

Hyperbaric oxygen therapy: its use in medical emergencies and its development in Hong Kong

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ABSTRACT

Hyperbaric oxygen therapy is widely accepted as life-saving treatment for decompression illness. Yet its use in acute carbon monoxide poisoning has remained controversial because of inconsistent findings in clinical trials. Hyperbaric oxygen therapy has an adjunctive role in managing gas gangrene, necrotising soft-tissue infection, and crush injury, as supported by case series. Several cases have been reported in the literature detailing the use of hyperbaric oxygen therapy in patients with severe anaemia in whom blood transfusion is not possible. Today, use of hyperbaric oxygen therapy in Hong Kong is limited by low awareness among physicians and patients, a lack of service access, and inadequate hospital and critical care support for the existing non-hospital facility. The recent introduction of a hospital-based facility is expected to benefit more

patients for whom hyperbaric oxygen therapy is appropriate. This article reviews the mechanistic basis of and emerging scientific evidence to support the use of hyperbaric oxygen therapy in a number of acute medical emergencies, as well as the past and future development of hyperbaric oxygen therapy in Hong Kong.

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Introduction

Hyperbaric oxygen therapy (HBOT) is not a new treatment modality. Medical use of alterations in ambient pressure can be traced back to 1662, when Henshaw built the first hyperbaric chamber (Domicilium), a century before the discovery of oxygen.¹ The beneficial effects of increased pressure as therapy for decompression illness (DCI) became evident more than 100 years ago, leading subsequently to the discovery of a synergy between pressure and high oxygen levels that provides the physical and biological basis of what is now known as ‘hyperbaric oxygen therapy’. This therapy is now used in a wide range of medical conditions and hyperbaric medicine has emerged as a clinical discipline in many countries. Today, the use of HBOT in Hong Kong is still limited because of low awareness among physicians and patients, and lack of access to an HBOT facility, especially in the hospital setting. In this article, we review the mechanistic basis of and evidence supporting the use of HBOT in five selected medical emergencies, as well as the past and future development of HBOT service in Hong Kong.

Clinical use of hyperbaric oxygen therapy

Defined by the Undersea and Hyperbaric Medical Society (UHMS), HBOT is “an intervention in

which an individual breathes near 100% oxygen intermittently while inside a hyperbaric chamber that is pressurised to greater than sea level pressure (1 atmosphere absolute, 1 ATA).² The pressure must exceed 1.4 ATA for clinical purposes and its application must be systemic to the patient’s body—that is, topical application is not considered HBOT. Hyperbaric chambers are classified according to occupancy. Monoplace chambers are designed for a single person and generally pressurised with 100% oxygen. Multiplace chambers are intended for concurrent use by more than one patient and are pressurised with air, with oxygen given via a face-mask, hood tent, or endotracheal tube.

Whereas pressure per se has some therapeutic effect in bubble-related diseases, the biological essence of HBOT is extreme hyperoxia, enabled via increased pressure. Under pressure, the physical behaviour of gases is governed by gas laws; those that are fundamental to understanding HBOT are summarised in Table 1.³ Of note, HBOT has complex biological effects that extend beyond increasing the amount of dissolved oxygen. Over the years, different additional mechanisms and applications have been reported in the literature.

At present, the UHMS approves 14 clinical indications for HBOT (Box).² Different treatment protocols (known as treatment tables), consisting of different combinations of pressure and durations

高壓氧治療於急症的應用以及在本港的發展

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高壓氧治療於減壓症的應用已被醫學界廣泛接受。然而，這種治療在急性一氧化碳中毒的應用在臨床研究中療效不一致，所以仍存在爭議。一些病例研究顯示，高壓氧治療在處理氣性壞疽、壞死性筋膜炎和擠壓傷有輔助作用。文獻中亦有數個病例，詳細描述當嚴重貧血患者不能接受輸血時，可以使用高壓氧治療。在香港，高壓氧治療仍未普及，主因是醫生和病人對此療法認知不足，服務渠道缺乏，以及對現有非醫院設施的住院和重症監護支援不足。近來在醫院層面引入這服務設施讓更多適合接受高壓氧治療的病人受益。本文回顧了高壓氧治療的機制，總結這療法在數種急症應用的新科學證據，報導這種治療在本港過去的情況和展望未來的發展。

