receptive to new ideas, will be interested in the formation of the society and any guidance it can give them.

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SIR,—It is somewhat unfair to the health visiting profession to assume, as Dr R P Snaith is tempted to do (14 August, p 512), that the absence of specific mention of postnatal depression in Shirley Goodwin's article (17 July, p 182) indicates a lack of concern with psychiatric disorder on the part of health visitors in general. As part of a programme of research undertaken here by the General Practice Research Unit, and funded by the Department of Health and Social Security, we are currently studying health visitors in one London borough, with particular reference to their role in the identification and management of psychosocial problems in the families they visit. Our preliminary results indicate that health visitors do not concern themselves merely with the detection of abnormalities in child development and with physical health but are keenly aware of the significance of emotional problems and of psychiatric ill health in families.[1] Indeed, the support and encouragement for this research provided by the Health Visitors' Association as well as the local health visitors themselves is, we feel, further evidence that the health visiting profession shares Dr Snaith's concern with the need to improve training and practice in this area.

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[1] Briscoe ME, Lindley. *Health Visitor* 1982;**55**:165-8.

Childhood drownings in private swimming pools: an avoidable cause of death

SIR,—We would endorse the suggestion by Dr W Barry and his colleagues that private swimming pools be required by law to be safely enclosed (21 August, p 542). This would also increase awareness of parents and the public about the hazards of drowning presented to toddlers in many other situations.

Between April and October 1981 four children under 3 years old were admitted to the Royal Berkshire Hospital, Reading, after drowning or near drowning. One had found his way through shrubs into an incompletely fenced private pool and died. One had fallen into a shallow garden pond and survived with severe brain damage. Two survived without brain damage: one was recovered from a private pool and the other from a bath, having been left unattended briefly.

One of us (R S M-K) has constructed a raised private pool which is cheaper and safer than the conventional sunken concrete pool (fig). Its walls are four feet high and access is by a three-step ladder from which the middle rung is removed so that, even should the ladder inadvertently be left down, a toddler would find it impossible to climb in.

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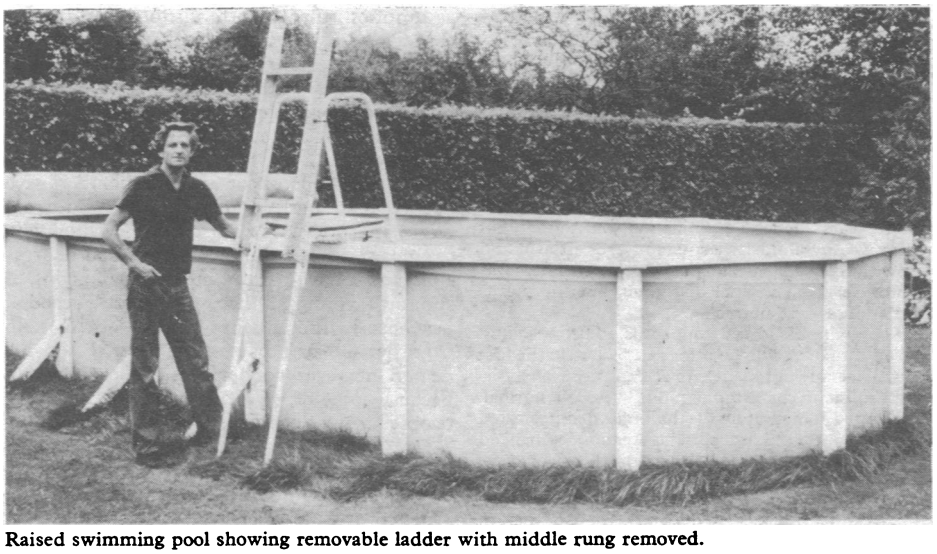

Raised swimming pool showing removable ladder with middle rung removed.

