pleural mesotheliomas, and we think that this possibility merits further study. Indeed, Seal reported a case of mesothelioma in which the only exposure was that the patient had built an asbestos garage many years previously.[2]

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[1] Law MR, Gregor A, Hodson ME, Bloom HJG, Turner-Warwick M. Malignant mesothelioma of the pleura: a study of 52 treated and 65 untreated patients. *Thorax* 1984;**39**:255-9.
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Hyperbaric oxygen for multiple sclerosis

SIR,—While dampening enthusiasm for treatments of multiple sclerosis that are not substantiated is appropriate, critics should avoid making equally emotive and inaccurate statements. Dr J Mertin and Professor W I McDonald are incorrect in stating that hyperbaric oxygen has failed to gain general acceptance for carbon monoxide poisoning and are much too imprecise regarding osteomyelitis and gangrene (31 March, p 957). The reference cited is merely reiteration of out of date knowledge.[1] Those wishing to gain a balanced view of current thinking on the role of hyperbaric oxygen in clinical practice should consult the 1983 Undersea Medical Society committee report.[2]

It is not correct to state that after hyperbaric oxygen "decompression has to be carried out slowly to prevent central nervous system damage." This is true for air or oxyhelium under pressure but not for oxygen. There are other reasons for slow decompression in some patients—for instance, chronic lung disease.

The dangers of hyperbaric oxygen have been exaggerated in order to make their point. While Dr Mertin and Professor McDonald are entirely correct about the need for specially trained staff in properly equipped centres, the incidence of complications is low.[3] In our experience of about 1500 treatments with hyperbaric oxygen over more than a decade we have had only four oxygen convulsions—three of these occurred in the first three months of operation. No patients have died, and other side effects such as otic barotrauma have been only of minor concern.

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[1] McAlpine D, Lumsden CE, Aicheson ED. *Multiple sclerosis—a reappraisal*. Edinburgh: Churchill Livingstone, 1972.
[2] Committee on Hyperbaric Oxygen Therapy, Undersea Medical Society. *Hyperbaric oxygen therapy*. Bethesda: UMS, 1983.
[3] Slack WK, Hansen GC, Chew HER, Cockerill G, O'Connor R. Analysis of complications of hyperbaric oxygen therapy in 455 patients treated in single person hyperbaric oxygen chambers. In: Wada J, Iwa T, eds. *Proceedings of the fourth international congress on hyperbaric medicine*. Tokyo: Igaku Shoin Limited, 1970:505-9.

SIR,—Dr Philip James mentions a patient (16 June, p 1831) previously referred to by Dr J Mertin and Professor W I McDonald (31 March, p 957), who had an episode of unconsciousness in a hyperbaric oxygen chamber. Dr James prefers to think that the patient choked on chewing gum. I looked after the patient, and the details are as follows.

The subject, aged 44, had suffered from multiple sclerosis for about 10 years and had begun a course of hyperbaric oxygen six weeks previously. At the time of his blackout he was receiving once weekly "top ups." When he was exposed to a pressure of 2·5 atm for 30 minutes witnesses reported sudden generalised shaking with incontinence of urine. Decompression was started at the usual rates, and he was immediately transferred to the Ipswich Hospital accident and emergency department. On arrival he was noted to be confused, drowsy, and restless, and he was complaining of headache and unable to recall events over the past 12 hours. He recovered rapidly and recalled that he might have choked on gum. An electroencephalogram showed a sharp wave disturbance in the left temporal zone compatible with epilepsy. Fifteen years previously he had blacked out, possibly because of a seizure, while walking downstairs carrying a baby. It was thought that he had suffered a primary, generalised seizure and that the story of choking on gum was irrelevant.

It is well recognised that seizures may develop in healthy subjects on exposure to oxygen under pressure. According to Hollin *et al* the major manifestation of central nervous system oxygen toxicity is a seizure; after multiple hyperbaric exposure at 2-3 atm up to 3% of patients will convulse, sometimes without premonitory symptoms.[1]

Epilepsy is a well recognised complication of multiple sclerosis and is the most reasonable explanation. The patient possibly preferred the choking story for fear of adverse publicity.

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[1] Hollin SA, Levine ME, Sukoff MH, Jacobson JH. Neurological complications of hyperbaric oxygen therapy. In: Silverstein A, ed. *Neurological complications of therapy*. London: Futura, 1982:434.

