OUR EXPERIENCES IN HYPERBARIC MEDICINE

By

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Hyperbaric Oxygen therapy has been employed at the Institute of Aviation Medicine, Bangalore for the last 3 years in the treatment of conditions associated with poor oxygenation of tissues. Over 200 cases of peripheral artery disease, 3 of tetanus and 2 of extensive vascular injuries of limbs have been successfully treated so far with this mode of therapy. Therapy has not proved a success in the treatment of chronic osteomyelitis probably because of the inability to maintain antibiotic cover during the period of exposures.

Two patients of peripheral artery disease, while undergoing treatment with hyperbaric oxygen, developed involvement of central Nervous System with EEG changes. This involvement was attributed to Oxygen toxicity. Another 2 cases developed myocardial ischaemia 15 to 19 hours after exposure to hyperbaric oxygen. One of these cases, who was found dead in his bed, showed presence of a fresh thrombosis and evidence of an old healed myocardial infarction. None of the patients developed toxic manifestations in the respiratory system.

Besides the risks of oxygen toxicity, there are other associated problems a few of which are heat and humidity in the high pressure chamber, hearing defects in compressed air environments and likelihood of mechanical damage to the tympanic membranes due to repeated and intermittent exposures to high pressure. Some of these problems have been sutdied and overcome while others are being investigated.

Interest in hyperbaric medicine, originated for the first time in the 19th Century. This interest was, however, watered down by the observed oxygen toxicity. Paul Bert (Jacobson et al[8]), in the year 1868, was the first worker who recognised the existence of problem of Oxygen toxicity. Lorrain Smith (Hopkinson & Williams[7]) produced evidence of pulmonary oxygen toxicity in experimental animals. Extensive clinical use of Hyperbaric Oxygen (HBO) actually started in different institutions in the world about a decade ago. Germans used HBO as a therapeutic procedure for the first time in 1962, while the first reported use of this facility in Japan was made in 1963. We, in India started work on HBO therapy at the Institute of Aviation Medicine about 3 years ago. The high pressure chamber was originally established at the Institute to provide emergency treatment for cases of decompression sickness occuring during high altitude flying or during exposure in some of the simulators in use at the Institute. In early 1968, the chamber was

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proposed to be put into clinical use. By then, enough literature on HBO was available to justify the use of this facility in clinical practice. A few cases of peripheral artery disease were spotted in the Local Military Hospital, and a pilot study on the use of HBO therapy was conducted in these cases. The results of this study (Wadhawan et al [17]), although inconclusive, gave us an idea that HBO might be beneficial in the treatment of peripheral artery disease provided it was administered in adequate doses. Another fruitful observation made by us was that HBO was not as toxic as we thought it to be. Not much of literature was available on the role of HBO in peripheral artery disease. Whatever literature was available, was discouraging. Spurred by our initial observations, a bold approach to the problem was made. Based partly on the experiences gained in the pilot study and partly on the knowledge acquired after perusal of the available literature, a schedule of treatment was drawn up for peripheral artery disease. The schedule consisted of daily exposures to HBO at 2.5 atmospheres absolute (ATA) for one and half hours daily to a total number of 36 such exposures. A close watch on pulmonary functions was maintained throughout the period of therapy. Luckily, we had no adverse experiences and the treatment went through with a reasonable success. Each patiect was evaluated once a week. The parameters selected for this evaluation were "claudication time" on a bicycle ergograph, skin temparature and healing of ulcers, wherever applicable. Most of the cases responded to treatment. Those with trophic ulcers, which had not shown any sign of improvement before commencement of therapy, healed with HBO. We had our share of failures also. Some of the cases did not show any improvement. Subsequent amputations done in these cases revealed an extensive vascular involvement with virtual absence of bleeding at the time of operation. Obviously, the pathology in their blood vessels completely prevented access of oxygen to the distal anoxaemic tissues leading ultimately to dry gangrene. All cases with frank or threatening dry gangrene were, therefore, considered unsuitable for HBO theraby. Those patients who failed to show any improvement even after 10 days of the commencement of therapy were considered failures and their therapy was discontinued. Patients who did respond, completed, their therapy and were sent on 2 months sick leave followed by a re-evaluation. The improvement in these cases was progressive throughout the period of 36 days and even after completion of therapy (Tables I, II and III). The initial improvement shown by these patients with HBO therapy was not only found sustained but in a number of cases, it was even more manifest after sick leave than before (Wadhawan et al [18]). Inspite of our best efforts, majority of our patients failed to report back for a final evaluation planned 6 months later. Those cases who could be persuaded to report back to us for final evaluation did not show any aggravation or recurrence. One of these patients who returned 20 months later showed no evidence of recurrence. Another patient who was an airman working at the Institute reported no relapse even 18 months after completion of therapy.

