any other anaesthetic agent" stresses the need for a detailed presentation of the methods used by the committee to ensure the scientific validity of the findings reported. Surely jaundice must have been observed after anaesthetics other than halothane in the United Kingdom during the nine-year period studied.

With regard to the "Results" section of the report we are, of course, particularly interested in the statement that "the mean interval between the last exposure to halothane and the onset of jaundice was very significantly shorter for patients exposed two or more times." In view of the unquestioned qualifications of the statistical consultants available to the authors we believe that it is inappropriate for them to make such a statement without a clear description of the statistical test applied to the data and the level of probability (P value) achieved. An open discussion of the statistical aspects of this report, lacking as it does any information on the incidence of the complication and any information relating to the overall safety of the drug, might prove to be in order and to be helpful to clinicians attempting to draw appropriate clinical inferences.

While on this point we feel constrained to comment on the letter sent to physicians in the United Kingdom by the Committee on Safety of Medicines on 3 January 1974. In this letter it is stated that the committee encouraged Dr. Inman and Professor Mushin to publish their report "so that anaesthetists might be made aware of this additional evidence when assessing all the relative risks of the various anaesthetic agents in the clinical circumstances of each case." We believe that such evidence can be accepted as scientific knowledge only after it has been held up to the scrutiny of the scientific community at large and has been accepted by it. Therefore until such time as our questions, and probably those of other interested parties, have been answered we believe that no one is in any position to evaluate the report, let alone apply the information contained in it during an assessment of "all the relative risks of the various anaesthetic agents in the clinical circumstances of each case." It remains a distinct possibility that the overall safety of halothane outweighs any hepatic or other toxicity. Unfortunately, the report by Dr. Inman and Professor Mushin cannot, by its very nature, illuminate that paramount question.—We are, etc.,

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Diabetes Mellitus and Infectious Hepatitis

SIR,—I have read with interest the article by Dr. F. C. Adi on "Diabetes Mellitus Associated with Epidemic of Infectious Hepatitis in Nigeria" (2 February, p. 183) in which he reported nine patients who developed diabetes mellitus. I am surprised that Dr. Adi had "been unable to find in the English language medical literature any record of

cases of diabetes mellitus associated with infectious hepatitis." In April 1970 there was published a detailed report from this hospital of seven Nigerians who developed diabetes mellitus in association with infectious hepatitis.¹ "Hepatogenous" diabetes mellitus—that is, diabetes mellitus in association with parenchymal liver disease—was recognized as long ago as 1906 by Naunyn,² who introduced the term.

In addition to the possibilities mentioned by Dr. Adi to explain the association of diabetes mellitus with parenchymal liver disease another factor may be the "bypassing" of the liver by endogenous insulin.^{3,4} Of the seven patients we reported, none showed clinical or biochemical evidence of pancreatitis. No correlation was found between the severity of the liver disease, as judged by either the serum bilirubin or aminotransferase levels, and the severity of the diabetes. Six of the seven patients developed diabetes after the age of 40 and have since been well controlled on chlorpropamide. The other patient, 19 years old at the onset, has been maintained on insulin (I.Z.S.) 40-60 units daily.

It is likely that Dr. Adi's patients have latent diabetes and that viral hepatitis, like any acute illness, precipitated overt diabetes mellitus. Follow-up of his patients may support this hypothesis.—I am, etc.,

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1 Francis T. I., Osuntokun, B. O., *Journal of the Nigeria Medical Association*, 1970, 7, 47.

2 Naunyn, A., *Der Diabetes Mellitus* (1906), quoted in *Disturbances in Carbohydrate Metabolism and its Disorders*, ed. F. Dickens, P. J. Randle, and W. J. Whelan, vol. 2, ch. 12. London, Academic Press, 1967.

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Gestational Diabetes

SIR,—We were interested to read your leading article on this subject (2 February, p. 167) and should like to comment on some aspects.

There is already a state of confusion in the terminology of diabetes in pregnancy, but this has been made worse by the incorrect use of the term "latent diabetes" in your first paragraph, where it is stated that "latent diabetes" defines the situation in which a normal glucose tolerance test becomes abnormal temporarily during some kind of stress." In fact latent diabetes, as agreed by the Medical and Scientific Section of the British Diabetic Association,¹ is the situation in which a person has a normal glucose tolerance test after having had a temporary asymptomatic or clinical diabetes

at some time during pregnancy, infection, or other stress or when obese. It follows that a latent diabetic in pregnancy has by definition a normal glucose tolerance at that time.

