HYPERBARIC OXYGEN THERAPY IN HOSPITAL PRACTICE

SAHNI T.K. *
JOHN M.J. *
MADHWAL A.M. **
SHARMA J.C. ***
SINGH INDERJIT ****

Hyperbaric oxygen therapy (HOT) or hyperbaric oxygen (HBO) involves intermittent inhalation of 100% oxygen under a pressure exceeding that of the atmosphere, that is greater than 1 atmosphere absolute (ATA). Therapy is given in recompression chambers which were primarily used to treat illnesses of deep sea divers. Over the last two decades, HBO therapy has been found to be useful in a large number of medical and surgical ailments and this has led to a renaissance of HBO the world over. At the Institute of Naval Medicine, patients are referred mainly from Naval Hospital Asvini, St. Georges and Bombay Hospital. An analysis of patients administered therapy in the last 5 years is presented along with a review of literature outlining other accepted categories of hospital patients who can benefit from this therapy.

Introduction

HBO therapy involves intermittent inhalation of 100% O2 under a pressure exceeding that of the atmosphere, that is greater than 1 atmosphere absolute (ATA).[1,2] As is true for many practices in medicine, there has also been a dynamic flux in its use, going through a typical evolution in which there is initially great enthusiasm and wide spread use, followed by a gradual decline.[3] In the 1960's HBO was used with great enthusiasm for a variety of ailments and Gibb and Robin list 132 past and present indication for HBO.[3] However as more and more clinical trials, showed little benefit with HBO therapy, it was viewed with skepticism by physicians.[3]

Over the last two decades, animal studies, clinical trials, and greater experience has produced a set of indications for which HBO appears to be beneficial.[1,2] This has led to a renaissance of HBO,[4,5] and hyperbaric facilities now form an important part of the intensive care units of many hospitals, in the UK and USA.[5] In the United States there has been an annual increase in the number of hyperbaric centres and increase in patients at the rate of 15 and 620 respectively. In 1990 there were 216 functional units and the number of patients treated annually had increased from 896 in 1971 to 12,047 in 1989.[4]

The main impetus for the development of clinical hyperbaric medicine was the 1975 hyperbaric Medicine Workshop held at University of California. The deliberations

* Specialist in Marine Medicine and Physician
** Head of the Dept of Marine Medicine
*** Director Institute of Naval Medicine
**** Commanding Officer INHS Asvini
Institute of Naval Medicine INHS Asvini, Colaba, Bombay 400 005.

of this group resulted in a textbook 'Hyperbaric oxygen therapy'[6] which has become the bible of clinical hyperbaric medicine.[7]

In Nov 1976 "The Undersea and Hyperbaric Medical Society (USA)" established a "Committee on Hyperbaric Oxygen", which was given the responsibility of presenting a sound rationale for general medical opinion, so as to accept HBO as a clinical treatment modality. The committee has listed the indications for HBO into two broad categories' Currently accepted' and "Investigational".[7]

However in India there has been no interdisciplinary review of HBO. And thus though sufficient evidence supports use of HBO in certain defined conditions, many patients go untreated because of the physicians unfamiliarity with recent research and overall uncertainty about the legitimacy and safety of HBO as therapy.

Mechanism of Action

HBO therapy can be given in a "Monoplace chamber" in which a single patient is placed in a chamber which is then pressurised with 100% Oxygen, or it can be given in a "Multiplace chamber" which is pressurised with compressed air and the patient breathing 100% oxygen at that pressure through a mask or hood or endotracheal tube. Most therapy is given at 2 or 3 ATA.[1]

Therapeutic Effects

1. **Mechanical effects**: In diseases such as air embolism and decompression sickness, the therapeutic effect is achieved by reduction in bubble size by increase in ambient pressure.[1]

2. **Physiological effects**: With increase in pressure, there is an increase in the amount of O_2 dissolved in the plasma. This facilitates O_2 diffusion to hypoxic tissues and enhances growth of fibroblasts, formation of collagen, angiogenesis and enhances the phagocytic capabilities of hypoxic leucocytes.[2] HBO has action on bacterial growth where in, it is bactericidal for Clostridia Welchii, and also inhibits the growth of aerobic bacteria at pressures greater than 1.3 ATA.[8] By providing molecular oxygen to neutrophils, it enhances their microbial killing and causes immune stimulation.[1] Action on the cardiovascular system is seen in the form of vasoconstriction but with an overall increased delivery of oxygen due to the hyperoxia. This is used in reducing oedema and tissue swelling, while maintaining oxygenation and is specially of use in burns, cerebral oedema and crush injuries.[1] HBO accelerates neo-vascularization in hypoxic areas and as new capillary beds are formed, the hypoxic zone diminishes, and there is increased capillary diffusion of oxygen. This is the basis of use in the therapy of radiation necrosis.[2]