TABLE I

Incidence of Ulcers Cured or Improved After HBO Therapy In 12 Cases

No. of cases with ulcers before therapy.	Number of cases after Therapy			Number of cases 2 months later		
	Cured	Improved	Not Improved	Cured	Improved	Not Improved
12	6	2	4	10	1	1

TABLE II

Mean of The Differences Between Mean Body Temperature And Peripheral Temperature (TOE) 37 Cases

(1) Before Therapy	(2) After Therapy	(3) 2 months later	(4) Difference between 1 & 2	(5) Difference between 1 & 3
4.8	3.5	2.9	N. S.	Significant (P=0.01)

A few other cases returned on their own for a second and in some cases a third course of therapy. Till today, we have provided HBO therapy to over 200 cases and still continue to do so. We are unable to account for the improvement shown by these patients. Arteriography studies might throw some light on this aspect but in view of some difficulties, these studies have not been carried out as a routine. Perhaps, in the near future, we may be able to do so. We presume that HBO keeps the tissues well oxygenated while the collateral circulation is developing which development continues, even after the cessation of therapy. This observation is based on the continued and sustained improvement noticed by us long after the therapy was discontinued. We are also unable to state whether the improvement shown by the patients is lasting. The fact stands that majority of these cases did show an improvement which lasted a minimum period of 2 months and much longer in a few cases. How this response compares with the other modes of therapy is a matter which should be examined by experts.

TABLE III

Bicycle Ergograph Data before Treatment, after Treatment and 2 months Later in 33* cases

(a) Before treatment and after HBO treatment.

Before treatment		After treatment (Time to pain in minutes)						
Time to pain in minutes	No. of Subjects	0	1–2	3–4	5–6	7–8	9–10	10
0	19	1	6	1	–	2	2	7
1–2	4	–	–	–	–	–	1	3
3–4	2	–	–	–	–	1	–	1
5–6	–	–	–	–	–	–	–	–
7–8	1	–	–	–	–	–	1	–
9–10	6	–	1	–	–	–	–	5
10	1	–	–	1	–	–	–	–
Total	33	1	7	2	–	3	4	16

(b) Before treatment and after sick leave.

Before treatment		After treatment (Time to pain in minutes)						
Time to pain in minutes	No. of Subjects	0	1–2	3–4	5–6	7–8	9–10	10
0	19	2	5	2	1	–	–	9
1–2	4	–	–	–	–	–	–	4
3–4	2	–	1	–	–	–	–	1
5–6	–	–	–	–	–	–	–	–
7–8	1	–	–	–	–	–	–	1
9–10	6	–	–	–	1	–	–	5
10	1	–	–	1	–	–	–	–
Total	33	2	6	3	2	–	–	20

* The remaining 4 cases were amputees.

