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Social and functional impact of minor fractures in elderly people

SIR,—The findings of J M Nankhonya and colleagues on the social and functional impact of minor fractures in elderly people will not surprise those working in accident and emergency medicine.¹

A six month study in the accident and emergency department of Charing Cross Hospital showed that 55 of 66 patients (83%) aged over 75 who attended with minor fractures of the ankle, wrist, or humerus could safely be discharged within two hours, during the normal working day of 9 am to 5 pm, if a prospective nursing and social assessment was made by a senior nurse before discharge. The nurse could then liaise with local social services and voluntary organisations to ensure that support was increased from the time the patient returned home. During a control period when no such assessment was made only 25 of 57 patients (44%) with similar fractures could be safely discharged.

Obviously some patients present outside the normal working day, when it is not possible to arrange increased social support. In these circumstances an accident and emergency ward is invaluable in providing temporary care until these patients can be fully assessed and the appropriate services arranged the next day. In our experience, patients rarely need to stay more than 24 hours after their injury while this support is arranged.

A careful social and nursing assessment of elderly people who present with minor fractures is as important as the management of their injury itself. By assessing their needs while they are in the accident and emergency department and coordinating the response of community services it should be possible to ensure the safe discharge of such patients from hospital while at the same time avoiding their inappropriate admission to hospital.

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Intensified conventional insulin treatment and neuropsychological impairment

SIR,—Per Reichard and colleagues conclude that episodes of serious hypoglycaemia did not cause permanent cognitive impairment in diabetic patients in their study.¹ For several reasons we suspect that they might have made a type II statistical error.

Firstly, there was insufficient separation between the groups in the putative key causal variable. Although the manipulation of the groups was done on the basis of treatment schedule, the authors make repeated reference to only one causal variable, severe hypoglycaemia. The mean number of episodes per patient per year was 1.1 in the intensified treatment group and 0.4 in the standard treatment group. Further, 29 (56%) of the standard treatment group had one or more episodes of severe hypoglycaemia during the study compared with 34 (77%) of the intensified treatment group.

Secondly, the statistical power of the study was not high. The power of detecting a difference of 0.5 standard deviation between the two groups was about 0.67, and the power of detecting a difference of 0.3 standard deviation was only 0.30.

Thirdly, their battery of neuropsychological tests contained some of doubtful sensitivity. The authors claim that their tests were well suited to detecting cognitive deterioration and that some of the tests had proved sensitive to acute hypoglycaemia (their references 28-30). What these references show is that tapping and digit span tests were likely to be insensitive to any effects of hypoglycaemia on the brain. The Necker cube test is unknown to most neuropsychologists, and the peculiar results of the 1959 paper that were used to support its inclusion in the battery of tests baffled even the original authors.² The gross brain damage to which this test seemed to be sensitive in the authors' supporting reference bears no comparison with the subtle deficits that may be expected after exposure to severe hypoglycaemia.

Fourthly, five years is too short to afford a clear judgment about the effects of insulin treatment on cognitive functions.

Finally, few contrary positive results were discussed. Using retrospective reports of experience of hypoglycaemia, Langan *et al* showed a significant correlation between a decrease in IQ from premorbid levels and reported severe hypoglycaemia in 100 insulin treated diabetic patients.³ The mantra of "prospective good, retrospective bad" should not lead to this result being dismissed. The retrospective reports proved valid and highly reliable.⁴ The reports covered a mean of over 10 years, and patients were tested with an extensive battery of cognitive tests, including Wechsler's adult intelligence scale (revised).

Thus the negative result reported by Reichard and colleagues might constitute a type II error. This important issue has not been settled definitively, and we recommend caution to those who might be tempted to believe that repeated severe hypoglycaemia does not have detrimental cognitive effects.

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AUTHOR'S REPLY,—A statistical type II error is always possible in a study like ours unless the number of subjects is very large, but for several reasons we think it not very probable.