ABC of Diabetes

SIR,—In his admirable series of articles on the ABC of Diabetes, Dr Peter J Watkins rightly stresses the increased need of insulin "even when appetite declines and vomiting begins" (31 July, p 360). I hope he will not mind me pointing out that this need is even more pressing when the vomiting is accompanied by diarrhoea, as in attacks of gastroenteritis, which are so liable to precipitate ketoacidosis in diabetics.

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SIR,—We read with interest the article by Dr Peter J Watkins on unstable insulin-dependent diabetes (17 July, p 192) and wonder whether the following points might be contributory.

Firstly, the definition of brittle diabetes needs to be standardised. If this is taken to be wide, unpredictable swings in blood glucose and ketones leading to severe disruption of life then (as Dr Watkins points out) there are many possible causes, including inappropriate treatment by the physician, deliberate or accidental errors in diet and insulin dosage on the part of the patient, intercurrent illness, menstruation, etc. We would like to summarise the evidence that, in addition to the above causes, there exists a "special type" of unstable diabetes (whose existence is doubted by Dr Watkins and others), which we believe may be characterised by the patients' pathophysiological responses to subcutaneous insulin administration.

In the last few years we have investigated 18 "difficult" diabetic patients who were distinguished in that they remained uncontrolled on the ordinarily optimal treatment of continuous subcutaneous insulin infusion, which argued against treatment errors as the cause of their instability. They were aged 12-37 years (mean 19 years) and all but one were women. Seventeen had been referred to us by other hospitals. On switching to intramuscular[1] or intravenous insulin infusion, metabolic control was immediately and greatly improved, making cheating unlikely and pointing to a specific defect in subcutaneous insulin absorption. This hindrance to subcutaneous absorption is further suggested by the higher daily subcutaneous insulin dose in these patients who were unresponsive to continuous subcutaneous insulin infusion compared with that in a group of stable diabetic patients who were matched for age, sex and duration, of diabetes.[2]

In similar but perhaps not identical subcutaneously resistant diabetics this defect has been attributed to increased subcutaneous insulin degradation, largely on the indirect evidence that the protease inhibitor aprotinin lowers insulin requirements and improves metabolic control.[3] We have not, however, been able to show accelerated in-vitro insulinolysis in subcutaneous tissue biopsy specimens taken from some of our brittle patients compared with that in stable patients and normal subjects.[4] Abnormal vascular responses do, however, seem to be a feature of the brittle group. Using the method of photoelectric plethysmography to examine changes in blood flow close to superficial subcutaneous injection sites, we found that insulin injection causes local hyperaemia in stable diabetics and normal subjects, but not in brittle diabetics.[5] Since blood flow is thought to be a major determinant of insulin absorption,[6] failure to increase local blood flow in these patients could therefore impair absorption and contribute to their instability. Unabsorbed injected insulin, sequestered subcutaneously, could be subject to partial degradation or be later irregularly absorbed, perhaps causing the unpredictable hypoglycaemic episodes characteristic of some brittle diabetics. Moreover, the beneficial effect of aprotinin in some of these patients may be due to the powerful local hyperaemia at its subcutaneous injection site,[7] rather than to inhibition of insulin breakdown.

Other mechanisms, such as metabolic hypersensitivity to emotion, stress, relative insulin deficiency, etc, may, of course, operate as well, and could modulate and even cause impairment of subcutaneous insulin absorption, by, for example, influencing injection-site blood flow. Cheating may also coexist, as patients who are frightened by their unpredictable diabetes and its medical and social consequences may be tempted to engineer readmission to the relative security of hospital.

The natural history of this type of "difficult" diabetes is obscure. The finding of only a few patients in older age groups may be due to spontaneous resolution of the brittleness, as Watkins suggests, or to an early fatal outcome of multiple emergency hospital admissions. In our series, no patient has died, although most have required treatment in the intensive care unit, but only one has remitted and the duration of brittleness has been up to 22 years. There is therefore a need for a largescale longitudinal study of the course of brittle diabetes.