Three cases of tetanus who were referred to us from St. Martha's hospital Bangalore on different dates, were treated with HBO at 3 ATA for 2 hours twice a day. From the time of commencement of therapy, ATS was discontinued and the patient was maintained only on antibiotics and sedatives (Chatterjee et al[5]). The first patient came to us within 36 hours after the first manifestation of disease. He showed distinct improvement after the very first exposure. He passed urine on his own and his rigidity subsided considerably. He was given a total number of 6 exposures and returned home cured. Another case of tetanus was sent to us in an advanced stage of disease. The patient was having seizures every few minutes and on slightest provocation. It was not possible to hold a mask on his face and he could not therefore be administered oxygen with success. HBO therapy was discontinued and the patient returned to hospital. He died next day. This was a failure but taught us a lesson which was beneficially employed in the next case who was referred to us 4 days after she was diagnosed and, as in the previous case, was having convulsions every few minutes. We got a tracheostomy done in this case and administered oxygen through the tracheostomy tubes; she was given a total number of 8 exposures of HBO over a period of 4 days and returned to the hospital after that for management of tracheostomy. This patient showed a distinct improvement which was manifest on the second day and, but for some residual neck rigidity, had fully recovered after the 8th exposure. Although the number of these cases is too small to permit any scientific conclusions, if correlated with the reports available in the literature, our limited experience in this field may provide avenues for an alternative safer and more reliable form of treatment of this disease. HBO is known to cause bacteriostasis of the anaerobes and oxidation of the tetanolysin (Brummelkamp et al[2]). It is also said to have a non specific effect on those anterior horn cells or cranial nerve nuclei which have become hyper-excitable because of the toxin getting fixed to these cells. HBO possibly makes these cells less excitable by some mechanism (Winkle et al[19]). However, this observation is open to criticism. At least our experiences in one case bilies this statement.

We have also had an opportunity of treating with HBO 2 cases of extensive vascular injuries of limbs threatening their blood supply, with impending gas gangrene. The credit goes to the surgeons of A. F. Hospital, Bangalore for their decision to with hold knife and try a few exposures of HBO. HBO was administered at 3 ATA for 2 hours daily on the first 3-4 days, followed by a gradual reduction to 2.5 ATA for one and half hours daily. The total duration of therapy with HBO lasted for 8-10 days. Not withstanding the bony injuries and their management, immediate amputations of limbs which were considered unavoidable because of extensive vascular damage, were averted in both these cases. HBO obviously helped by keeping the tissues well oxygenated while the collateral circulation was developing. Extensive vascular injuries calling for radical amputations are a common occurrence, both during war and peace. HBO, as a supportive therapy, should have a definite place in their treatment for saving both life and the limb. One has, however, got to exercise caution in planning the therapy. Severely toxic patients are likely to manifest toxicity at relatively low pressures of oxygen (Brummelkamp[3]). A convulsive seizure in a patient with an

* The remaining 4 cases were amputees.

extensive vascular or bony injury may lead to an irrepairable damage.

We have also had disappointments. Two cases of chronic osteomyelitis taken up for treatment with HBO, did not show the expected improvement. Increased pressure of oxygen in tissues leads to bacteriostasis of both aerobes and anaerobes and makes them more susceptible to antibiotics (Slack et al [14]). HBO, therefore, is not a substitute for the usual conventional form of treatment. Perhaps an appreciation of this aspect by all those concerned with the treatment would better serve the patients interest. Unfortunately, antibiotic cover in both our patients could not be adequately maintained due to certain reasons. The treatment was, therefore, discontinued.

HBO can also be used with advantage in a large number of other clinical conditions. It has been successfully employed in the treatment of Carbon Monoxide poisoning (Sluijter[15]), Cancer (Churchill Davidson and Emery[8]), Myocardial ischaemia (Cameron et al[4]), Ischaemic skin flaps (Perrins[13]) and as a supportive measure in cardiac (Meijne et al[11]) and brain surgery (Moor et al[12]). The therapy may, in short, be useful in the management of all cases associated with poor oxygenation of tissues.