Toxic Effects/Complications

Complications can arise in air filled cavities during change in pressure and are called, Barotraumas. Commonest is aural barotrauma as a result of inability to equalize pressure on both sides of the tympanic due to a blocked eustachian tube. Pneumothorax and air embolism are more dangerous complications due to tear in pulmonary vasculature but are not so common.[6]

Pulmonary oxygen toxicity (POT) (Lorraine Smith effect) and Neurological oxygen toxicity (Paul Bert effect) can occur due to breathing 100% Oxygen under pressure for prolonged periods at low pressure and short periods at high pressure respectively.[9]

Other toxic effects documented include

retrolental fibroplasia and cataracts after prolonged HBO therapy. Fire is a realistic hazard and the patient may be claustrophobic.[1] Rare instance of hypersensitivity to O_2 are also documented.[10]

Indications for HBO Therapy

Table 1 shows the "Current Conditions Classification" as recommended by the Undersea and Hyperbaric Medicine Society (UHMS).[7] Conditions included in the "Accepted" category are those in which HBO has shown without doubt to be effective in obtaining therapeutic results as either the primary or adjunctive mode of treatment. In the "Investigational" category are those conditions where the clinician is encouraged to undertake research to prove the efficacy of HBO and rationalise the mechanism of action.[7]

Review articles published in many reputed journals have placed HBO therapy in its proper perspective. Gabb and Robin in the "Chest" have highlighted the controversies relating to HBO and have documented 132 past and present indications for HBO therapy.[3] Colonel Fredrick S. Kramer in Medical Corps International has discussed the role of HBO therapy in the care of the injured soldier.[11] Gerald H Cohn in Post graduate medicine has discussed the role of HBO therapy in promoting healing in difficult cases.[2] Gothleb, Grim RS et al in the JAMA have reviewed HBO therapy in its present perspective.[1] Use of HOT in treatment of severely brain injured patients is reported by Rockswold et al.[12] Cerebral air embolism has been treated by Catron PW successfully.[13] Kindwall et al discuss the role of HBO therapy in Plastic Surgery,[14] and Urayama H et al, the role in occlusive arterial diseases in the extremities.[15] Role of hyperbaric oxygen in osteomyelitis is discussed by Calhoun JH et al,[16] and its use in necrotising fascitis by Risemann et al.[17] A trial of HBO in neonates is reported by Vazquez RL et al.[18] Besides these articles, scattered reports of use of HBO in various pulmonary disorders, neurological disorders, non healing ulcers, infectious

Table 1
HBO—Current Conditions Classification

Accepted*	Experimental/Research
1. Radiation Necrosis	1. Head and spinal cord injury (traumatic.)
2. Decompression sickness	2. Bone grafts
3. Carbon monoxide poisoning	3. Carbon tetra chloride poisoning
4. Gas embolism	4. Cerebrovascular accidents—Acute (thrombotic/embolic)
5. Gas gangrene	5. Fracture healing
6. Osteomyelitis (Refractory)	6. Intra abdominal and intracranial abscesses
7. Soft tissue infections due to mixed aerobic and anaerobic organism with tissue necrosis.	7. Hydrogen sulphide poisoning
8. Crush injuries with traumatic ischaemia	8. Lepromatous leprosy
9. Compromised skin grafts or flaps and healing in problem wounds	9. Meningitis
10. Selected refractory mycoses	10. Pseudomembranous colitis.
11. Acute cyanide poisoning	11. Radiation myelitis, cystitis, enteritis, proctitis.
12. Cerebral oedema, acute.	12. Sickle cell crisis.
13. Thermal burns	13. Retinal artery insufficiency
14. Anaemia-exceptional blood loss	14. multiple sclerosis
	15. Retinopathy
	16. Pyoderma gangrenosum and actinomycosis

* reimbursed by Insurance

diseases etc are also available in journals of various descriptions. "The Hyperbaric Oxygen Review"[19] is a journal dealing only with the clinically significant uses of HBO. Proceedings published during annual scientific meetings of the UHMS and European UHMS are also a rich source of scientifically compiled information.