The management of these patients is difficult and demanding, and the various options are too complex to discuss in detail

here. Not only does insulin treatment present problems, but most patients also have considerable emotional problems related to chronic ill-health, fear of dying, loss of employment, inability to keep up at school, conflict with the family, and so on. Although brittle diabetes unresponsive to continuous subcutanecus insulin infusion is rare, we feel that it is a definite and important entity with enormous impact on the life of the patient and on health service resources. Study of its pathophysiological basis may also help us understand the lesser degrees of unexplained instability which trouble many insulin-dependent diabetics.

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[1] Pickup JC, Home PD, Bilous RW, Keen H, Alberti KGMM. *Br Med J* 1981;**282**:347-50.
[2] Pickup JC, Williams G, Keen H. *Proceedings of the international symposium on artificial systems for insulin delivery*. New York: Raven Press (in press).
[3] Freidenberg GR, White N, Cataland S, D'Orisio TM, Sotos JF, Santiago JV. *N Engl J Med* 1981;**305**:363-8.
[4] Pickup JC, Williams G, Keen H. *Diabetologia* (in press).
[5] Williams G, Clark AJL, Cooke E, Bowcock S, Pickup JC, Keen H. *Diabetologia* 1981;**21**:516.
[6] Lauritzen T, Binder C, Faber OK. *Acta Paediatr Scand* 1980;**283**, suppl:81-5.
[7] Williams G, Pickup JC, Bowcock S, Cooke E, Keen H. *Diabetologia* (in press).

Impact of maternal serum alpha-fetoprotein screening on antenatal diagnosis

SIR,—With reference to the paper by Dr David J H Brock (31 July, p 365) on the impact of maternal serum alpha-fetoprotein screening on antenatal diagnosis, I wish to make the following points:

Firstly, only 10% of mothers who underwent amniocentesis because a raised maternal serum alpha-fetoprotein concentration had been discovered had a spina bifida pregnancy. In other words, 90% of amniocentesis tests were unnecessary as the fetal loss and serious morbidity after amniocentesis is at least 2%.[1] Dr Brock makes no mention of the perfectly normal fetuses that must have been aborted in his series. Secondly, even after amniocentesis, three of the pregnancies aborted were not spina bifidas, which represented a false-positive rate of approximately 5%. Thirdly, what Dr David Brock fails to mention is that half of all spina bifida pregnancies are stillborn and a further half die in the first month of life and any "success" that a maternal serum alpha-fetoprotein concentration screening programme produces has to be judged in the light of these facts.[1] Finally, the whole question of antenatal screening invokes great anxiety in mothers and I simply do not believe that the results that he has reported justify the wholesale anxiety that is caused by offering routine antenatal screening. In my own practice, where I have carefully counselled all pregnant women at an early stage of pregnancy, well below half wish to proceed with routine antenatal screening for serum alpha-fetoprotein concentration. In the opinion of my patients the results of the screening programme are simply not worth the increased risk of losing a normal pregnancy.

In summary, therefore, I think that Dr Brock's conclusion that his figures constitute a strong case for the continuation of an alpha-fetoprotein screening programme is totally false. What he has done is looked at the abnormal fetuses that have been terminated and sought to justify the screening programme from these results. If all patients entering a casualty department had laparotomies to see whether they had ruptured spleens without doubt some ruptured spleens would be diagnosed earlier but at tremendous cost and suffering. In my opinion this is what is occurring with routine antenatal screening, and for Dr Brock to try to extrapolate from his results that it is worth while continuing with this screening is totally false.

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[1] Working Group on Screening for Neural Tube Defects. *Report*. 1979: 16, 27. (Black Report.)

ZX81 epilepsy—a case for lumping

SIR,—The rapid multiplication of species from the genus "space invader epilepsy"[1] is getting out of hand. We believe that all these entities should be lumped together with photoconvulsive epilepsy, rather than split into many syndromes, each with the rubric of the particular piece of electronic hardware that precipitates the seizure. The following case of a seizure induced by a home computer illustrates our point.

