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We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters should be typed with double spacing between lines and must be signed personally by all their authors, who should include their degrees. Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included.

Poor management of unconscious rugby player watched by millions

SIR,—Early in the second half of the televised Scotland versus France rugby international in March there was an incident which should have been an example to all of how to manage an unconscious patient, but this valuable teaching opportunity was lost to the millions of television viewers. A long ball was thrown at the line out, and in an attempt to gather the ball the French scrum half, Jerome Gallion, was accidentally knocked out by the Scottish flanker, David Leslie. He fell to the ground unconscious, where he lay on his back until play broke down.

He was attended first by the referee, who left him lying on his back while he called on the French trainer. He arrived on the scene and again left him on his back with no apparent attempt to clear or maintain an airway. By this stage it was apparent that it would be impossible for him to play on and a medical officer was called on to the pitch. He again left him on his back and summoned the stretcher bearers. Two first aid men hurried on to the pitch carrying an outdated stretcher, consisting of a canvas and two poles without even spacer bars. Gallion was bundled on to the stretcher with no apparent concern for any cervical spine injury and carried off, still unconscious and still on his back, to the sympathetic applause of the crowd. Fortunately he made an uncomplicated recovery, but the outcome might have been tragically different.

Being a rugby playing doctor I am concerned with both immediate and delayed treatment of sports injuries, and I think that severely head injured patients (as any unconscious patient must be) are at present badly managed. This particular incident is a prime example.

The priority is the airway and its maintenance, and the patient should be put in the coma position and his mouth cleared of any obstruction—that is, the tongue or gum shield. After recent publicity about cervical spine injuries in sport (7 January, p 37) people may be reluctant to move an unconscious patient. In comparison to head injuries, however, serious injuries to the cervical spine are rare. In the unlikely event of the two injuries coexisting further potential damage to the cervical spine can be avoided by ensuring that both the head and trunk are turned as one (log rolling).

While I do not recommend that all injuries on the pitch be handled by the referee, I do think that they should be given instructions in first aid, especially on the care of the unconscious patient. This would be particularly relevant in club rugby, where the referee may be the only one trained in first aid. As an extension of this there may be a case for the referee preventing a player from continuing after he has recovered from unconsciousness by sending him off—much as any boxer would expect. Rugby is an

amateur game, and the prospect of a dazed player bravely soldiering on is a sight that most doctors would rather not witness.

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Hyperbaric oxygen for patients with multiple sclerosis

SIR,—Dr J Mertin and Professor W I McDonald (31 March, p 957), while not being too sceptical, are right to question the evidence for the effectiveness of hyperbaric oxygen in multiple sclerosis. Their review is timely as the treatment is currently under worldwide evaluation.

Those of us who have witnessed the improvement in some patients for ourselves have no doubt that the effect is real and not simply due to the unpredictable course of the disease—it is too dramatic. In one severely afflicted patient, for example, diplopia resolved, his speech became intelligible, and he was able to lift his head off the pillow for the first time in years. Whether the response is prolonged or indeed of sufficient magnitude to justify the routine treatment of all patients will be known only when the results of controlled trials become available in a few years.

It is unfortunate that many of the centres taking part have access only to monoplace chambers and are using pure oxygen at 2 atm while following Fischer's protocol, which he described when using a multiplace chamber.¹ As Dr Mertin and Professor McDonald point out, leaking face masks can lead to the delivered oxygen pressure falling to 60% so that some of his patients may have received only an equivalent of 1.2 atm of oxygen. His results showed that better clinical improvement occurred in patients having arterial oxygen pressures equalling those in a monoplace chamber at 1.4-1.6 atm. It is therefore understandable why several centres using monoplace equipment are experiencing no appreciable effect or even deterioration.

From his experience of treating over 700 patients, Neubauer advocates the use of variable low pressures starting at 1.5 atm and titrating each patient according to his response.² He appreciates that research workers will find it difficult to use a variable regimen in controlled studies and suggests that better results might be attained with a steady pressure of 1.5 atm throughout when using a monoplace chamber (R A Neubauer. An unpublished open letter to all baromedical physicians, 1983.)

Before a final verdict on the treatment is reached it is essential that trials are properly designed so that groups of patients are evaluated who have received comparable treatment regimens. In the multicentre trial being directed by the US Multiple Sclerosis Society the choice of treatment regimen is left to the individual therapist when using a monoplace chamber. If sufficient clinical material becomes available a computer analysis will be able to separate patients into such groups.

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¹ Fischer BH, Marks M, Reich T. Hyperbaric-oxygen treatment of multiple sclerosis. A randomised, placebo-controlled, double-blind study. *N Engl J Med* 1983;308:181-6.

² Neubauer RA. Hyperbaric oxygen therapy of multiple sclerosis. A multicentre survey. Ocean Medical Centre, 4001 Ocean Drive, Lauderdale-by-the-Sea, Florida 33308, 1983.

SIR,—I have treated 32 patients with multiple sclerosis at the hyperbaric oxygen unit in this hospital—16 for three hours at 1.5 atm, and 16 for three hours at 2 atm. All patients had chronic disease, were severely handicapped, and had shown no remission over the preceding 12 months. They were treated for four consecutive weeks from Monday until Friday using the Vickers single patient hyperbaric oxygen bed. All were assessed neurologically by myself before treatment and again after treatment and three months later.

