

The Controversy over Hyperbaric Oxygenation Therapy for Multiple Sclerosis

Richard A. Neubauer, M.D.
Virginia Neubauer
Sheldon F. Gottlieb, Ph.D.

The emotionalism and political maneuvering surrounding claims and counterclaims for the use of hyperbaric oxygenation therapy (HBOT) in multiple sclerosis (MS) has been nearly unique in intensity. Though widely used throughout the world, the application of HBOT in central nervous system conditions in general remains a subject of great debate, even among physicians specializing in hyperbaric medicine.

A Serendipitous Observation

In the late 1970s, the senior author (R.A.N.) was treating a patient with osteomyelitis (a conventional use of HBOT) who had a concomitant diagnosis of MS. The osteomyelitis required repeated treatments, and it was noted that each time the patient received a series of HBOT, the MS symptoms abated. He also learned that Drs. Raphael Pallotta¹ in Italy and Jacques Baixe in France (personal communications) were seeing the same type of improvements in MS patients.

Because MS is often a relapsing/remitting disease of wild exacerbations and remissions, a few such observations could well be coincidental. Indeed, Boschetty and Chernoch,² in Czechoslovakia, had concluded that HBOT was of no value, despite small transient improvements in 26 patients treated in 1970, because symptoms promptly recurred. They did not attempt a trial of continuing follow-up treatments.

R.A.N. published his initial findings and continued treating MS patients, carefully following their progress.³ He used as a guide the statement of George Schumacher, M.D., former chairman of the International Federation of Multiple Sclerosis Societies, in response to a question about recommending a treatment for MS. Schumacher said that a therapy for multiple sclerosis can be considered meritorious if a large percentage of patients are no worse two years after the initiation of a therapeutic modality.⁴

Two years later, in 1980, having accrued a series of 262 patients, R.A.N. again published his results.⁵ The conclusions were:

1. HBOT was not a cure for MS.
2. Response was dose sensitive.
3. Long-term intermittent follow-up treatment was needed.
4. HBOT favorably altered the natural history of the disease.

The clinical impression was that patients who received the therapy and continued with occasional follow-up treatments did not progress as far or as rapidly as those who never received the therapy. As a clinician and practitioner, as opposed to a university/pharmaceutical researcher under grant, R.A.N. was not in a situation to perform formal double-blind, placebo-controlled studies.

Publication was delayed for months as the journal grappled with a wide spectrum of reviewers' opinions. According to the

accompanying editor's note, views ranged from "must be published" as it "may turn out to be most important paper ever published in our search for the understanding of multiple sclerosis" to "do not publish" because it was "very speculative" and had "no scientific basis."⁵

R.A.N. simply continued to treat his patients, and they just continued to stabilize and improve.

Formal Studies

As a result of these published case studies and the inevitable publicity, the National Multiple Sclerosis Society (NMSS) had to test this new therapeutic modality, even though little was known about the mechanism(s) whereby oxygen under pressure might produce beneficial effects. The use of oxygen under greater-than-atmospheric pressure certainly did not seem to fit with the long-standing microbial (viral or bacterial) and/or autoimmune concepts for the etiology of MS, which then and now are still accepted by the NMSS despite lack of proof.

In 1980 the NMSS awarded Dr. Boguslav Fischer, a professor of neurology at New York University, \$250,000 to reproduce or refute these findings—preferably, it has been strongly suggested, to do the latter. The subjects were well-matched (a very difficult achievement in MS) in a placebo-controlled, double-blind and excellently executed study. Fischer's results were highly positive and confirmed R.A.N.'s original observations⁶—apparently much to the sponsor's chagrin.

Fischer's valuable contribution did get headlines in the popular press ("HBOT Helps MS") but was not well received in the medical community. Nor was he permitted to carry out the follow-up studies that he suggested. Instead, the medical administration went out of its way to make his subsequent professional life difficult. He was fired, his chamber was dismantled as junk, and he was ordered never again to treat another MS patient with oxygen. Some surmise that he was forced to understate his results and conclusions. In Fischer's own words:

The NMSS has to be credited with funding the research project, although there was an undercurrent, though never publicly expressed at that time, of the inefficiency of this particular approach to treating MS. When we found HBO to be effective, they instituted a campaign to discredit our work. They even went so far as to attempt to prevent its publication.⁷

Fischer's study, however, had already attracted attention worldwide. As early as 1982, after he presented prepublication data at a meeting in Long Beach, California, a group of five patients from Dundee, Scotland, started a community-based treatment center under the direction of Dr. Philip James, later joined by Dr. David Perrins. The results of a longitudinal study of 30 patients, monitored by a local neurologist, were so encouraging that other charity centers began to open throughout the UK, and are still in existence.