of oxygen and air breathing, have been devised for different conditions. The incidence of adverse effects is reported to be between 5 and 50 per 1000 HBO₂ exposures, depending on the indication, clinical setting, treatment protocol, and patient's

condition.⁴ The adverse effects of and contraindications to HBO₂ are summarised in Tables 2 and 3, respectively. Because of limited space, this article focuses on five acute medical conditions encountered in the emergency setting: DCI, carbon monoxide (CO) poisoning, acute infections, acute crush injuries, and severe anaemia. Readers are advised to refer to the relevant literature for clinical indications not covered in this article.

Decompression illness

Decompression illness is caused by an acute reduction in ambient pressure leading to formation of intravascular or extravascular gas bubbles. Gas embolism and decompression sickness (DCS) are the two major forms.

Gas embolism occurs when gas enters the arterial (arterial gas embolism [AGE]) or venous (venous gas embolism [VGE]) circulation. Diving and iatrogenic gas embolism are the two main causes.⁵ Diving embolism is precipitated by rapid ascent, breath-holding, or the presence of pre-existing lung pathology. Overexpansion of the lung during ascent causes barotrauma, alveolar or small airway rupture, and air entry into the pulmonary veins followed by air transit to the arterial circulation. Iatrogenic gas embolism can occur as a complication of a variety of invasive medical procedures, including central line placement, cardiopulmonary bypass, laparoscopic surgery, and a range of open surgical procedures.⁶ In general, VGE is relatively better tolerated because gas in the venous system is filtered by the pulmonary vessels. Venous gas, however, can migrate to the arterial system when there is right-to-left shunting (eg, through a patent foramen ovale) or pulmonary filtration overload (large amount of bubbles), causing 'paradoxical embolism'.⁷

In AGE, the gas bubbles can occlude any end artery, directly inducing distal tissue ischaemia. However, most AGE pathology probably accrues from damage to the endothelium, triggering a cascade of haemostatic and inflammatory responses, endo-

BOX. Indications for hyperbaric oxygen therapy approved by the Undersea and Hyperbaric Medical Society²

1. Air or gas embolism
2. Arterial insufficiencies—central retinal artery occlusion and enhancement of healing of selected problem wounds
3. Carbon monoxide poisoning
4. Clostridial myonecrosis (gas gangrene)
5. Compromised grafts and flaps
6. Crush injuries and skeletal muscle compartment syndromes
7. Decompression sickness
8. Delayed radiation injuries
9. Idiopathic sudden sensorineural hearing loss
10. Intracranial abscess
11. Necrotising soft-tissue infections
12. Refractory osteomyelitis
13. Severe anaemia
14. Thermal burns

TABLE I. Gas laws and their implications for hyperbaric oxygen therapy³

Gas law	Description	Implications
Boyle's Law	At a constant temperature of a fixed mass of gas, the volume of a gas is inversely proportional to its absolute pressure.	It governs the change in the volume of gas bubbles and air space in the body during decompression and recompression, as well as air-filled components of therapeutic devices, such as endotracheal cuffs and pressure bag.
Gay-Lussac's Law	At a constant volume, the absolute pressure of a given mass of gas is directly proportional to the absolute temperature.	In a fixed-volume chamber, the ambient temperature increases during compression and decreases during decompression.
Dalton's Law	In a mixture of ideal gases, the total pressure is equal to the sum of the partial pressures of the component gases.	This is the basis for increased amount of dissolved gas in blood and tissue when the ambient pressure increases.
Henry's Law	At a constant temperature, the amount of gas dissolved in a liquid is directly proportional to the partial pressure of that gas in equilibrium with that liquid.	—