This renewed interest in HBO therapy has withstood the tests of scientific scrutiny, and it is now accepted as a therapeutic modality in established clinical situations. The hyperbaric chamber is now an integral part of hospital services and/or Intensive Therapy Unit in the British Isles and in centres in the USA.[5]

Table 2
HBO Therapy given at institute of naval medicine Bombay (both monoplace and multiplace)

Year	No. of HBO sittings	No. of patients		
		Monoplace	Multiplace	Total
1992	335	37	6	43
1991	503	31	20	51
1990	620	30	25	55
1989	417	30	22	52
1988	246	19	7	26
Total	2121	147	80	227

Table 3
Analysis of patients administered hot accepted category

Disease name	1992	1991	1990	1989	1988
Radiation necrosis	01	—	—	—	—
Decompression sickness	01	02	—	01	—
Gas embolism	—	—	—	01	
Gas gangrene	02	08	—	—	01
Osteomyelitis	01	—	—	01	01
Soft tissue infections	12	02	06	05	05
Crush injury/traumatic ischaemia	10	07	05	07	03
Healing in problem wounds	16	17	14	17	08
Cerebral oedema (acute)		01			
Burns	02		02	04	01
Total	45	37	27	36	20

Experimental Category

Disease name	1992	1991	1990	1989	1988
Head and spinal cord injury	02	—	02	02	—
Cerebrovascular disorders (Including frost bite)	—	—	03	—	—
Fracture healing	07	16	15	12	01*
Abscess	—	—	06	—	01
Retinopathy	01	—	01	—	—
Pyoderma gangrenosun	—	—	01	01	—
Refractory anaerobic infections and actinomycosis	—	—	01	—	—
Liver diseases	04	04	06	03	07*
Bells palsy	—	—	01	—	01*
Total	14	20	36	18	10

* Projects taken up at the Institute of Naval Medicine.

Table 4
Institutions referring patients for HBO therapy

	1992	1991	1990	1989	1988
INHS Asvini	13	25	23	32	14
Bombay Hospital	24	26	31	17	10
St. Georges	13	—	1	—	01
Others	03	—	—	02	01
Total	43	51	55	52	26

Our Experience

At the Institute of Naval Medicine we have both a monoplace and a multiplace chamber and over the last 5 years (records of which have been analyzed), we have treated 227 patients and a total of 2121 HBO sittings have been given (Table 2). A larger number of patients have been treated in the monoplace as compared to the multiplace chamber.

Maximum number of patients administered HBO therapy were to help in the healing of problem wounds such as non healing ulcers, bed sores, gangrene etc. Traumatic ischaemia and crush injuries along with soft tissue infections formed another large group. As in other centres of the world decompression sickness forms a very small percentage. Special interest at our centre has been in the use of HBO in Liver diseases, fracture healing and Bells Palsy and research projects are being carried out on these conditions (Table 3).

On analysis it was interesting to note that the maximum number of patients treated were from Bombay Hospital followed by Naval Hospital, St. Georges and others (Table 4).

Conclusion

The role of hyperbaric oxygen therapy is scientifically established in certain well defined conditions, commonly encountered in hospital practice. Doctors in all fields must familiarise themselves with recent thinking on this mode of therapy, so that their patients are not denied the gains of this modern treatment.

REFERENCES

1. Grim PS, Gottlieb LJ, et al; Hyperbaric oxygen therapy (review); JAMA, 1990; 263 (16):2216-20.
2. Cohn GH; Hyperbaric oxygen therapy—promoting healing in difficult cases; Postgraduate medicine, 1986, 79(2): 89-92.
3. Gabb G, Robin ED; Hyperbaric oxygen: a therapy in search of disease; Chest, 1987, 92:1074-82.
4. Myers RAM; The Maryland institute for emergency medical service system hyperbaric oxygen registry—an analysis of two decades, Annual scientific meeting of the Undersea and Hyperbaric medical society; Undersea Biomedical Research, 1991, Vol. 18.
5. Personal communication from Dr. Hamilton Farrell, Vice chairman of BIGHT (British Isles Group of Hyperbaric therapists) Whipps Cross Hospital London.
6. Davis JC, Hunt TK; Hyperbaric Oxygen Therapy; Undersea Society. Bethesda MD (USA); 1986.
7. Myers RAM, Schnitzer M.B.; Hyperbaric Medicine; State of the Art 1984, Proceedings of Eighth International Conference on Hyperbaric Medicine; Best Publishing Co. California 1987; 294-303.
8. DJ Bakker; The use of Hyperbaric Oxygen in the treatment of certain infectious diseases especially gasgangrene and acute dermal gangrenes Drukkerij Veenam BV, Wageninger 1984, 24-29.
9. Edmonds C, Lowry C, Penkefather J; Oxygen toxicity, In Diving and Subaquatic Medicine. Biomedical Marine Service, Sea forth 2092; Australia, 1984: 244-46.
10. Van Den Brenk: The hazards of Oxygen toxicity in a Hyperbaric Environment; In GS Innes: The production and hazards of a Hyperbaric Oxygen Environment. Pergamon Press, London 1970:49.
11. Cramer FS; Care of the Injured Soldier: A medical Readiness role for Clinical Hyperbaric Oxygen

Therapy; Medical Corps International; 1990 Vol 5, No. 2.