After Fischer's data were published in the *New England Journal of Medicine*, 14 double-blind controlled studies were undertaken worldwide. Almost all were methodologically flawed.

Fischer had treated his patients in a multiplace chamber pressurized to 2 atmospheres absolute (ATA), with the oxygen delivery via mask. Owing to leakage/slippage of the mask, however, the effective pressure the patients received was actually equivalent to 1.2 – 1.8 ATA. Although he published the mean paO_2 measurements, he did not directly correlate this to the delivered pressure and stated that there was no clear-cut relation between the partial pressure and the clinical response. Thus, it was assumed that 2 ATA was the appropriate pressure.

R.A.N.'s original report concerned treatments in a monoplace chamber (where the pressure is exact) at pressures of 1.5 to 1.75 ATA. According to Holbach and Wassmann, pioneers in the use of HBOT in neurosurgical patients, pressures of 2.0 ATA and higher were not beneficial, and perhaps injurious, to the injured brain (and thus potentially in any CNS condition, including MS).⁹

Overlooking this point, and thinking that they were following Fischer's protocol, doctors treated many MS patients at 2 ATA for 90 minutes per treatment in monoplace chambers (exact pressure), rather than the effective pressure of closer to 1.5 ATA utilized by Fischer. These patients either did not improve or worsened—a result that may have been partly explained by the administration of an inappropriate dose of oxygen. Even healthy divers must follow protocols for the use of enriched-oxygen gas mixtures (Nitrox) because of oxygen toxicity at high doses.

Some studies used long-term patients whose Kurtzke Expanded Disability Status Scale (EDSS) was so advanced that a course of only 20 treatments of any drug or therapy was highly unlikely to elicit any change in their clinical status. (Such patients were and are generally excluded from almost all clinical studies because of the degree of irreparable damage already sustained.)

Despite flaws, some studies did confirm some of Fischer's findings. Even otherwise negative studies found a statistically significant improvement in bowel/bladder control in the treated subjects. This finding was dismissed as unimportant to the MS patient because drugs to control this problem were readily available. The conclusions were phrased in a negative way, and on publication of the first and best known of the 14 studies,¹⁰ headlines proclaimed "Study Shows that HBOT Doesn't Work for MS."

After formal protests in the letters to the editor of *Lancet*, Barnes and Bates reassessed their data, and concluded that their results were positive after all. They republished the data in this new light,¹¹ but the damage was done. The news that HBOT really worked was completely obscured by the headlines saying that it did not. Dr. Barnes went on to be neurologic consultant on rehabilitation to one of the ARMS (Action for Research in Multiple Sclerosis) charity centers in the UK treating MS with HBOT. His consulting neurologist was Dr. David Bates.

In 1984, R.A.N. began a study of the effects of HBOT on more objective measures, such as MRI (then called nuclear magnetic resonance or NMR), before treatment and after one and a series of 20 treatments. Improvements were seen in T2-weighted lesions; one or more lesions disappeared in 11 out of 35 patients, and 8 patients showed a diminution in signal intensity, interpreted as a favorable response. The remaining patients showed no change.¹² Improvements were also noted in visual evoked potentials, brainstem auditory evoked responses, and somatosensory evoked potentials.¹³

Although R.A.N. and S.F.G. gave a number of invited presentations of these results internationally, no opportunity was given to present and discuss them at comparable meetings in the United States. The Undersea and Hyperbaric Medical Society (UHMS) held tightly to its conviction that HBOT would not work for MS, just as the NMSS held to its belief that MS is an autoimmune or viral disease. After R.A.N.'s first presentation at a U.S. scientific/medical forum in Mobile, Alabama, at the invitation of Dr. Sheldon Gottlieb (S.F.G.), then president of the Gulf Coast Chapter of the UHMS, both were labeled as heretics for disputing widespread, long-ingrained ideologies of both the nature of MS and the lack of potential benefits of HBOT.