The British Diabetic Association classification did not include a definition of "gestational diabetes." According to the use of the term applied in two main centres in Britain it is impossible to diagnose gestational diabetes until it has been found that the glucose tolerance has returned to normal post partum. In the absence of such a postpartum test there is no possibility of making the diagnosis of gestational diabetes in this system of nomenclature, as indeed is stated in the final paragraph of your article. We therefore suggest that one should recognize that diabetes has been diagnosed during pregnancy at that time irrespective of what happens postpartum and use the term "gestational diabetes" for this situation, for it has important obstetric consequences. "Gestational" in this context merely describes the time of diagnosis of the diabetes and not its degree, so that it would be appropriate to subdivide gestational diabetes into chemical (subclinical, asymptomatic) or overt (clinical).

Further confusion may arise from your article in the definition given of "prediabetes," stated by you to be "the time before diagnosis of overt diabetes." We submit that the diagnosis of prediabetes is retrospective and refers to the period before clinical or chemical diabetes has been diagnosed. Otherwise according to your definition an individual could have chemical diabetes during the prediabetic period.

We have tried to clarify our suggestions in the accompanying diagram and hope that this or an improvement on it can be adopted by the Medical and Scientific Section of the British Diabetic Association to avoid the sort of ambiguity which occurred before their definition of the stages of diabetes was published in 1964.¹

Returning to the content of your article we would like to make points about two of the items it covered.

To your reference to the work done in Aberdeen on the significance of the individual indications for testing these potential diabetics we should like to add that there is a significant correlation between the number of indicators present in any pregnant woman and the likelihood of her being diabetic.^{2,3} There appears also to be a correlation between the number of indications to test and an increased perinatal mortality, which is higher if there is diabetes, but still present if there is none. In other words even potential diabetes in the mother carries an increased risk for the baby. Thus in centres where no glucose tolerance testing facilities are available the obstetrician should heed

this risk—and at the least avoid prolonged pregnancy.

The second item concerns our double-blind trial of chlorpropamide 100 mg a day in the treatment of gestational chemical diabetes in women of average body weight. We do know that such a dose of chlorpropamide is effective in reversing chemical diabetes in the mother in pregnancy—without apparent harm to the fetus⁴—though we do not recommend this form of treatment for diabetes of a more severe degree, which seems to require more delicate control during pregnancy.⁵—We are, etc.,

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- 1 Fitzgerald, M. G., and Keen, H., *British Medical Journal*, 1964, 1, 1568.
- 2 Sutherland, H. W., Stowers, J. M., and Whetham, J. C. G., *Excerpta Medica International Congress Series*, 1973, 280, 153.
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- 4 Sutherland, H. W., Stowers, J. M., Cormack, J. D., and Bewsher, P. D., *British Medical Journal*, 1973, 3, 9.
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Temperature Change and Multiple Sclerosis

SIR,—Within a period of one year, two leading articles in the *B.M.J.* dealing with multiple sclerosis have presented incomplete accounts of our current understanding of the mechanism of temperature effects in this condition.

The omission in the first of these leading articles (2 December 1972, p. 506) was pointed out by Drs. W. I. McDonald and T. A. Sears in a letter (30 December 1972, p. 794) in which they summarized experimental studies in demyelinated nerve indicating a heightened sensitivity to thermal block,^{1,2} which by itself gives a sufficient explanation of the clinically observed temperature effects. In the more recent leading article (15 December 1973, p. 626) this clinical temperature phenomenon is discussed in an identically incomplete manner. I do not intend to repeat the contents of the earlier letter from Drs. McDonald and Sears; however, an additional point can be made.

