occupational exposure to hand-held vibrating tools. Follow-up in 1988 by review of case notes and by questionnaire showed that 7 men (8 hands) had died. The study included 15 men exposed to and 28 men not exposed to vibrations (17 and 33 hands, respectively). Therapeutic results of surgical carpal tunnel decompression are shown below:

	Exposure	
Therapeutic result	*Yes*	*No*
Excellent	10 (59%)	26 (79%)
Relapse after initial recovery	3 (18%)	3 (9%)
No effect	4 (24%)	4 (12%)
Total	17	33

Results were non-significant. Median and ulnar nerve function was examined in 15 hands exposed to vibrations. Conduction times outside the normal range in the ulnar nerve indicate a neuropathic condition not explained by CTS. 4 out of 9 vibration-exposed hands that were improved by surgical intervention had normal ulnar nerve conduction times. For vibration-exposed hands not improved by surgical intervention, 4 of 6 had normal conduction times suggesting that ulnar nerve damage is not correlated with a poor result of carpal tunnel decompression.

Thus, most patients with both CTS and a history of occupational exposure to handheld vibrating tools had a good outcome after carpal tunnel decompression. This result supports others,[5] and suggests that patients with CTS should receive surgical decompression irrespective of exposure to vibrating machinery.

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Terminal dehydration and intravenous fluids

SIR,—Intravenous fluid infusion (IFI) can be given to terminally ill cancer patients. The reasons for and against such care have been recorded,[1-3] but very little is known about these patients' state of hydration or electrolyte balance.[4] We have therefore reviewed our experience with two groups of terminally ill cancer patients—those who received IFI and those who did not—48 h or less before death.

The study population was 68 consecutive patients admitted for palliative care. Patients were assessed for their level of consciousness according to a local alertness scale (LAS; 1 = fully conscious, 2 = responsive to visual/vocal stimuli, 3 = responsive to painful stimuli, 4 = fully unconscious). Blood count, routine biochemical tests, and urinanalysis were done and indices were compared between the groups and correlated with level of consciousness. Only 13 patients were treated by IFI, usually after family members' requests, or as a continuation of such treatment while on other wards. We regarded serum urea concentrations of 12 mmol/l or more as indicating substantial dehydration, and 59 patients met this criterion (mean serum urea 23·6 mmol/l); the other 9 patients had a mean serum urea of 8·8 mmol/l. Urea concentrations did not differ between patients receiving or not receiving IFI (two-tailed *t*-test; 50·9 *vs* 54·5 mmol/l). However, serum sodium differed significantly between the groups ($p < 0·02$, 139 [7·3] *vs* 148·5 [10] mmol/l). A significant correlation was shown between state of consciousness and serum sodium concentration (Pearson correlation $p < 0·01$), but not for potassium concentrations, which were higher than 5·5 mmol/l in 31 of 68 patients (mean 7·25 [1·67] mmol/l). No other significant associations were found. Moreover, level of consciousness did not correlate with the the use or non-use of IFI (Pearson chi-squared test).

Since most of these patients had severe hydration and electrolyte imbalance, including those receiving IFI, decisions about treatment with IFI should be based on the preference of the patient and his family.

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Monitoring cerebral glucose in diabetics by proton MRS

SIR,—Developments in localised proton magnetic resonance spectroscopy (MRS) have considerably enlarged the number of metabolites that can be studied in the human brain in vivo.[1,2] We have applied this technique to monitor brain tissue glucose in four patients aged 69–79 with raised plasma glucose concentrations due to insulin-dependent diabetes mellitus. Normoglycaemic volunteers, including age-matched controls, showed no detectable glucose resonances in spectra of grey or white matter. However, cerebral glucose was observed in the grey-matter spectra of all four diabetic patients but not seen in white-matter. The brain/blood glucose ratio varied among the patients, perhaps because of differences in severity and duration of disease. Direct monitoring of cerebral glucose may help to improve treatment.