Theory Determines Therapy

Autoimmunity and "Disease-Modifying" Drugs

Since the original description of MS in 1869 by Charcot, a bacterial or viral etiology has been considered, although even open biopsies of active lesions have failed to isolate the organism. Immune-mediated destruction of myelin, whether in response to an infectious agent or other cause, is widely believed to be the pathogenetic mechanism.

Interferon gamma, a substance naturally occurring in the body, was tried because of its antiviral effect but proved to be harmful in MS. Interferon gamma promotes inflammation, an effect that is countered by interferon beta. Betaseron (interferon beta 1-b), and Avonex or Rebif (interferon beta 1-a) are now commonly used. Double-blind studies have shown these agents to decrease the frequency of exacerbations in relapsing/remitting MS, and to decrease the number of lesions visible on MRI. However, these drugs have not been shown to prevent ultimate progression of disability.

Another drug in common use is Copaxone (glatiramer acetate), an immune modulator. This also reduces relapses but without demonstrably affecting long-term disability.

The only other "disease-modifying" drug listed on the website of the NMSS (www.nationalmssociety.org) is the antineoplastic agent Novantrone (mitoxantrone). This drug is thought to act in MS by suppressing the T cells, B cells, and macrophages involved in attacking the myelin sheath. It is the first drug ever approved for the treatment of secondary progressive multiple sclerosis in the United States. The lifetime cumulative dose is limited because of possible cardiac toxicity.

Of note is the difficulty in performing a double-blind study with mitoxantrone, which turns the urine and potentially the sclera blue. Therefore, the adequacy of the blinding was questioned in an initial study that showed no effect of treatment on disability measures. A later study used an infusion of methylene blue in the placebo arm, as if it were an inert compound. Methylene blue, however, is known to be neurotoxic when administered intraventricularly or intrathecally, and up to 22 % of placebo patients had openings in their blood-brain barrier at the time the substance was administered, according to documents filed with the FDA by Immunex Corp. The statistical significance of lesser deterioration in the treatment group depended on the finding of greater deterioration in the methylene-blue group.¹⁴

The latest attempt by modern (pharmaceutical) medicine to cure MS was Tysabri (natalizumab), the first humanized monoclonal antibody ever approved by the FDA for relapsing MS. The drug was granted "fast-track accelerated" FDA approval after

one year of a two-year trial. Only three months later, in February 2005, the drug was withdrawn from worldwide use and all clinical trials when two MS patients developed progressive multifocal leukoencephalopathy (PML), a rare neurologic disorder. A review of the data then revealed another death from PML in a patient being treated with the same drug in a clinical trial for Crohn's disease. PML is caused by a virus carried by most adults, which does not cause harm until a weakened immune system allows it to become active.¹⁵⁻¹⁶ Ironically, the treatment managed to create an even more virulent form of the pathology caused by the disease itself! And unlike MS, PML is actually proven to be of viral etiology.

Other Possible Pathogenetic Mechanisms and HBOT

As treatments aimed at a primary viral or autoimmune etiology have not proved to be greatly successful, consideration of a different pathogenetic mechanism—with the observed immune phenomena as a secondary effect—would be reasonable. The same immunologic markers as in MS have been shown to occur at the same levels in stroke,¹⁷ which is clearly not a viral or autoimmune disease. Dr. Philip James of Dundee, Scotland, proposed decompression sickness as a model for MS, based on pathology¹⁸ and clinical course.¹⁹ James also suggested fat emboli as the counterpart of gaseous microemboli in everyday life and a possible etiology of a primary vascular lesion (endothelial damage) in MS.²⁰

If MS is the result of a “wound” to the CNS caused by circulatory impairment, then the efficacy of HBOT would not be surprising. Investigating alternate theories for the pathogenesis of MS is not, however, a research priority.

Long-Term Results

According to the NMSS Consensus Statement, a disease-modifying drug should be started as soon as possible following a definitive diagnosis and continued until the side effects are intolerable or a better treatment becomes available.²¹ While the NMSS believes that there may be a reduction in future disability with such drugs, there is currently no evidence that this occurs. In addition, the long-term health effects of these immunosuppressant drugs are not fully known, although some short-term side effects are serious and debilitating in many patients.

