improved and nine remained unchanged. This result is significant (p < 0.03), the same level of significance found by Fischer et al and by Barnes et al in their preliminary report.

It is inappropriate, in view of the pathology found in multiple sclerosis, to expect severely affected patients to experience dramatic benefit and Schumacher has pointed out that the object of any therapy is to stabilise the disease. The chronic nature of the blood-brain barrier changes in multiple sclerosis strongly argues for the continuation of oxygen therapy, but it is difficult to test this under double-blind conditions. Nevertheless a long-term study of hyperbaric oxygen with continuation therapy has now demonstrated benefit.

Barnes et al emphasise the level of barotrauma experienced in their study, but this appears to have been provoked by pressurisation over a fixed period, as part of their double-blind protocol. To generate 21 cases of barotrauma, including the perforation of an ear drum, when it is possible to avoid this problem by using a slower pressurisation is regrettable. There are now 56 centres in the United Kingdom using hyperbaric oxygen in the management of multiple sclerosis, attended by several thousand patients. Over 50,000 individual sessions have been undertaken with no significant barotrauma.

The reduction of the amplitude of the visual evoked responses in some patients, found by Barnes et al, is attributed to them to changes in the retina. The emotive term damage is used to describe the effects, which is surely inappropriate when they were found to be reversible. Nichols et al actually caused complete loss of vision in one eye of a patient with a history of retrobulbar neuritis by a six hour exposure to oxygen at 2 ATA. The patient was taking part in experiments on pulmonary oxygen toxicity. The loss of vision was completely reversed over a period of 14 hours.

As Fischer et al argued, it is necessary to reiterate that the demonstration of small benefits to severely disabled patients with advanced sclerosis, indicates that studies of oxygen therapy should be undertaken when the first symptoms typical of multiple sclerosis develop. At this time the majority of patients have multiple areas affected and there is the prospect of minimising the development of the irreversible sclerosis. The use of hyperbaric oxygen therapy in the presence of focal oedema and blood-brain barrier dysfunction is based on fundamental scientific principles and supported by experimental and clinical data. Despite the benefits in chronic patients being modest, they can make important contributions to the quality of life and can generally be sustained by continuing hyperbaric oxygen therapy. In this area a neurologist (Davidson DLW, personal communication) has adopted the sensible policy of advising patients of the mild benefits from therapy and its availability in the self-help setting provided by the charity Action for Research into Multiple Sclerosis (ARMS).

Barnes et al must be congratulated for being prepared to revise their negative stance on the efficacy of oxygen supplementation in chronic multiple sclerosis, but it remains to be seen if they can reverse the damage caused by the premature conclusions of their preliminary report.

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References

Matters arising


Barnes et al replies:

We must point out to Dr James that we have not fundamentally changed our view about hyperbaric oxygen. The conclusion of our first paper was that "hyperbaric oxygen is unlikely to have a role in the management of a patient with multiple sclerosis". The conclusion after our final results was that we felt it "unlikely that hyperbaric oxygen would play any practical role in the management of a patient with chronic multiple sclerosis". We have not denied the subjective benefit in urinary symptoms found in some patients nor do we deny that there appeared to be a slowing of cerebellar deterioration in the treated patients. We must emphasise that the improvement of bladder symptoms was mild and of a degree that could be obtained by the prescription of an anticholinergic drug. Whether the lack of deterioration in cerebellar function is a real or statistical phenomenon must wait the results of further studies. Our study does not indicate a sub-group of patients that benefitted from oxygen therapy and in practical terms we do not feel that an unpredictable and mild benefit could justify the widespread use of hyperbaric oxygen.

We must also point out to Dr James that pressurisation was not conducted over a fixed period but was dependent on patients' ability to equilibrate pressure in the middle ear. In most cases barotrauma was of grade one severity. For those who are not familiar with diving medicine terminology this simply means discomfort in the ear accompanied by injection of the tympanic membrane. It is not usually of clinical significance and our percentage of grade one barotrauma is entirely compatible with experience in diving medical practice.