12. Rockswold GL; Ford SE; et al; results of a prospective randomised trial for treatment of severely brain injured patients with Hyperbaric Oxygen; Journal of Neuro surgery June 1992; 76(6):929-34.
13. Catron PW, Dutka AJ, Biondi DM et al: .Cerebral Air Embolism treated by Pressure and Hyperbaric Oxygen; Neurology, 1991:41(2(pr 1)):314-5.
14. Kindwall EP; Gottlieb FJ; Larson DL; Hyperbaric Oxygen therapy in Plastic Surgery, Plastic and Reconstructive therapy, 1991: 21, 888-908.
15. Urayama H, Takemura H, Kasajima F, et al; Hyperbaric Oxygen therapy for chronic occlusive arterial diseases of the extremities; Journal of Japanese Surgical Society, Apr 1992; 93(4):429-33.
16. Calhoun JH, Cobos JA, Mader JT; Does Hyperbaric Oxygen have a place in the treatment of Osteomyelits? Orthopaedic Clinics of North America July 1991: 22(3):467-71.
17. Riseman JA; Zambons WA; Curtis A, et al; Hyperbaric Oxygen for necrotising fascitis reduces mortality and need for debridements; Surgery Nov 1990: 108(5):847-50.
18. Vazquiz RL, Spahr RC; Hyperbaric Oxygen use in Neonates. A report of four patients; American Journal of Diseases of Children Sept 1990: 144(9):1022-4.
19. Hyperbaric Oxygen Review, journal of the undersea medical society, bethesda MD. (USA).

CLASSIFYING ACUTE LIVER FAILURE

"Acute" or "fulminant" liver failure encompasses conditions with various courses and causes' and several classifications, all implying that the syndrome begins with encephalopathy, have been proposed. In 1986 we suggested that "fulminant" and "subfulminant" liver failure could be distinguished by the duration of the interval between the onset of jaundice and the onset of encephalopathy, shorter or longer than 2 weeks, respectively.

A new terminology, still based on the interval btween jaundice and encephalopathy, is now proposed by O'Grady et al, who suggest hyperacute, acute, and subacute liver failure, depending on whether this interval is 0-7 days, 8-28 days, or 29 days to 12 weeks, respectively. Like us, O'Grady et al include cases of pre-existing symptomless chronic liver conditions, thus dropping Tery's criterion of no history of hepatic disease. O'Grady et al note the prognostic value of their classification, survival rates being 36% in hyperacute but only 7% and 14% in acute and subacute cases. Although prognosis was not the main objective of our distinction between fulminant and subfulminant, we too found the case fatality rate to be higher with the slower (subfulminant) course. Furthermore, the term "acute liver failure" is now ambiguous, being used, to designate both the whole syndrome and a particular subgroup of patients, Lastly, O'Grady et al emphasise 'encephalopathy as the "mandatory clinical feature" for a diagnosis of acute hepatic failure. We disagree. Encephalopathy is not specific to liver failure; it does not always reflect deterioration in liver function. Second, comparison of patients with acute liver disease and encephalopathy based on cause, age, and prothrombin or factor V level (all independent prognostic indicators) will be more accurate than that based only on the interval between the onset of jaundice and encephalopathy, an independent prognostic factor in London but not in Paris. Finally, we think that the liver fails before encephalopathy. The pre-encephalopathy stage is characterised by a fall to below 50% of normal coagulation factor concentrations. At this early stage, which can last from a few hours to several weeks, encephalopathy may or may not develop, much more importantly, referral to a liver unit is associated with a high survival rate.

We would extend the concept of acute liver failure to all cases of acute liver disease where prothrombin or factor V concentrations are below 50% of normal and employ the terms fulminant or subfulminant liver failure as soon as encephalopathy develops during acute failure. This terminology, covering the entire spectrum of the failing liver syndrome in acute liver disease, should encourage earlier diagnosis and improve assessment of prognosis, thus reducing the requirement for emergency transplantation.

Lancet, 1993: Vol. 342, 252