One published multicenter study, funded in part by the NMSS, purportedly intended to test our recommendations for long-term HBOT treatment.²² There was no control group, but Kindwall et al. concluded that the trial failed on the basis of their statement of the Schumacher criteria: “If 90% to 100% of such patients [pursuing a downhill course or with frequent exacerbations] failed to get worse, the efficacy of the treatment would be manifest.” No current treatment modalities could meet such a stringent criterion. However, what Schumacher actually said was that “there could be no question about the statistical significance of such a result.”²⁴ This was in the context of recommending a pilot study of patients with early but active disease, using subjects as “their own controls,” with the conclusion of benefit resting on absence of exacerbation or prevention of major additional neurologic dysfunction in the “overwhelming majority (90 to 100 percent)” over a two-year period. Less stellar results would not disprove a significant benefit.

Of 383 patients who were initially enrolled by Kindwall et al., upon referral by one of 170 neurologists, 312 started treatment, 237 finished the initial series of 20 HBOT sessions, and only 28 (9%)

completed two years of monthly boosters. Only 39 patients (10.9%) reported “complications” after the first 20 treatments. More than 90% consisted of simple ear discomfort. Occasional nausea was also reported. The investigators attributed the high drop-out rate to the “cumbersome” nature of the treatment and inability of patients to see any benefit.²²

The 62 ARMS centers in Britain, now known as the Federation of Multiple Sclerosis Treatment Centers, followed 703 patients in detail since first receiving treatment, and have 10-14 year follow-up data on 447 patients.²³ Such information is virtually impossible to obtain in grant-funded studies because of the high cost.

In contrast to the claims for drug effects—primarily a reduction in exacerbations at a high price in adverse effects, with the hope that this will result in less future disability—HBOT centers report symptomatic relief in the majority (see Table 1) and apparent stabilization or slowing of progression in a significant fraction (see Table 2). Most remarkably, in the UK alone, more than 1.5 million sessions of HBOT have been administered to more than 14,000 patients without significant incident.²³ Minor problems with pressure on the eardrums (mild barotrauma), which did not necessitate discontinuing treatment, are reported in about 17%,²⁴ and about 7% reported transient myopia.²³

While the Kindwall study was only able to retain 9% of 312 patients for 2 years—or 12% of those who completed the initial course—about two-thirds of 705 UK patients who completed an initial course continued intermittent treatment for at least 3 years. Based on the UK data, the results reported by Kindwall were not surprising because so few patients continued with their follow-up treatments. It appears that about 300 treatments in 10 years, or once a fortnight, are needed to retard progression in patients with relapsing/remitting disease, and weekly treatments are more effective.²⁵

Table 1. Patients' Assessment of the Symptomatic Effect of the Initial Course of HBOT

Symptom	Improved %	No Change %	Worse %
Fatigue	70	22	8
Speech	64	34	1
Balance	59	37	4
Bladder	68	30	0
Walking	77	19	4

Source: Federation of Multiple Sclerosis Treatment Centers^{23,24}

Table 2. Results of Regular HBOT for 10-14 Years

Status	Relapsing/Remitting (N=112)	Chronic Progressive (N=259)	Chronic Static (N=76)	Total (N=447)
Improved	14 (13%)	12 (5%)	4 (5%)	30 (7%)
Unchanged	23 (21%)	31 (12%)	19 (25%)	73 (16%)
No worse (improved + unchanged)	37 (33%)	43 (17%)	23 (30%)	103 (23%)
Worse	75 (67%)	216 (83%)	53 (70%)	344 (77%)
Mean No. of Treatments	338	257	266	

Source: Federation of Multiple Sclerosis Treatment Centers^{23,24}

Including all patients available for follow-up for at least 10 years, even those who had chronic progressive disease or relatively few treatments, 23% were no worse. Even more remarkably, 30 patients (7%) actually improved.²³

The rate of progression is inversely related to the frequency of HBOT. In the UK series, the five relapsing/remitting patients who had fewer than 10 follow-up treatments had deteriorated by 2.0 on the EDSS after 10+ years, while the 31 such patients who received more than 400 treatments had deteriorated by only 1.1 points ($P < 0.001$).²³ One subgroup of patients met the Schumacher criterion: none of the early relapsing/remitting patients with relatively limited damage (mean EDSS 2.3), who received at least eight treatments in every quarter, had deteriorated on the EDSS at the end of 6 years, and four patients actually improved by a mean 0.8 on the EDSS.²⁴

Conclusions

At this time, the main and only realistic objective in the treatment of established MS is not to effect a “cure” for existing lesions or scarring, but to limit further deterioration and progression of the disease. After 27 years of experience in treating MS patients with HBOT, our conclusions are the same as listed above: HBOT is not a cure, but there is evidence to suggest some symptomatic benefit in a majority of patients and apparent stabilization or slowing of progression in a significant fraction (17 to 33%) of those who receive continuing therapy over a period of 10 years or longer. HBOT in MS has few side effects, mostly minor.