In a recent study³ the thermal properties of demyelinated nerve have been extensively investigated by the numerical solution of a well-known set of differential equations describing conduction in a model demyelinated axon. In agreement with clinical findings in multiple sclerosis and experimental animal studies this model predicts that the temperature at which conduction block occurs is a steep function of the extent of demyelination alone, so that small temperature increments can block large numbers of conducting fibres. To put the situation in another way, given a population of nerve fibres with varying amounts of myelin loss (such as would be expected to occur in a real lesion in multiple sclerosis) a small increase in temperature would substantially decrease the number of conducting fibres whose blocking temperature exceeds the normal body temperature.—I am, etc.,

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- 1 Davis, F. A., and Jacobson, S., *Journal of Neurology, Neurosurgery and Psychiatry*, 1971, 34, 551.
- 2 Rasminsky, M., *Archives of Neurology*, 1973, 28, 287.
- 3 Schaaf, C. L., and Davis, F. A., *Journal of Neurology, Neurosurgery and Psychiatry*. In press.

Changing Incidence of Congenital Pyloric Stenosis

SIR,—The letter from Dr. D. E. Murfin reporting an increasing incidence of infantile hypertrophic pyloric stenosis in Barbados (2 February, p. 198) is of some interest because it notes a contrary trend to that which is occurring in most European countries. In Sweden the frequency fell from 4 to 2 per 1,000 live births during a decade.¹ In Belfast County Borough the incidence fell from 3.09 per 1,000 live births in the 7-year period 1957–69 to 2.22 per 1,000 live births in the 6-year period 1964–9 ($\chi^2 = 6.2$; D.F. = 1; $0.025 > P > 0.01$).² A superficial observation of the numbers of affected infants seen at Llandough Hospital, Cardiff during the past two decades shows a similar decrease. In both Belfast and Cardiff there was a rather striking increase in numbers in 1962 which is unexplained. In contrast Strödm³ reported an increase in the number of cases seen at a hospital in West Germany, but this was not related to population incidence and may be explicable in other ways.

Despite Dr. Murfin's observation that Negro infants may suffer from this condition more commonly than we have supposed and his suggestion that the rising incidence may reflect environmental changes there is little doubt that racial differences in incidence are at least partly genetically determined. An important study in Hawaii recorded the relative incidence in different racial communities. The preponderance of Caucasians was confirmed and no instances were detected among 11,274 Chinese infants. The authors claimed that cultural differences were of minor importance in Hawaii, that medical facilities were shared, and that their findings reflected variations in susceptibility which were determined by race.⁴—I am, etc.,

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- 2 Dodge, J. A., M.D. Thesis, University of Wales, 1970.
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Irresponsible Poisoning

SIR,—I was very pleased to see that you have returned to the problem of childhood poisoning in your leading article "Irresponsible Poisoning" (23 February, p. 296). I agree entirely with your conclusion that the dangers of tricyclic antidepressant drugs need to be made clear to all who prescribe them, but was disappointed to see that you decry the use of child-resistant containers. There is very good evidence that the use of these containers would reduce the risk of poisoning by tablets as much as by 90%.^{1,2} It is no good relying on pious exhortations to keep medicines out of the reach of children and it is time that positive action was taken. You are also wrong in saying that

"most children are at least as resourceful as their parents." At the age of 1–3 years they certainly are not, and this is the age at which poisoning occurs.

What worries me, as a member of the committee of the British Standards Institute concerned in the drafting of the protocol for testing child-resistant containers (not the actual design of the containers as your article states), is that so far as I am aware no further action has occurred since the draft was published. Meanwhile the poisoning goes on.—I am, etc.,

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- 2 Scherz, R. G., *Pediatric Clinics of North America*, 1970, 17, 713.
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Gamekeeper's Thumb on the Ski Slopes

SIR,—Your leading article (9 February, p. 213) notes yet another type of skiing injury. However, I cannot see how extension of the thumb can damage the ulnar collateral ligament. I believe that, like gamekeeper's thumb, the loose grip is to blame and that this injury can be prevented if bindings have been checked.

The ski stick is normally held through a leather loop on top of the handle that is wrapped around the thumb (see fig.). This loop's main purpose is to prevent loss of

the ski stick in soft snow. If the other fingers do not grip well, twisting and jarring movements when skiing are transmitted through the thumb, which is caught between the stick and the taught loop. The ulnar collateral ligament of the thumb is weakened and later tears on falling (often still trapped in this position).

Skiers can prevent this injury by gripping the ski stick harder—even when they fall. At times the stick can be held without using the loop—for example, on a dry ski slope. Since adopting these simple measures I myself no longer suffer from painful thumbs after skiing.—I am, etc.,

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