Image-controlled MRS studies were done at 2·0 T on a Siemens whole-body 'Magnetom'. Localisation in parietal periventricular

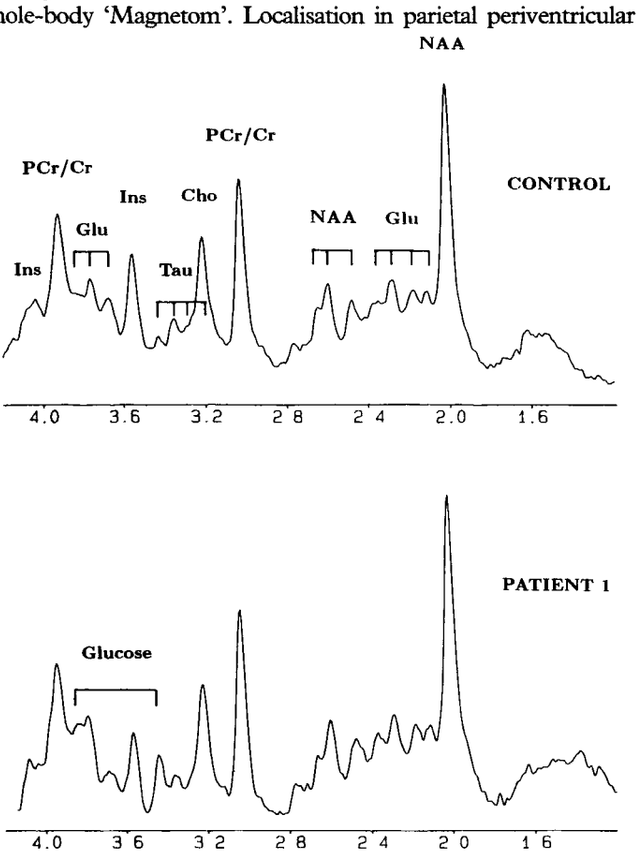

Fig 1—Short echo-time localised proton MRS of grey-matter.

Upper: normoglycaemic 60-year-old female control. Lower: 69-year-old female diabetic patient (patient 1) with plasma glucose of 9 mmol/l. Volume-of-interest is 18 ml. Metabolites are: N-acetylaspartate (NAA), glutamate (Glu), creatine (Cr) and phosphocreatine (PCr), choline-containing compounds (Cho), taurine (Tau), and *myo*-inositol (Ins).[1,2]

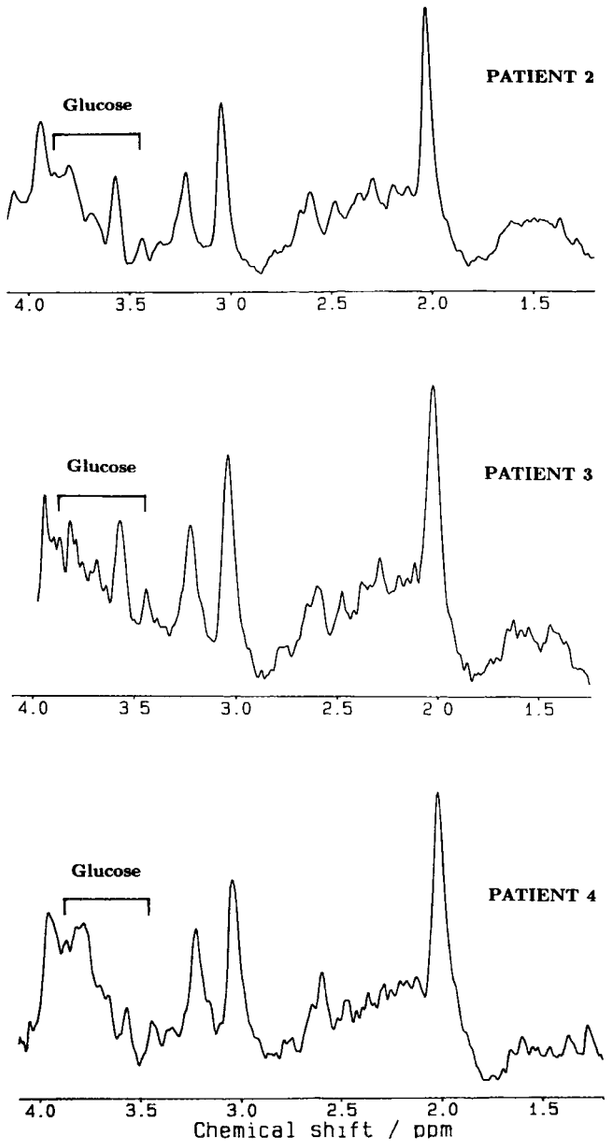

Fig 2—Localised proton MRS of parietal grey-matter in three other diabetic patients.