Firstly, the separation between the groups was such that the number of serious hypoglycaemic episodes per patient was almost three times higher

in the intensified treatment group. Our prime aim was to study the effects and side effects of an intensified treatment programme, not to see whether any number of serious hypoglycaemic episodes was harmful to the brain. We found that the improvement in the metabolic control achieved led to a retardation of diabetic microvascular complications.¹ This treatment, with the increased frequency of hypoglycaemic episodes observed, did not lead to cognitive deficits. This does not mean that serious hypoglycaemia, especially if accompanied by unconsciousness, never causes brain damage. It does cause such damage in rats, but only when the electroencephalogram is isoelectric.²

Secondly, the statistical power of our study was such that we ran some risk of statistical type II errors if we used only one or two tests on only one occasion. We used several tests, and some had previously been used after three years' study.³ There were no differences between the groups with regard to the results from any test at any time.

Thirdly, the sensitivity of our tests varied, and the most sensitive were probably the trail making test, the perceptual maze test, and the reaction time test with auditory inhibition. The Necker cube test should be seen only as a complement to other tests to determine whether a tendency towards frontal lobe damage concerns unilateral or bilateral changes.

Fourthly, five years of intensified treatment did not lead to any detectable cognitive deficit. Of course a longer period might be necessary, and we will follow up our patients for 10 years altogether.

Finally, to study changes over time you must perform prospective studies. Langan *et al*'s study was retrospective, and the authors can never exclude selection bias. Determining premorbid status retrospectively is associated with a great risk of errors.

In the Stockholm diabetes intervention study intensified insulin treatment led to a retardation of complications. The increased frequency of hypoglycaemic episodes (mostly without coma) did not cause any detectable brain damage. Frequent or prolonged hypoglycaemic unconsciousness might have detrimental long term effects and is also immediately dangerous and unpleasant. It should be avoided.

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Intravenous magnesium in suspected acute myocardial infarction

SIR,—K K Teo and colleagues describe the possible beneficial effects of intravenous magnesium in acute myocardial infarction.¹ Magnesium remains an enigmatic treatment. It has been used empirically to treat arrhythmias and began to be used for acute myocardial infarction comparatively recently. Nevertheless, no guidelines exist regarding the salt that should be used, the dose, the infusion times, and the duration of treatment. This is reflected in the different treatment schedules in the overview. At a practical level in different

hospitals different preparations (citing contents in g, mg, or mmol) are available. Treatment is often initiated empirically. This lack of familiarity with the drug is exemplified by the few data available in British textbooks; this contrasts with practice in North America, where intravenous magnesium has been used as an anticonvulsant in pre-eclampsia.

The mode of action of magnesium is unknown. A decrease in acute arrhythmias is unlikely to account for the decrease in mortality from acute myocardial infarction as most such life threatening arrhythmias are adequately treated in coronary care units. The increase in plasma magnesium concentration achieved in the studies cited varies but is slight. The effect that this may have on cellular processes is unknown. Basic investigations in isolated heart models have shown that a large increase in serum concentrations to about 15 mmol/l confers protection from ischaemia and improves energetic processes.^{2,3} These, however, are concentrations that would cause cardiorespiratory depression in humans.

What effect a short term increase in extracellular magnesium concentration has on intracellular free magnesium is also unknown. Exchange processes across the sarcolemma are species specific,⁴ and the characteristics of these in humans in physiological and ischaemic conditions are unknown. Intracellular magnesium rises during ischaemia and remains raised during reperfusion,^{5,6} which may inhibit uptake of calcium by sarcoplasmic reticulum with coincidental diastolic dysfunction.⁷ An increase in intracellular calcium may be deleterious, particularly during reperfusion. The effects of magnesium in acute myocardial infarction after treatment with thrombolytic agents may therefore be modified. Too large an increase in intracellular magnesium may have adverse consequences. Certainly, optimal mechanical recovery of a rat heart required magnesium concentrations in cardioplegic solutions to be centred on about 15 mmol/l: both higher and lower concentrations were associated with less than optimal recovery.⁸

Intravenous magnesium at differing doses may have a combination of beneficial and deleterious effects. Clearly we need to investigate and rationalise the use of magnesium rather than continue with empirical treatment.

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The first thyroid scan

SIR,—The obituary of Norman Veall credits him with having performed the first thyroid scan.¹ I do not wish to detract from his many

achievements, but this requires correction.

To the best of my knowledge I, with J Rotblat, performed the first thyroid scan in Liverpool University's physics department on 10 September 1947. We were able to demonstrate an intrathoracic extension of the thyroid.² The original notebook still exists.