With any chronic disease, long-term compliance with therapy, particularly if it does not produce instantly obvious results, is difficult to assure.

There appears to be a double standard in the United States, with highly favorable treatment accorded to pharmaceutical interventions—in research effort, insurance coverage, and attention in the medical and popular literature. Despite high cost, frequent distressing adverse effects, unknown long-term toxicity, and limited efficacy, chronic drug treatment is apparently the standard of care for MS. Long-term outcome of therapy with currently popular drugs is as yet unknown. At the same time, high-dose oxygen (HBOT) is largely unfamiliar to physicians, is often considered strange and controversial, and remains unavailable to all but a few.

The course of MS is highly variable, making the assessment of long-term therapy especially problematic. More longitudinal data are urgently needed. Additionally, research is needed on the value of longer initial courses of HBOT treatment; the determination of optimal dosage and treatment duration and frequency; and the comparative efficacy of HBOT, drugs, or combinations.

Richard A. Neubauer, M.D., is Medical Director of Ocean Hyperbaric Neurologic Center. Contact: ran@oceanhbo.com. **Virginia Neubauer** is Research Director, Ocean Hyperbaric Neurologic Center. **Sheldon F. Gottlieb, Ph.D.**, is a consultant to Ocean Hyperbaric Neurologic Center.

REFERENCES

- 1 Pallotta R, Aneschi F, Costagioni N, et al. Prospettive di terapia iberbarica nella sclerosi a placche. *Ann Med Navale* 1980;85:57-62.
- 2 Boschetti V, Cernoch J. Aplikace kysliku za pretlau u nekterych neurologickych onemocneni. (Predbezne sdeleni). *Bratisl Lek Listy* 1970;53:298-302.

- 3 Neubauer RA. Treatment of multiple sclerosis with monoplace hyperbaric oxygenation. *J Fla Med Assn* 1978;65:101-104.
- 4 Schumacher GA. Critique of experimental trials of therapy in multiple sclerosis. *Neurology* 1974;24:1010-1014.
- 5 Neubauer RA. Exposure of multiple sclerosis patients to hyperbaric oxygen at 1.5-2 ATA: a preliminary report. *J Fla Med Assn* 1980;67:498-504.
- 6 Fischer BH, Marks M, Reich T. Hyperbaric-oxygen treatment of multiple sclerosis: a randomized, placebo-controlled, double-blind study. *N Eng J Med* 1983;308:181-186.
- 7 Gottlieb SF. The deadly influence of ideology in the treatment of multiple sclerosis. In: *The Naked Mind*. Flagstaff, Ariz.: Best Publishing; 2003:539-594.
- 8 Gottlieb SF, Neubauer RA. Multiple sclerosis: its etiology, pathogenesis, and therapeutics with emphasis of the controversial use of HBO. *J Hyperbaric Med* 1988;5:142-164.
- 9 Holbach KH, Caroli H, Wassmann H. Cerebral energy metabolism in patients with brain lesions at normo- and hyperbaric pressures. *J Neurol* 1977;217:17-30.
- 10 Barnes MP, Bates D, Cartlidge NEF, French JM, Shaw DA. Hyperbaric oxygen and multiple sclerosis; short-term results of a placebo-controlled, double-blind trial. *Lancet* 1985;1(8424):297-300.
- 11 Barnes MP, Bates D, Cartlidge NEF, et al. Hyperbaric oxygen and multiple sclerosis: final results of a placebo-controlled, double-blind trial. *J Neurosurg Psychiatry* 1987;50:1402-1406.
- 12 Neubauer RA, Gottlieb SF, Kagan RL. Magnetic resonance imaging in multiple sclerosis following hyperbaric oxygen. First Swiss Symposium on Hyperbaric Medicine, Basel, Foundation for Hyperbaric Medicine, 1986.
- 13 Neubauer RA, Morariu MA. Evoked potentials in multiple sclerosis patients treated with hyperbaric oxygen: 26 cases. Ninth Annual Conference on Clinical Applications of Hyperbaric Medicine, Acapulco, Mexico, June 1984.
- 14 Goodin DS, Arnason BG, Coyle PK, Frohman EM, Paty DW. The use of mitoxantrone (Novantrone) for the treatment of multiple sclerosis: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2003;61:1332-1338.
- 15 Berger JR, Koralnik IJ. Progressive multifocal leukoencephalopathy and natalizumab—unforeseen consequences. *N Engl J Med* 2005;353:414-416.
- 16 Drazen JM. Patients at risk. *N Engl J Med* 2005;353:417.
- 17 Wang WZ, Olsson T, Kostulas V, et al. Myelin antigen reactive T cells in cerebrovascular diseases. *Clin Exp Immunol* 1992;88:157-162.
- 18 Palmer AC, Calder IM, McCallum RI, Mastaglia FL. Spinal cord degeneration in a case of “recovered” spinal decompression sickness. *Br Med J* 1981;283:888.
- 19 Dyer J, Millac P. Late deterioration after decompression illness affecting the spinal cord. *Br Med Bull* 1996;30:362-363.
- 20 James PB. Evidence for subacute fat embolism as the cause of multiple sclerosis. *Lancet* 1982;1(8268):380-386.
- 21 National Multiple Sclerosis Society. Disease Management Consensus Statement, May 2005. Available at: www.nationalmssociety.org. Accessed July 6, 2005.
- 22 Kindwall EP, McQuillen MP, Khatri BO, Gruchow HW, Kindwall ML. Treatment of multiple sclerosis with hyperbaric oxygen: results of a national registry. *Arch Neurol* 1991;48:195-199.
- 23 Perrins DJD, Neubauer RA, James PB. HBO therapy in multiple sclerosis. In: Jain KK. *Textbook of Hyperbaric Medicine*. 4th ed. Cambridge, Mass.: Hofgreffe and Huber; 2004:280-286.
- 24 Multiple Sclerosis National Therapy Centres. Oxygen. Available at: www.ms-selfhelp.org. Accessed July 7, 2005.
- 25 Perrins DJD, James PB. Long-term hyperbaric oxygenation retards progression in multiple sclerosis patients. *IJNN* 2005;2:45-48.