While one can visualize a future for hyperbaric medicine, one has to be cautious in its usage. Like any other therapeutic procedure, HBO has its own hazards, if used indiscriminately. Oxygen at high pressures than those normally available in the atmosphere, is known to be toxic. It leads to enzymic changes with the consequent metabolic disturbances which, directly or indirectly, may be responsible for the toxic manifestations (Stadie et al[16]). We have observed 2 cases of toxicity of the Central Nervous System amongst our patients undergoing routine therapeutic exposures to HBO at 2.5 ATA (Krishnamurti and Wadhawan[9]). This form of toxicity has been reported to follow acute exposures to oxyzen at 4 ATA (Behnke[1]). We, however, feel that repeated exposures to subtoxic doses of HBO may, in some way, lower the threshold of neuronal irritability. Both these patients produced EEG changes which disappeared one week later.

HBO is also known to cause pulmonary damage which may result in oedema, atelectasis and thickening of alveolo-capillary membrane (Wittner & Rosenbaum[20]) causing diffusion disturbances. The earliest evidence of this form of toxicity has been stated to manifest in the Maximum Breathing Capacity and Vital Capacity (Lambertsen[10]) Frequent administration of HBO is reported to lead to cumulative effects on pulmonary functions.

In a recent work at the Institute, some of the pulmonary functions of patients intermittently exposed to HBO at 2.5 ATA were evaluated. It was observed that these exposures do not lead to any significant pulmonary damage provided the functions before commencement of therapy are within normal limits. Subnormal pulmonary functions, however, may predispose an individual to pulmonary oxygen toxicity with HBO.

Two of our patients developed myocardial infraction while undergoing treatment with HBO at 2.5 ATA. One of these patients, who died while asleep approximately 19 hours after his 22nd exposure, showed presence of a fresh

thrombus in the left coronary artery with evidence of an old healed myocardial infarction. The second patient developed an attack of acute coronary thrombosis about 16 hours after his 24th exposure. While HBO has been employed as a therapeutic procedure in myocardial ischaemia, it has not been known to precipitate an attack of this disease. The association of these cases with HBO, therefore, may be a mere coincidence.

HBO may also affect other physiological functions in the body. It has been postulated to produce lipid peroxidation of cell membrane which, in RBC may lead to haemolysis. This disturbance has been observed by Zinkle[21] after acute exposures to oxygen at 4 ATA.

HBO may also lead to disturbances in body tissues purely through its mechanical effects. We have observed macroscopic changes in tympanic membranes amongst our patients exposed repeatedly to high pressure environments. Hearing, in hyperbaric environments, may show disturbances because of increased density of air. In a work carried out at our Institute, we found a hearing loss of 15-20 db in frequencies around 3000 CPS at pressures of 2.5 ATA. This observation has necessitated training of both the chamber operator and the medical observer to speak slowly and pronounce each word separately and clearly while communicating with each other.

Another important biproduct of HBO therapy is the heat and humidity in the chamber specially when a number of subjects have to be simultaneously exposed for periods form 2 to $2\frac{1}{2}$ hours. Although a certain amount of ventilation is provided in the chamber, this is grossly inadequate when the number of subjects exceeds 3, more so when the atmospheric temperature and humidity are high. We have mitigated the heat stress to a great extent with the help of a locally developed 'Freon Heat Exchanger' installed inside the chamber.

At present, we in the Institute, are studying the possible effects of repeated exposures to HBO on EEG, blood and tympanic membrane.

Conclusions

From the experience gained so far, we feel that HBO therapy has a vast scope in clinical practice. For the therapy to achieve the success it merits, the clinician has to be closely associated with it since the dosage and duration of therapy has to be varied in each case with the progress shown by the patient, surgical intervention has to be appropriately timed and full antibiotic cover maintained throughout the period of therapy. It has also to be appreciated that HBO is a supportive form of treatment and not a substitute for surgery and other specific treatment. This support should be utilized early and not thought of as a last resort. The risk of Oxygen toxicity has to be kept in mind but not exaggerated. Lastly, it should be appreciated that the person administering HBO therapy is as much interested in following up his cases as any other clinician. Follow up of cases may not only satisfy his ego but also enable him to learn from his mistakes.

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