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AUTHORS' REPLY.—We regret that shortage of space led us to oversimplify the history of thyroid scanning. Veall himself clearly acknowledged G Ansell's contribution.¹ We agree that Ansell and Rotblat did the first study, designed to locate the extent of a large retrosternal goitre leading to mechanical obstruction.² But the physical bulk of the Geiger-Müller type 4 counter, then the only type available, restricted them to measurements at nine locations chosen by the operators over a very large gland together with 23 background sites.

Veall's contribution was to design an elegant collimator, which improved the resolution of a then novel type of Geiger-Müller counter by a factor of six compared with Ansell and Rotblat's apparatus.¹ He used a Perspex jig drilled with locating holes at regular intervals, which in effect turned this apparatus into a hand driven rectilinear scanner. As Taylor and Stewart later showed, Veall's apparatus gave scope for imaging a much broader range of thyroid disease and removed the influence of unconscious operator bias in the generation of images.³

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The new disability living allowance

SIR,—I have just attended a seminar run by the Department of Social Security on the introduction of the disability living allowance. I am perturbed at the consequences of what is planned.

In April attendance allowance for the under 65s and mobility allowance will cease to exist and will be replaced by the disability living allowance. Attendance allowance continues in name for those over 65, but the application procedure still follows that for the disability living allowance.

Under the current system a person applying for mobility or attendance allowance has to fill in a short form and send it off. Every applicant is then visited by a doctor (usually a general practitioner), who fills in a four page report which forms the basis of the assessment. Under the new system the customers (as now called) fill in the form themselves, and this is the basis of whether the allowance is granted or not. The form is 26 pages long.

Currently, the report from the doctor is received and assessed by a doctor working for the Department of Social Security. In the new system the department's doctor is replaced by a lay person who has been trained in the application procedure but has no medical knowledge. If that lay person does not understand something he or she will

consult with a department doctor or write to other professionals concerned; if it is still not clear a doctor (still usually a general practitioner) will be asked to visit. It is estimated that only a fifth of applications will require a visit by a doctor. Visiting doctors are being told to stop using precise medical terminology and instead use terminology understandable to a lay person.

The money paid to the visiting doctor forms a tiny percentage of even just one year's allowance. I do not understand why such a valuable and cheap report is being abandoned. Also, I am concerned that the 26 page document will form an insurmountable hurdle to many, particularly uneducated people, elderly people, and the mentally ill. Applicants are losing the benefit of a consultation with, and a report from, a medical practitioner whereby the often subtle nuances of a disabling condition can be brought out and explained on their behalf. I would very much like this to work as there is too much at stake, but I have my doubts.

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Medicine in Europe

SIR,—The series of articles looking at medical issues in Europe is instructive, but I take issue with the way that Tessa Richards dismisses the activities of the royal colleges and, particularly, the European committee of the Royal College of Physicians. The European committee was not set up as a reaction to anything happening in other colleges, and the one thing it does not do is organise European scientific meetings and exchange visits.¹

The BMA has worked long and patiently in Europe, particularly through the Standing Committee of Doctors of the EC, which was established in 1959. But, as Richards stated in a news item² and is echoed in the sixth article in the series,¹ "Time and time again [the Standing Committee of Doctors of the EC] has been left shaking its metaphorical fist as directives emerge whose contents have at times been considerably at odds with the best interest of patients and the profession." Far from the BMA and the royal colleges competing in their activities in Europe, it is essential that they cooperate and complement each other's efforts. The colleges, however, have to find and pursue their own particular role in Europe, and the conference of colleges must use its organisation to coordinate those activities.

The Royal College of Physicians' European committee therefore has a major interest in the activities of the monospecialty sections of the European Union of Medical Specialists as well as the Advisory Committee on Medical Training. It is also keen to build up a profile of the various medical, and particularly medical academic, bodies in the various countries so that it can enter into discussion and dialogue with them.

The most difficult problem for the colleges, the BMA, and British government departments is to find ways of influencing the legislative system in Europe. Unfortunately, at present this seems unlikely to be achieved through the European Union of Medical Specialists, the Standing Committee of Doctors of the EC, or the Advisory Committee on Medical Training. If these bodies cannot be made more effective some different structure must be devised. Whichever way, we will certainly need to work together if we are to achieve any success.

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