Hyperbaric Oxygen Therapy, or HBOT, is a specialized form of medical treatment administered by delivering 100% pure oxygen to the body through increased atmospheric pressure greater than 1.3 ATA in an enclosed hard chamber. At pressures greater than normal, the body can incorporate more oxygen into blood cells, blood plasma, cerebral-spinal fluid, and other bodily fluids. The increased oxygen absorption experienced during Hyperbaric Oxygen Therapy significantly enhances the body's ability to aid in its healing. The History of Hyperbaric. Though Hyperbarics has received a greater amount of atte PDF | Bennett M, Heard R. Treatment of multiple sclerosis with hyperbaric oxygen therapy. Undersea Hyper Med 2001; 28(3):117-122.- Despite considerable | Indeed, in preclinical models, demyelination is prevented if adequate oxygenation is maintained by raising inspired oxygen concentrations. In agreement with this interpretation, there is a predilection of lesions for the anterior and occipital horns of the lateral ventricles, namely regions located at arterial watersheds, or border zones, known to be especially susceptible to hypoperfusion and hypoxia. Finally, mitochondrial dysfunction due to genetic causes, as occurs in leucodystrophies or due to free radical damage, as occurs in MS, will compound any energy insufficiency resulting from hypo Hyperbaric oxygen therapy has long been a treatment for divers with decompression illness, but it has other uses, too. Find out more. What to expect. Controversy. Risks. Summary. Hyperbaric oxygen therapy (HBOT) involves breathing almost pure oxygen in a special room or small chamber. Its main use is to treat diving-related illness, but it may enhance healing in people with various other conditions. In 1662, a physician built the first hyperbaric chamber "a sealed room with a series of bellows and valves. The belief was that pressure could help treat certain respiratory diseases. In the 1940s, HBOT became standard treatment for military divers in the United States. There is no cure for multiple sclerosis (MS) yet. As a complex neurodegenerative disease of the brain, it is incredibly difficult to treat. Despite the development of new and sophisticated therapies to control the inflammation and physical symptoms of the disease, these treatments don't work for everyone. This is because MS comes in many guises and one treatment does not fit all. Perhaps for this reason people with MS are turning to alternative means of controlling their condition. Many of the 100,000 people with MS in the UK have taken charge of managing their treatment. With the assistance o