72-year-old woman (plasma glucose 9·4 mmol/l) (patient 2); 72-year-old man (12·2 mmol/l) (patient 3); and 79-year-old man (7·2 mmol/l) (patient 4). Major peaks at 3·0 and 2·0 rpm are PCr/Cr and NAA, respectively.

white-matter and paramedian grey-matter was achieved with a short echo time (T_E 20 ms) and 8–18 ml volumes of interest.[1] After a fast-scan magnetic resonance imaging protocol proton MRS was done within measuring times of 6 min (T_R 3000 ms, 128 accumulations).

Fig 1 shows proton MR spectra of parietal grey/matter of a normoglycaemic 60-year-old woman with coronary heart disease (control) and a 69-year-old woman with diabetes. The control MRS exhibits no abnormalities, the spectrum being similar to that seen in healthy volunteers. The absence of glucose resonances in normoglycaemia indicates that the cerebral glucose concentration in man is 1 mmol/l or less (relative to a 10 mmol/l concentration of NAA). This value is well below the 2–3 mmol/l determined for the anaesthetised rat by biochemical analysis[3] and proton MRS (unpublished).

The grey-matter proton MR spectrum of the patient in fig 1 exhibits clear glucose resonances at 3·43 and 3·80 ppm. White-matter spectra in the patients with diabetes were very similar to those of normoglycaemic controls and there was no unambiguous detection of glucose resonances, in line with experimental evidence of lack of accumulation of glucose by glial cells.[4]

The spectra in fig 2 are three further examples of raised brain tissue glucose in patients with increased blood sugar due to diabetes.

Until now there has been no direct non-invasive method for measuring the concentration of brain glucose in conscious man. Localised proton MRS offers this possibility. With an internal standard (10 mmol/l NAA) the brain glucose concentration varied between 3 and 6 mmol/l in these four diabetic patients. Brain/blood glucose ratios varied, being about 0·3 in patients 2 and 3 and 0·6 in patients 1 and 4. An alteration of the affinity for blood glucose and a concomitant decrease in intracellular brain glucose may explain why in chronic diabetes warning symptoms of hypoglycaemia are seen while plasma values still indicate normoglycaemia.

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"Open eyes" during examination of the motor system

SIR,—A patient was admitted to hospital with acute onset of muscular weakness in the left arm and leg. Examination by the resident physician revealed hemiparesis and paraesthesia on the patient's left side. Repeat clinical examination, under the supervision of the staff physician, showed that the motor deficit was completely corrected when the patient directed his vision towards the apparently paretic limbs. When the left arm and left leg were lifted and dropped the fall of the limb did not follow a vertical path; the left arm appeared to make pseudoathetoid movements. Similarly, examination of voluntary movement and gait were normal when completed under direct vision by the patient. Both superficial and deep sensation were impaired in the arm and leg. Computed tomography showed a small infarct in the right thalamus.

Patients are often asked to close their eyes during tests of motor function. For instance, in the Barré manoeuvre the patient is requested to hold his arms horizontally with eyes closed. In addition, ataxia may be partly compensated by visual aid. Thus, examination of the motor system with a patient's eyes closed may lead to a mistaken diagnosis for cases in which the true cause of the "muscular weakness" is ataxia. The correct diagnosis would be a disturbance in coordination. For patients in whom a true motor deficit and sensory loss coexist, failure to take account of this confounding variable may lead the physician to assume that the motor deficit is worse than it really is.

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Ticlopidine

SIR,—Your Feb 23 editorial ends with the statement that "Ticlopidine has now been approved by the Food and Drug Administration for prevention of strokes in patients intolerant of aspirin". On Dec 17, 1990, an FDA advisory committee recommended that the FDA grant Syntex permission to market ticlopidine in the United States to reduce the risk of strokes in patients who cannot take aspirin. However, the FDA has not yet approved ticlopidine for marketing.

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