Use of hyperbaric oxygen therapy in Hong Kong

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The Recompression Treatment Centre on Stonecutters Island has been operating in Hong Kong for more than 5 years and has been used to treat a variety of diving-related and other conditions by means of hyperbaric oxygen therapy. Up to the end of December 1997, 295 treatment sessions had been conducted for 39 patients. This article reviews the usefulness of and indications for hyperbaric oxygen therapy.

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Introduction

Up to 1994, recompression therapy was provided in Hong Kong by the United Kingdom (UK) Royal Navy at one of its bases, the HMS Tamar. Therapy at the base was only available for the treatment of diving-related conditions. As the presence of the UK Armed Forces in Hong Kong was scaled down in the years leading up to the change of sovereignty in 1997, a full Royal Navy diving unit could no longer be maintained, and the recompression chamber was closed down and returned to the UK. The functions of HMS Tamar were then moved to Stonecutters Island. With the change of government, recompression treatment became the responsibility of the Hong Kong Government.

The Recompression Treatment Centre (RTC) on Stonecutters Island was opened in April 1994. Hyperbaric oxygen (HBO) therapy is given to patients by means of a compression chamber. The facility is operated by the Hong Kong Fire Services Department under the medical supervision of the Occupational Medicine Division of the Department of Health.

Mechanism of action and effects of hyperbaric oxygen therapy

The basic mechanism of action involves the increase of oxygenation of tissue fluids. The normal arterial oxygen partial pressure (PO_2) of inspired atmospheric air is approximately 0.2 bar (1 bar is approximately 1 atmosphere absolute, or 100 kPa, or 750 mm Hg). The maximum PO_2 that can be theoretically achieved by using normobaric 100% oxygen therapy is 1.0 bar. In practice, it is very difficult to achieve this level because of the improper fit of the equipment. By raising the ambient pressure in the chamber and by breathing 100% oxygen through a tight fitting mask, however, the inspired PO_2 can be raised to levels up to 3.0 bar. The inspired oxygen levels that are most commonly used range from 2.4 to 2.8 bar. At these levels, the amount of oxygen in simple solution in the plasma is enough to meet all the needs of the body,[1] and haemoglobin is saturated on the venous side. Beneficial effects of increased oxygenation include the suppression of clostridial alpha-toxin production,[2] enhancement of the killing ability of leukocytes,[3] increase in fibroblast growth and collagen formation,[1] reduction in tissue oedema,[4] and increased capillary proliferation.[5] The Box lists the various conditions that benefit from HBO therapy.[6,7]

Side effects of hyperbaric oxygen therapy

There are few side effects to HBO therapy. Uncommonly, the following may occur: cough, dyspnoea, retrosternal discomfort, pulmonary oxygen toxicity, and convulsions.[1] Before compression is commenced, patients must have their ability to equalise pressure across both tympanic membranes and all sinuses checked, and care must be taken to assess eustachian and sinus function. Eustachian function is often compromised by ostial damage from radiation treatment (eg cancer patients) and it is appropriate that this group of patients be referred to an ear, nose, and throat specialist for an opinion before therapy is begun. Patients with upper respiratory tract infections or sinusitis should be considered unfit for therapy

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Conditions for which hyperbaric oxygen therapy is useful [1,2]

Related to diving and compressed-air work
Decompression illness
Arterial gas embolism

Acute indications
Carbon monoxide poisoning
Clostridial myonecrosis
Soft-tissue necrotising infections

Chronic indications
Radiation tissue damage
Refractory osteomyelitis
Enhancement of healing in selected problem wounds

until they have recovered. If urgent treatment is required, a prophylactic myringotomy is sometimes performed.

Contra-indications

Concurrent drug therapy may interact with HBO therapy. Patients being treated with doxorubicin, disulfiram, cisplatin, or mafenide acetate should not receive HBO therapy, although therapy may be possible when the course of treatment has been completed.[1] In addition, patients with an untreated pneumothorax cannot be given HBO therapy; however, therapy can proceed in a patient with a chest drain in situ. Patients who have recently had a subclavian line inserted should have a chest X-ray before therapy is started.

Conditions related to diving and compressed-air work that require hyperbaric oxygen therapy

Decompression illness

Decompression illness results from the presence of bubbles of inert gas, usually nitrogen, in the body tissues. At depth, gas dissolves in the body tissues but as decompression occurs, the solubility of the gas falls. If the rate of decompression is gradual, the excess tissue inert gas load can be removed by the passive transfer of gas down a concentration gradient from the tissues to the blood and then to the lungs, where blood gas tensions can equilibrate with those of the inspired gas mixture. If the rate of decompression exceeds the capacity of this mechanism to remove gas, then bubbles form in the tissues. The individual's symptoms will depend on the location and quantity of bubbles that form. Table 1 shows some examples of presenting symptoms. Decompression illness can appear from 10 minutes to 2 weeks after exposure; more severe illness usually presents earlier.[8] All persons suspected of having decompression illness should be given 100% oxygen and referred for recompression therapy without delay.