We feel that retinal damage is an appropriate term to describe the effects that we found on visually evoked potential testing. Our findings were reversible and not accompanied by changes of clinical significance. We simply wish to emphasise the potential hazards of long term oxygen therapy on the vulnerable retinal circulation.
Matters arising

We hope that our papers have adopted a balanced rather than a negative view of the practical role of hyperbaric oxygen for patients with chronic multiple sclerosis.

References


Neubauer writes:

Sir: I must disagree with the final conclusions of Dr Barnes et al1 in regard to the effectiveness of hyperbaric oxygen in multiple sclerosis. My points of contention are: (1) Side effects: their series represented some of the highest incidence of side effects that have ever been reported in the hyperbaric literature. None of these occurred in their control series because the patients were not pressurised equivalently with air. They have drawn conclusions from their own problems that hyperbaric oxygen is fraught with side effects. Little do they realise that the majority of all hyperbaric oxygen pressurisations throughout the world are given in a lay setting on oil rigs with no physician in the chain. In a well run hyperbaric centre, even the slightest side effects of barotrauma are seen only in 1–2% of the cases. The extensive ARMS series in the United Kingdom reports only minimal discomfort. (2) The expense of the treatment: the ARMS charity institution in the United Kingdom again attests to both safety and cost effectiveness of this treatment. There are currently 56 ARMS centres where several thousand patients are undergoing treatment. These treatments are performed by trained lay persons. If the patient cannot afford the treatment, it is not withheld. It is my understanding that the treatment now runs about £6 (approx. $10.50), per treatment; this being the lowest fee for HBO in the world. (3) Lack of effect: in spite of possibly preconceived ideas, their data do show significance in regard to the urinary tract improvement. Such data have been previously documented.2–4 To a multiple sclerosis patient this is of extreme importance. These authors may have had significantly different results if only they had followed the original clinical protocol which stipulated individual pressurisation (dose) and continued treatment with HBO.

Hyperbaric oxygen to the multiple sclerosis patient is analogous to insulin in the diabetic because of the dependence of the level of vasoconstriction on the inspired partial pressure of oxygen. How one would expect 20 treatments of any modality to permanently affect the continuing lesions is not reasonable. In my original publication, it was stressed that no patient had ever been cured, but hyperbaric oxygen does alter the course of multiple sclerosis. It must be used at the proper time and at the proper dose and continued treatments are mandatory. It is unfortunate that Dr Barnes et al used inappropriate pressure, had multiple side effects, and neglected their own data.2 Obvi-ously they are proficient neurologists, but they are not involved in the practice of hyperbaric oxygen therapy.

Data continue to unfold confirming my original reports. Previous substantiated effects on the bladder, Barnes et al’s lack of cerebellar deterioration and the long term positive double-blind studies by Pirovano et al6 certainly belie their negative conclusions.

RICHARD A NEUBAUER, MD
President
American College of Hyperbaric Medicine

References


Barnes et al reply:

Dr Neubauer makes very similar points and the only new point in his letter that we feel needs response is his suggestion that we should have followed his original clinical protocol which stipulated individual pressurisation and continued treatment with HBO. We must point out that a variable dosage is simply not possible in a double blind clinical trial setting and in any case patients did not report any response, objective or subjective, until at least fourteen days of treatment. This obviously makes individual pressurisation according to the patients’ response quite impossible. We cannot deny that further benefits may have become apparent after continued therapy. We must point out again that there has been no claim for later improvement with continued therapy but only continuation of improvement that was induced by the original course of oxygen. If there is no original improvement then it seems unlikely that there will be later improvement.

Book reviews


The first edition of Michael Aminoff’s textbook of electromyography has now been expanded and in some sections rewritten to take account of the advances in the subject that have occurred over the last ten years. The resulting second edition, however, keeps faith with the author’s original aims to review the manner in which electromyography may be of value in the investigation of patients and to make clinicians more aware of the scope and limitations of the investigative procedures. Not surprisingly then, the strength of the book lies in its discussions of the clinical relevance or otherwise of the neurophysiological findings. For example, there is an excellent chapter on the investigation of root and plexus lesions—the ‘bête noire’ of neurophysiology. The pros and cons of needle examinations, motor and sensory nerve conduction studies, H-reflex and F-wave studies, somatosensory evoked potentials and dermatomal evoked poten-