidence that folic acid aggravates seizures is not

conclusive (and certainly not in all cases),² we would still urge caution in the use of these vitamins, especially until more data is forthcoming. We also agree with your correspondents who question the "antagonistic" or "protective" effect of vitamin B₁₂ on seizures mentioned in your annotation. There does not appear to be any published evidence for this at present, in fact the reverse.²—We are, etc.,

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Race and Commonwealth

SIR,—The *B.M.J.* of 19 September has just reached me and after reading Dr. G. W. Gale's letter (p. 705) in connexion with the "expulsion" of the Medical Association of South Africa from the Commonwealth Medical Association I turned back to your issue of 29 August to read again the leading article (p. 475) and the report on the Fifth Biennial Council Meeting of the C.M.A. in Kuala Lumpur and Singapore (p. 516).

It is always good to read important documents twice, as I am sure you will agree. This time I have been struck by the fact that you made much of the dust and omitted the concrete basic fact. In the report it is stated "that the Commonwealth Foundation had proposed that in the event of the Association finding it possible to broaden its membership a three-year grant of £30,000 would be made to it on certain conditions." Your report does not state what the "certain conditions" were. Why did you not state that the conditions attaching to the grant were that South Africa and Rhodesia should no longer be members of the Commonwealth Medical Association? Did you think that your members would consider it to be a "bribe" with strings?

All the dust of your report seems successfully to have disguised this basic fact, but surely the older members of the Commonwealth Medical Association should have known that if the Medical Associations of South Africa and Rhodesia had been told of this possible gift and the strings attached they would have withdrawn from the Commonwealth Medical Association, regretfully it is true, but knowing that to do so they would have materially helped the parent association.

What a pity that the whole matter could

not have been treated openly and honestly instead of airing the views of people who really know nothing of South Africa or Rhodesia except possibly from distorted hearsay, and what a dust they did raise!—I am, etc.,

ARNOLD H. TONKIN.

Pretoria, South Africa.

*. Our report of the Fifth Biennial Council Meeting of the C.M.A. (29 August, p. 516) included the following: "Three business meetings were held at Singapore, and were concerned largely with the question of membership of the Commonwealth Medical Association by the Medical Associations of South Africa and Rhodesia. The C.M.A. had received a letter from the Commonwealth Foundation (which has given financial support to it), and the question had been raised at the Kuala Lumpur meeting whether the Commonwealth Foundation should or would continue to support an association that included representatives from South Africa and Rhodesia. The debate opened by Dr. D. L. Kippen (Canada) suggesting that the letter from the Commonwealth Foundation should be separated from the discussion on membership of the C.M.A." It was after this that the resolution proposing omission of South Africa and Rhodesia was introduced. The motion itself, which we gave in full, also referred to financial support from the Commonwealth Foundation.—Ed., *B.M.J.*

Discretion and Ethics

SIR,—Dr. S. S. Vazifdar's proposition (17 October, p. 182) that it is a gross breach of medical ethics for doctors to discuss their patients with their wives would not be so bad were it not for his attempt to support it by a travestied account of the Kitson v. Playfair action.

Mrs. Kitson, the plaintiff in the action, was the wife of Mrs. Playfair's younger brother. This brother, a remittance man, settled in Australia and in late 1892 shipped Mrs. Kitson back to England. She was given no money by her husband and lived in London on an allowance provided by her brother-in-law, Sir James Kitson, a Liberal Member of Parliament.

In January 1894 after a persistent illness Mrs. Kitson insisted that Dr. Playfair be called in to assist her regular general practitioner in arriving at a diagnosis. What they saw decided both men that Mrs. Kitson had had a miscarriage in the previous three months. Since her husband had been in the Antipodes for the past sixteen months there was only one conclusion to be drawn. Meanwhile the diagnosis was confirmed by a cousin, Dr. Hugh Playfair, who examined a part of the placenta under a microscope.

Playfair now realized that the family's good name in society was at stake, and deciding that neither he nor his wife could associate any longer with an adulteress he felt forced to tell his wife exactly why. Mrs. Playfair felt that her brother Sir James, the source of Mrs. Kitson's allowance, had a right to know and he too was told. Publication of the allegations seems to have been thus limited and there was none of the

gossiping among woman-friends that Dr. Vazifdar asserts.

At this stage Mr. Kitson returned from Australia intent on supporting his wife's story, and in February 1895 Mrs. Kitson initiated proceedings against both Dr. and Mrs. Playfair for libel and slander. The case was heard the following year before Mr. Justice Hawkins, who Edward Clarke said was the worst judge he had ever known: "he had no notion whatever of what justice meant, or of the obligations of truth and fairness."

Rather than plead justification and damn the family name for ever, Dr. Playfair at the instigation of his legal advisers (who said that he would succeed on this ground alone) relied solely on the defence that his remarks were privileged: that is to say, that Dr. Playfair was duty-bound to tell his wife and that she was duty-bound to tell Sir James. This privilege could be rebutted by the plaintiff only if she could show that the defendants had acted maliciously.

To support Dr. Playfair three most eminent colleagues, Bland Sutton, Sir John Williams, and Sir William Broadbent entered the witness-box and stated that they would have acted just the same. The judge interrupted one of these men to ask rhetorically whether if a doctor found that a patient of his had been the subject of a recent abortion, the doctor was entitled to report his findings to the police. "I very, very much doubt it," said the judge.

The crux for Dr. Playfair came when counsel for Mrs. Kitson rose to cross-examine and the first question put was: "Do you still retain an opinion adverse to this lady's honour?" Knowing and believing what he did, Dr. Playfair could only answer yes. With the wretched doctor caught between his failure to justify his allegations in court and his refusal to withdraw them, the jury were invited to find that this could only mean that the words were spoken maliciously. This they did and awarded Mrs. Kitson damages of £12,000—a sum subsequently reduced on appeal to £9,200.

Your readers, Sir, may feel that Dr. Playfair was less the victim of his own breach of ethics and his wife's garrulous tongue than of some bad legal advice and his own Victorian sense of family honour. If they take that line, they will share the views of Mr. Gladstone who wrote to Dr. Playfair from Hawarden that he had been the victim of an astounding miscarriage of justice: "You do not seem to have done either more or less than your duty."—I am, etc.,

YOUR LEGAL CORRESPONDENT.

¹ Playfair, G., *Six Studies in Hypocrisy*, p. 165. Secker and Warburg, 1969.

Pay of S.H.M.O.s and Medical Assistants

SIR,—The deliberate exclusion of nearly all S.H.M.O.s and most medical assistants from participating in the recent pay awards must only be interpreted as an act of callous indifference towards these belaboured groups.

What other profession, let alone trade union, would tolerate this selective victimization of a minority group? I am surprised that the British Medical Association can countenance it.—I am, etc.,

T. L. HILLIARD.

Newport, Mon.