Pulmonary barotrauma

Decompression barotrauma most often affects the lung. The cause may be a pre-existing pulmonary disorder (resulting in air trapping) or breath-holding during ascent. There may be chest pain, shortness of breath, dyspnoea, cough or mild haemoptysis, change in voice tone or subcutaneous emphysema present. In some cases, the patient may have a frank pneumothorax because alveolar gas has escaped into the pleural space. The symptoms are commonly of a sharp pain, which is worse on inspiration, and shortness of breath. There is mediastinal shift to the affected side, a hyperresonant percussion note, reduced breath sounds and movement on the affected side, and cyanosis. A frank pneumothorax can become a tension pneumothorax on ascent, with tracheal shift away from the affected side, cyanosis, shock, and unconsciousness developing. Death can result if the pressure is not relieved by insertion of a chest drain.

The most catastrophic effect of pulmonary barotrauma is when an arterial gas embolism arises. In this situation, gas from a ruptured lung enters the pulmonary circulation and travels to the left side of the heart via the pulmonary vein. It causes an 'air lock' in whichever artery it ends up in; symptoms depend on the target organ deprived of blood. In the case

Table 1. Examples of presenting symptoms of patients with decompression illness

System	Symptoms
Skin	Formication, pruritus, rashes, mottling or marbling
Joints	Dull ache, not usually made worse by movement, with an often insidious onset, poor localisation, and no classical signs of inflammation (shoulder and knee often affected)
Respiratory	Chest tightness, retrosternal pain
Neurological	Symptoms can be multifocal and subtle, and can involve the central and peripheral nervous systems, eg paraesthesia, anaesthesia, loss of coordination, visual and auditory disorientation, drowsiness, unconsciousness, convulsions, and sphincter control can be impaired
Audiovestibular	Vertigo, nystagmus, loss of hearing, and tinnitus

of the brain, the presentation is similar to that of a cerebrovascular accident and in the heart, similar to myocardial infarction. This condition requires emergency recompression and patients must be sent to the chamber by the quickest available means.

Both decompression illness and arterial gas embolism are usually treated with oxygen at a pressure of 2.8 bar. More than one treatment may be required.

Acute conditions requiring hyperbaric oxygen therapy

Carbon monoxide poisoning

Patients with carbon monoxide poisoning benefit from HBO therapy by a variety of mechanisms. The carbon monoxide combines with haemoglobin to form carboxyhaemoglobin (COHb) but also has a considerable affinity for cerebral mitochondrial cytochromes. The half-life of carbon monoxide within the cerebral mitochondria is approximately 10 times longer than that of COHb and consequently, although normobaric oxygen can rapidly reduce COHb levels, the brain can still contain large amounts of carbon monoxide.[9] For this reason, it should be noted that the clinical condition of the patient is more important than the measured COHb level when determining if HBO therapy should be used or not. Those with low COHb levels who show severe poisoning or have a recurrence of symptoms as well as those with COHb levels above 40% are recommended for referral to receive hyperbaric oxygen therapy. Levels of carbon monoxide in the brain and COHb are reduced by a high inspired PO_2 and full oxygenation can be restored but the main effect is probably that of lipid peroxidation stopping. Carbon monoxide is also particularly dangerous to the foetus, and HBO therapy is strongly recommended for pregnant women who have had significant exposure to this gas.[10,11]

Therapy with HBO is also effective in preventing the delayed neuropsychiatric problems that often develop 10 to 21 days after severe carbon monoxide poisoning.[12,13] Regular psychometric testing is therefore necessary, as such testing usually determines the number of therapy sessions an individual needs. Therapy is usually at a pressure of 2.8 bar and takes between 1 hour 30 minutes and 2 hours 15 minutes. More than one session is usually required; the average is four.