SIR,—We are writing to endorse and emphasise points made by Dr J Mertin and Professor W I McDonald (31 March, p 957). The difficulty of evaluating therapeutic trials in multiple sclerosis has led concerned organisations to propose guidelines for controlled clinical trials.[1-3] Several published reports claiming that use of hyperbaric oxygen benefits patients with multiple sclerosis stimulated the controlled trial sponsored by the National Multiple Sclerosis Society (USA).[4] These investigators conclude: "Because of the small sample and the short follow up period in our study, the results must be viewed with caution and regarded as preliminary. They also await confirmation by other independent research centres."[1] A number of hyperbaric facilities have, however, proceeded with "treatment" of multiple sclerosis with hyperbaric oxygen without providing for controlled clinical trials.

With a consortium of six university related neurology departments and hyperbaric medicine facilities we proposed a controlled multicentre trial of hyperbaric oxygen for patients with multiple sclerosis in the USA. We fully agree with Dr Mertin and Professor McDonald that further clinical trials should be performed to evaluate the efficacy of hyperbaric oxygen for multiple sclerosis in a large group of patients. Hyperbaric oxygen should not currently be considered an established treatment. We also agree that hyperbaric treatments should be given *only* under the supervision of qualified hyperbaric physicians. We are concerned to learn that many treatment units are being established in Britain for the treatment of multiple sclerosis with hyperbaric oxygen by non-medical personnel. We mean no disrespect to the professionalism of trained hyperbaric technicians, but patients given hyperbaric treatment warrant full medical supervision.

Thus we agree with the major conclusions of Dr Mertin and Professor McDonald but we disagree with their undue emphasis on the risks of hyperbaric treatments. The risks of oxygen toxicity in a brief exposure are minimal, and the rate of compression with hyperbaric oxygen treatment can be rapid, limited primarily by middle ear pressure equilibration. These comments are based on data assembled during the past 10 years by the hyperbaric registry of the Undersea Medical Society, the international professional organisation for trained hyperbaric physicians.[3] During the past four years 16 000 people have received hyperbaric oxygen treatments in the USA for a range of conditions with minimal complications.[3] Thus we do not agree that hyperbaric oxygen poses "The possibilities of serious side effects and of a lower threshold for oxygen toxicity. . . ."

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[1] Fischer BH, Marks M, Reich T. Hyperbaric-oxygen treatment of multiple sclerosis. A randomized, placebo-controlled, double-blind study. *N Engl J Med* 1983;**308**:181-6.
[2] Gamache FW, Myers RAM, Ducker TB, Cowley RA. The clinical application of hyperbaric oxygen therapy in spinal cord injury. A preliminary report. *Surg Neurol* 1981;**15**:85-7.
[3] Myers RAM, Schnitzer B. Hyperbaric oxygen usage, update 1983. *Postgrad Med J* 1984 (in press).
[4] Schnitzer B, Myers RAM, Britten G, *et al*. *Hyperbaric chambers, USA and Canada*. (Undersea Medical Society Corporation.) Hollywood, Maryland: McGregor and Werner, 1983.

**This correspondence is now closed.—ED, *BMJ*.

Dilate the pupil and see the fundus

SIR,—Professor C I Phillips (16 June, p 1779) refers to tropicamide and phenylephrine as drugs with similar time courses. In support he misquoted our work which in fact shows phenylephrine to be a much slower mydriatic: 20 minutes after a single eyedrop, phenylephrine 3% had reached only 10% of its maximal effect compared with tropicamide 0·5% reaching 93% of the maximum.[1] The exact time to maximum has been measured in healthy subjects as 39 minutes for tropicamide 0·5% and 70 minutes for 10% phenylephrine.[2]

Professor Phillips did not consider which mydriatic to choose for patients with diabetic retinopathy, who are notoriously resistant to conventional regimens. We have shown that the eyes of these patients, who are especially sensitive to topical phenylephrine because of pupillary sympathetic neuropathy,[3] dilate excellently to a combination of tropicamide and phenylephrine.

Finally, although systemic absorption of topical phenylephrine (an α agonist) may result in a pressor response which could be exaggerated in patients taking tricyclic antidepressants[4] it does not affect myocardial