No patient showed any objective neurological improvement. Two patients, one from each group, thought bladder function was marginally improved.

We have now ceased to offer this treatment to patients because I do not believe it is effective. It is regrettable that the Association for Research into Multiple Sclerosis is collecting money in order to establish hyperbaric oxygen chambers which will be used by non-medically qualified persons. Hyperbaric oxygen is not without risk, and the action of the Association for Research into Multiple Sclerosis in going ahead without any good

clinical evidence for the efficacy of the treatment is in my opinion unwise.

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Effect of aspirin in "aspirin sensitive" patients

SIR,—I was interested in this paper by Dr S I Asad and others (10 March, p 745). Clinicians must now consider whether or not to attempt desensitisation in their patients who develop urticaria or asthma in response to aspirin and therefore need to know more about the background of the patients described.

There are three main clinical circumstances in which aspirin will cause attacks of urticarial weals. Firstly, patients with chronic urticaria will have exacerbations of the rash. This was first pointed out by Dr C D Calnan, and I wrote the first paper on the subject in 1960.¹ The conclusion then, before any link with prostaglandin metabolism could have been considered, was that the phenomenon had a non-immunological basis. It is now known that 30% to 40% of patients with chronic urticaria will have exacerbations after doses of aspirin—often quite severe and distressing. Secondly, patients who have very minor chronic urticaria with occasional weals may give a history of exacerbations after aspirin containing drugs. Thirdly, isolated attacks occur often at long intervals when the subjects accidentally take aspirin.

The patients described by Dr Asad and his colleagues may have fallen chiefly into the third category, but clinicians need further information about this point. To attempt desensitisation in the first category might well cause severe exacerbations followed by a period when the condition is worse. I would advise the greatest caution in any attempt to desensitise patients who react to aspirin and have active chronic urticaria.

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¹ Warin RP. The effect of aspirin in chronic urticaria. *Br J Derm* 1960;72:350.

Dicyclomine in babies

SIR,—Dr Jeff Williams and Dr Rob Watkins-Jones report side effects of dicyclomine in small babies (24 March, p 901). I have recently reported three similar cases to the Committee on Safety of Medicines.

The first baby, aged 7 weeks, was admitted with a history of being drowsy and irritable, and our working diagnosis was meningitis. But the lumbar puncture was clear, all other investigations were negative, and his condition quickly improved. It subsequently emerged that he had been given twice the usual dose of dicyclomine syrup because of confusion between the diluted and undiluted preparation.

The second baby, aged 10 weeks, was admitted with a history of gasping respirations, restlessness, and animation; and the general practitioner thought she had stridor. By the time she reached the ward she was well, and all investigations proved negative. It subsequently emerged that she had been given 5 ml of undiluted dicyclomine syrup.

The third baby was admitted aged 8 weeks with a history of shallow respirations and either an apnoeic attack or a convulsion. On admission he

was pale but no other abnormality was noted. Routine investigations were negative, and he rapidly improved. Again it emerged that he had been given 5 ml of undiluted dicyclomine syrup. He also made an uneventful recovery.

The problem is that 5 ml of dicyclomine syrup (Merbentyl) contains 10 mg of dicyclomine hydrochloride, which is a dose double that advised for a child of up to 6 months. These three children were all under that age, and confusion has arisen because of the concentration of the drug and the instruction to pharmacists to dispense in 5 ml doses.

I rarely use dicyclomine as I find it quite ineffective for so called infantile colic, but other doctors seem to find it effective. The Committee on Safety of Medicines tells me that 13 reports have been received of various incidents related to this drug, usually in small babies. I am sure that there are many more cases that have not been reported.

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SIR,—We wish to draw attention to a problem we recently encountered in a previously healthy five week old boy who received Ovool (dicyclomine and dimethicone). It is commonly prescribed for dubious indications. Our infant had a respiratory arrest 15 minutes after receiving the recommended dose and was resuscitated. All investigations performed were negative, and he appears to have made a good recovery, although his parents remain extremely anxious. The manufacturers are now including advice in their data sheet. This has not been reported with Ovool in the past.

We strongly endorse the recommendation that the indiscriminate use of this group of drugs for all manner of symptoms in babies should be discouraged, especially in the first two months of life.

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Motorcyclists should be encouraged to wear full face crash helmets

SIR,—Mr P G Richards (10 March, p 758) reported four cases of severe head injury in motorcyclists following crash helmet detachment during their accidents. This phenomenon was also noted in two previous surveys.^{1,2} But the type of crash helmet is also important: the full face helmet provides much more protection than the traditional "standard" and "Jet" (open face) designs.

Our survey (unpublished) over 18 months in one casualty department showed that after road traffic accidents 13 of 38 motorcyclists wearing the open face crash helmet sustained head or neck injuries compared with only 11 of 156 wearing the full face helmet ($p < 0.001$). Ten of the 14 facial injuries also occurred in the open face helmet group ($p < 0.001$). Only one helmet, a full face design, became detached during an accident. Motorcyclists who wore open face helmets, therefore, had 10 times the risk of facial injury and five times the risk of head or neck injury.

The increased protection for the face, head,