Clostridial myonecrosis

Gas gangrene arises because of infection of tissue by anaerobic bacteria; *Clostridium perfringens* is responsible in a majority (>90%) of cases.[1] The infections are very fast-moving and life-threatening owing to the extremely pathogenic nature of the organism. *Clostridium perfringens* produces alpha-toxin, which liquefies muscle on contact, is profoundly haemolytic, and necroses renal tubules. The result is profound and often fatal septic shock. The classical treatment is aggressive antibiotic therapy combined with radical debridement or high amputation. Even so, mortality can be high and usually more than 15%.[8]

Hyperbaric oxygen is toxic to clostridia and acts to shut down the production of alpha-toxin.[2] Using HBO means that the infection can be controlled, the patient can be brought out of shock, and the immediate requirement to surgically remove the infected area can be eliminated. Subsequently, debridement of dead tissue only may be carried out and any amputation can be more conservative or avoided. Therapy is performed at a pressure of 3.0 bar for 1 hour 30 minutes and is used three times within the first 24 hours and twice daily for the next 4 to 5 days. The addition of HBO therapy to the combination of surgery and antibiotics can reduce mortality to around 6%.[14]

Crush injuries and compartment syndrome

A crush injury results in considerable damage to the microcirculation because of the mechanical trauma and oedema. In severe cases, the oedema may progress to compartment syndrome whereby the damaged microcirculation is unable to allow erythrocytes to pass and the area deprived of circulation consequently becomes ischaemic or progresses to become necrotic. Because HBO therapy can fully supply oxygen needs by solution in plasma, the compromised areas become oxygenated; oedema is also reduced and the area is able to revascularise.[4] Therapy is carried out at a pressure of 2.4 bar for 1 hour 30 minutes and is usually conducted three times daily for 2 days, followed by twice daily for 2 days, then daily for 2 days.

Soft-tissue necrotising infections

Necrotising infections of soft tissue include necrotising fasciitis and Fournier's gangrene. The mortality in these conditions is high and, although the primary treatment is surgery with antibiotics, giving HBO is useful as an adjunctive therapy. Therapy is given at a pressure of 2.0 to 2.5 bar for 1 hour 30 minutes twice daily initially and then daily; up to 30 sessions may be required. Necrotic fascia and skin should be excised prior to HBO therapy.[8] Tissues that have a compromised blood supply are preserved by HBO and the killing activity of leukocytes is enhanced. When infections are due to anaerobes, HBO has a toxic effect on the organisms.[14]

Chronic conditions requiring hyperbaric oxygen therapy

Radiation tissue damage

Radiation tissue damage most commonly follows radiotherapy treatment for malignant disease. The radiation destroys the malignant tissue but also causes the death of cells in the vicinity. Blood vessels gradually sclerose over several months and cells that die are not replaced. Consequently, tissue becomes hypovascular, hypocellular, and hypoxic. The changes may be severe enough to lead to radionecrosis, particularly if the tissue suffers further insult such as surgery or other wounding; the tissue is too hypoxic to either heal spontaneously or to accept grafts. Prior to the advent of HBO therapy, the only solution was to remove irradiated tissue and replace it with fresh tissue in the form of grafts or free-tissue transfer. In the case of osteoradionecrosis of the mandible, this procedure resulted in the low cure rate of only 8%.[5]

Hyperbaric oxygen therapy raises the PO_2 in the irradiated area and allows fibroblast division, collagen production and neoangiogenesis.[1] The growth of new capillaries enables the area to be oxygenated after therapy stops and permits healing and graft acceptance. The combination of HBO with surgery means that cure rates in excess of 98% can be achieved.[5] Therapy sessions are given for 1 hour 30 minutes at 2.4 bar. Depending on the stage of the osteoradionecrosis, protocols require that 20 or 30 sessions be given before surgery and a further 10 sessions be given afterwards.

Refractory osteomyelitis

The usual reason for the persistence of bone infections is ischaemia and loss of white cell effectiveness due to a low tissue PO_2. As a protective measure, areas of infection that cannot be eradicated by natural defences are walled off by fibrosis. This process further lowers the PO_2 and also disrupts blood supply, making it more difficult to deliver antibiotics to the infected area.[1] Traditionally, surgery is used to remove dead, compromised and walled-off tissue, thereby leaving bleeding bone.

Table 2. Use of the Recompression Treatment Centre from April 1994 to December 1997

Year	No. of patients	Total No. of treatment sessions
1994	5	9
1995	9	58
1996	16	113
1997	10	116
Total	39*	295

* One patient was admitted in 1995 and again in 1996

Raising the tissue PO_2 by using HBO enables neovascularisation to occur and consequently gives antibiotics access to the infected area.[15] The ability of leukocytes to kill bacteria is also enhanced by a high PO_2. Patients in whom conventional therapy has been ineffective and those with infections of the skull or sternum should be considered candidates for HBO therapy.[8] Therapy sessions last 1 hour 30 minutes at a pressure of 2.4 bar. Many treatments are sometimes necessary.

