Diabetic Nephropathy

SIR,—Your leading article (5 July, p. 5) carefully avoids the important problem of pathogenesis by substituting a plea for study of the "natural history." I also would wish to avoid discussion of the interesting biochemical theories which will account for the thickened permeable basement membrane. However, I wish to draw attention to other points which may truly lie at the root of the matter. The first is that in animal experiments I have shown that the mesangial cells show a diminished phagocytic capacity in streptozotocin-induced diabetes.¹ This could explain why fibrinoid material accumulates in the mesangial areas.

You note in your leading article that there comes a stage when a downhill course is certain. Dr. George Drivas and myself think that we have identified such a stage because our data on the clearances of microaggregated iodinated human serum albumin show a difference between diabetics without renal disease and those with established nephropathy. This may be briefly summarized as follows:

Half Life in Minutes of Microaggregated Albumin

8 "normal" diabetics	12.6±2.0	} $t=3.74$ for 13 d.f. $P<0.005$
7 K.W. diabetics	18.7±3.6	

Thus in these patients there is an impairment of the phagocytic capacity of the Kupffer cells of the liver. As yet the reasons are speculative, and we are still performing more detailed kinetic studies. However, it might mean that these cells are loaded with lipoproteins. It might also indicate a general indisposition of phagocytic cells in diabetes and therefore an enhanced susceptibility to infection. Indeed, it is well known that leucocytes function at a disadvantage even early in diabetes. It may be that we are now able to identify those patients who have entered the "vicious cycle."—I am, etc.,

E. N. WARDLE

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¹ Wardle, E. N., *Biomedicine*. In press, 1975.

Fifty Years in Tavistock Square

SIR,—The article Fifty Years in Tavistock Square (12 July, p. 90) calls to mind the controversial subject of the Epstein statues. When the third B.M.A. House at 429, Strand, was reconstructed, Jacob Epstein was commissioned to erect a series of statues on the front facing Agar Street at the level of the first floor. When these were revealed to the public there was at once a flood of violent criticism. They were condemned by a large number of critics as hideous and indecent. The sculptor at this time was only little known and his work was not appreciated as it subsequently came to be. In this case the B.M.A. was in advance of its time.—I am, etc.,

C. J. GORDON TAYLOR

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Abortion (Amendment) Bill

SIR,—May I, through you, invite doctors or nurses to write to the Select Committee giving their views on the proposed alteration of the 1967 Abortion Act? Especially,

as a member of the Select Committee, I should be interested to have views on the impact the phrases "grave risk to the life of the pregnant woman," and "risk of serious injury to the physical or mental health of the pregnant woman."

What the sponsors intend by these phrases is that Parliament wants to make it clear that "abortion on demand" was not the intention of the 1967 Act. Indeed, the sponsor, Mr. David Steele, introducing his Bill used these words: "It is not the intention of the promotion of the Bill to leave a wide open door for abortion on request."¹—I am, etc.,

JAMES WHITE

House of Commons,
London S.W.1

¹ *Hansard*, House of Commons, 22 July 1966, col. 1075.

SI Units

SIR,—Hospital districts are receiving the circular H.S.C. (1S)140 (Metrication: Introduction into Medicine of the International System of Units) stating this should be implemented by 1 December 1975. This is in response to recommendations of a working party of eight bodies "representing scientific interests," and there appears to have been no consultation with the royal colleges (except that of the pathologists) or those in clinical charge of patients. In some instances SI units have nothing to do with metrication whatsoever.

This is being rushed through without adequate consultation, and clinicians should express their views strongly at district level before this becomes fait accompli, or else, like the recent reorganization, it will be too late to influence the course of events. Meanwhile SI units should not be accepted in hospital practice until there is a clear consensus opinion of their value and dangers expressed by clinicians with direct responsibilities for patients.—I am, etc.,

A. N. G. CLARK

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Brighton

Pyridoxine and Oral Glucose Tolerance in Pregnancy

SIR,—We should like to congratulate Drs. H. J. T. Coelingh Bennink and W. H. P. Schreurs on their study of the effect of pyridoxine on gestational diabetes (5 July, p. 13). Their view of the possible role of this vitamin in preventing insulin antagonism is extremely interesting, especially at a time when we are beginning to realize that the resistance to insulin on the peripheral uptake of glucose found in pregnancy¹ does not apply to its antilipolytic actions in adipose tissue from pregnant women (T. M. Coltart and Christine Williams, unpublished findings).

We feel, however, that another explanation of the effect of B₆ administration on glucose tolerance is possible. It can be seen that in situations where there is a relative deficiency of pyridoxine the production of nicotinic acid via this pathway is reduced. Since nicotinic acid is a potent antilipolytic agent² free fatty acid output in this situation could be enhanced. As the authors stated, free fatty

acids have been suggested as a possible insulin antagonist³ and could be responsible for the development of insulin resistance. The administration of B₆ with the concomitant increase in nicotinic acid production may result in a fall in free fatty release from adipose tissue, thereby removing another possible insulin antagonist.

Furthermore, we should like to sound a word of caution concerning the possible influence of diet and changes in weight on their results. They state on the one hand that patients were put on a diet and in the next breath they renounce any effect, in their hands, of diet on glucose tolerance. The example they cite of a 27-year-old para 2 who lost 2.5 kg in weight between 29 and 36½ weeks is an unphysiological situation by obstetric standards. Since both diet and weight changes are known to influence oral glucose tolerance we feel these factors should be taken into account before coming to any definite conclusions.—We are, etc.,

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CHRISTINE WILLIAMS

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Guy's Hospital Medical School,
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¹ Burt, R. L., and Davidson, I. W., *Journal of Obstetrics and Gynecology*, 1974, 43, 161.

² Butcher, R. W., Baird, C. E., and Sutherland, E. W., *Journal of Biological Chemistry*, 1968, 243, 1705.

³ Randle, P. J., *et al.*, *Lancet*, 1963, 1, 785.

Cigarette Smoking and Plasma Cholesterol

SIR,—The plasma cholesterol levels that we found in a study of 30 subjects who had been smoking cigarettes for over one year are interesting.

Blood samples were collected in the post-absorptive state after overnight abstinence from smoking.¹ The subjects were then asked to smoke one cigarette of their own brand. Immediately after and again after 90 minutes blood samples were collected and analysed for plasma cholesterol. There was a statistically significant increase in plasma cholesterol from the basal presmoking level (t by null hypothesis is 10.90, $P<0.0001$). After 90 minutes cholesterol levels in more than 65% of cases were still a little higher than the basal levels (t is 2.068 and $P=0.05$).

We know of no report about the statistically significant increase in plasma cholesterol immediately after smoking one cigarette in humans except that of Short and Johnson,² who reported changes in blood cholesterol for 60 to 90 minutes in five normal habitual smokers while they were smoking cigarettes. There was no mention of the number of cigarettes smoked. A slight tendency to an increase in cholesterol during tobacco smoking was noted.

Recently there was a report on cigarette smoking and serum chemistry by Dales *et al.*³ Their experimental situation was different from ours. They compared age-specific cholesterol values in White and Black smokers and non-smokers. They observed that cholesterol levels were higher in the White men who smoked but not in the Black male smokers. We report our results, firstly, because of the association of smoking cigarettes with an immediate rise in plasma cholesterol, and, secondly, to show that if the basal level is to be obtained from a cholesterol estimation the subject must refrain from smoking for a period of at least