A 15-year-old boy was referred to the neurological clinic because of a seizure while playing with a microcomputer which was on display in a shop. He had been programming the computer, a Sinclair ZX81, without ill effect but when he set the program to run using the fast compute mode (which generates a flashing irregular zig-zag pattern across the screen during the computation), he began to feel dizzy, then, according to the witness, abruptly lost consciousness and fell to the ground, with twitching of all four limbs. He regained consciousness after a few minutes, but felt nauseated and had a headache; he had not bitten his tongue or been incontinent of urine.

He had been admitted to hospital 14 months earlier after a blackout while playing football. In that attack he had experienced a similar kind of dizziness, followed by abrupt loss of consciousness, and twitching of his arms and legs. He became conscious again after some 30 minutes; at the hospital no abnormal neurological signs were found. An electroencephalogram recorded a few days later showed a photoconvulsive response to photic stimulation. The event was therefore diagnosed as a probable seizure, and no treatment was given. He remained well with no further attacks until his recent computer-induced seizure, and he had played space invaders without ill effect. He had not, however, used this particular computer before.

We think that this case should be lumped with space invader epilepsy and electronic space war video game epilepsy (12 June, p 1751) and be classified as photoconvulsive epilepsy rather than be split off into the new nosological entity of ZX81 epilepsy. This, we hope, will stem the flood of specific epilepsies that could be described if each piece of electronic hardware on the market now or in the future had a form of epilepsy named after it.

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[1] Rushton DN. *Lancet* 1981;i:501.

Manpower proposals

SIR,—I have voted against in the ballot on the manpower initiative of the CCHMS. This is not because I regard the Short proposals as satisfactory—indeed, if they were implemented in full they would be disastrous. The proposals of the CCHMS are a little better in that they will ameliorate but not solve the manpower problem.

There is no doubt to my mind that the manpower problem will be solved only if two things are accepted: firstly, that there is a service requirement for junior staff; and, secondly, that there must be two career grades of hospital doctors with full clinical responsibility.

At SHO level it should be accepted that time should be spent in a job with primary service commitments. There is a large service commitment in the present SHO posts. It is not unreasonable to ask doctors who have been recently trained, largely at the public's expense, and who are paid a full salary to commit some of their time to wholly service posts. The serious consequences of a post not being approved for training result in low standards of approval. The acceptance of a period in service posts would remove the disastrous effect of non-approval of their posts for training purposes on the affected hospitals and their services. This would enable more realistic assessment of those posts that are indeed worthy of the title of training post.

I sympathise with the CCHMS's proposals about senior registrars but would like to see one grade at the registrar level which would include the present registrar grade and the lower half of the present senior registrar grade.

At senior level there should be two grades of hospital doctor. Clinical responsibility would be taken at specialist level, a grade through which all doctors would pass. They would work entirely in hospital and be responsible for inpatient and outpatient care which requires technical skill rather than consultant opinion and be fully clinically responsible for their patients in the same way as general practitioners are. Recruitment of consultants would be from this grade. They would perform a consultant function to their specialist and general practitioner colleagues and to the health authorities. Only by competition to attainment to this grade will standards be maintained.

It is essential in a competitive structure in a profession that not everyone reaches the top but equally that those who fail to do so have a satisfactory career. Those of us who have been on appointment committees sometimes see people who are medically adequately trained but probably lack the ability, drive, or the will to perform the non-clinical tasks expected of a consultant or director of department. There is no place for these doctors to go now in the absence of another career grade, particularly as it is no longer practicable for them to transfer easily into general practice with its strict training requirements.

Those in the specialist grade would have to be prepared to do more cover, which would mean immediate cover though not necessarily living in. Older specialists who did not make the consultant level would be able to transfer to service appointments—for example, specialist clinics—which would not require the same acute cover as when they were