Problem wounds

Problem wounds are typically ischaemic, lower limb wounds or ulcers in people with diabetes or arteriosclerosis. For patients with very poor large vessel patency, healing is unlikely even with HBO. Doppler studies or angiography may be required and may need to be followed by angioplasty or bypass grafting.[8] Therapy usually consists of once- or twice-daily sessions lasting 1 hour 30 minutes each at 2.4 bar.

Use of the Recompression Treatment Centre

Table 2 shows data for the figures relating to the use of the RTC from when it first opened, in April 1994, to December 1997. Individuals with decompression illness provided most of the admissions (Table 3), thus reflecting the large number of Hong Kong residents who dive (particularly for recreational purposes). A smaller proportion of patients were admitted for carbon monoxide poisoning and radionecrosis; one case of osteomyelitis of the skull and a patient with Klippel-Trénaunay-Weber syndrome were also treated. As the pathology of the latter was of an ischaemic nature, a course of 30 sessions at 2.4 bar was used for the patient.

Table 3. Causes of admission to and treatment at the Recompression Treatment Centre from April 1994 to December 1997

	Diagnosis				
	DCI* or suspected DCI	Carbon monoxide poisoning	Radionecrosis	Other	Total
Patients (No. [%])	25 (64)	7 (18)	5 (13)	2 (5)	39
Treatment session (No. [%])	35 (12)	19 (6)	172 (58)	69 (23)	295

* DCI decompression illness

Analysis of the workload of the RTC shows that many patient sessions were devoted to the treatment of radionecrosis (Table 3). This finding reflects the prolonged treatment regimen that these patients need to receive.

Conclusion

Local experience has shown that giving HBO is a very useful adjunctive therapy. However, due to the remote location of the RTC at Stonecutters Island and the lack of hospital facilities, the problems of distance and the risks and cost of transporting potentially critically ill patients raise some unique difficulties. Strong support from the referring hospital is essential if further development of the potential of the RTC to treat conditions other than diving-related ones is to occur.

References

1. Kindwall E. Hyperbaric medical practice. Flagstaff (AZ): Best Publishing Company;1994.
2. Van Unnik AJ. Inhibition of toxin production in *Clostridium perfringens* in vitro by hyperbaric oxygen. Antonie Van Leeuwenhoek Int J Gen Mol Microbiol 1965;31:181-6.
3. Adams KR, Roberts RM, Mader JT. In vitro killing of *Clostridium perfringens* by oxygen with and without polymorphonuclear leucocytes [abstract]. Undersea Biomed Res 1990; 17(Suppl):123S.
4. Nylander G, Nordstrom H, Lewis D, Larsson J. Metabolic effects of hyperbaric oxygen in post-ischaemic muscle. Plastic Reconstr Surg 1987;79:91-6
5. Marx RE. A new concept in the treatment of osteoradionecrosis. J Oral Maxillofac Surg 1983;41:351-7.
6. Undersea and Hyperbaric Medicine Society. Hyperbaric Oxygen Therapy Committee Report, 1996. Kensington (MD): Undersea and Hyperbaric Medicine Society; 1996.
7. Camporesi EM. Recommendations of the Jury Lille. Proceedings of the European Consensus Conference on Hyperbaric Medicine; 1994; Lille.
8. Bennett P, Elliott D. The physiology and medicine of diving. 4th ed. London: WB Saunders Company Ltd.; 1993.
9. Cross M. Carbon monoxide poisoning. Guidelines for referral of cases to the South Western Medical Centre, Plymouth. Plymouth: South Western Medical Centre; 1993.
10. Hollander KE, Nagey D, Welch R, et al. Hyperbaric oxygen therapy for the treatment of acute carbon monoxide poisoning in pregnancy. J Reprod Med 1987;32:615-7.
11. Van Hoesen KB, Camporesi EM, Moor RE, et al. Should hyperbaric oxygen be used to treat the pregnant patient for acute carbon monoxide poisoning? A case report and literature review. JAMA 1989;261:1039-43.
12. Werner B, Back W, Akerblom H, Barr PO. Two cases of acute carbon monoxide poisoning with delayed neurological sequelae after a ‘free’ interval. J Toxicol Clin Toxicol 1985;23:249-65.
13. Youngberg JT, Myers RA. Use of hyperbaric oxygen in carbon monoxide, cyanide and sulphide intoxication. In: Camporesi EM, Barker AC, editors. Hyperbaric oxygen therapy: a critical review. Bethesda (MD): Undersea & Hyperbaric Medicine Society; 1991:23-53.
14. Heimbach RD, Boerema I, Brummelkamp WH, Wolfe WG. Current therapy of gas gangrene. In: Davis JC, Hunt TK, editors. Hyperbaric oxygen therapy. Bethesda (MD): Undersea Medical Society Inc.;1977:153.