TABLE 2. Adverse effects of hyperbaric oxygen therapy

Type of effects	Adverse effects
Effects caused by barometric pressure change	<ol style="list-style-type: none"> 1. Middle-ear barotrauma (myringotomy may be needed) 2. Sinus barotrauma 3. Lung barotrauma—haemoptysis, pneumothorax, pneumomediastinum, subcutaneous emphysema, or arterial gas embolism 4. Gas leakage or rupture of air-containing cavity in the body
Effects caused by oxygen toxicity	<ol style="list-style-type: none"> 1. Neurological—muscle twitching, dysphoria, nausea and vomiting, convulsion 2. Pulmonary—increase in the work of breathing due to increase in gas density, tracheobronchitis (cough, dyspnoea, and chest tightness), deterioration in lung function 3. Ocular—reversible tunnel vision, temporary myopia, cataract after prolonged treatment

thelial leak and vasogenic oedema, and ischaemia-reperfusion injury.^{5,6} Clinical manifestations, usually sudden in onset (or for divers, within a few minutes of surfacing), depend on the location of the gas embolus and the quantity of gas. Of note, AGE can result in severe morbidity or even death if it involves coronary or cerebral arteries. Coronary artery emboli can lead to myocardial ischaemia, cardiac failure, dysrhythmia, or even cardiac arrest. Cerebral AGE can present as a stroke with focal neurological deficits, loss of consciousness, seizure, or even coma.

Decompression sickness occurs when the rate of ambient pressure reduction exceeds that of inert gas (mainly nitrogen) washout from tissue. When a diver ascends following a period of time underwater, the partial pressure of dissolved inert gas in capillaries and tissues is greater than the ambient pressure (Henry’s Law and Dalton’s Law) and off-gassing occurs. If supersaturation results, bubbles form in venous blood and/or tissues. The reported threshold dive depth for DCS is about 6 m but problems arise only after very prolonged times at such shallow pressures.⁸ Such sickness is very uncommon after diving to depths of less than 10 m. The risk is also affected by multiple factors such as immersion (vs dry hyperbaric chamber exposure), exercise, and temperature. In DCS, extravascular (autochthonous) bubbles cause mechanical distortion of tissues, leading to pain or dysfunction, depending on the tissue involved; intravascular bubbles cause a VGE that can arterialise.⁹ Decompression sickness has a wide range of potential manifestations that begin minutes to days after surfacing, including constitutional symptoms such as malaise; joint pain that commonly involves the knees and characteristically improves on local tissue pressure; mild neurological symptoms, such as numbness or paraesthesia; and skin rash (livedo reticularis). Severe cases can present with cardiopulmonary collapse, loss of consciousness, incomplete or complete spinal cord paresis, or severe vestibular dysfunction.

Diagnosis of DCI is primarily based on clinical findings. Clinicians should be aware of the

TABLE 3. Contra-indications to hyperbaric oxygen therapy

Type	Conditions
Absolute	<ol style="list-style-type: none"> 1. Untreated pneumothorax 2. Acute severe bronchospasm 3. Concomitant treatment with doxorubicin 4. Concomitant or recent treatment with bleomycin
Relative	<ol style="list-style-type: none"> 1. Upper respiratory tract infection 2. Allergic rhinitis 3. Chronic sinusitis and otitis 4. Chronic obstructive pulmonary disease with emphysema 5. History of pneumothorax or thoracic surgery 6. History of ear, nose, and throat surgery 7. Epilepsy 8. Optic neuritis 9. Uncontrolled hypertension 10. Uncontrolled heart failure 11. Claustrophobia 12. Dangerous behaviour

possibility of AGE or DCS when patients present with compatible symptoms and a history of recent diving. The possibility of AGE should be considered in any case of sudden-onset clinical deterioration after high-risk medical procedures. Differentiation between AGE and DCS in divers may be difficult but is not necessary when selecting patients for recompression therapy.⁹

Supportive treatment is the mainstay of pre-hospital and initial emergency treatment for DCI. High-flow oxygen is used to correct hypoxia and to create a diffusion gradient from tissue to alveolar gas for the egress of nitrogen and other gases from the bubbles. For both AGE and DCS, recompression with HBOT is widely accepted as the definitive and potentially life-saving treatment despite a lack of randomised controlled trials (RCTs) in humans. Its use is supported by more than 100 years of clinical experience, rigorous mechanistic and outcomes-based research in animal models and human volunteers. The initial response to therapeutic pressurisation is in accordance with Boyle’s Law—at 2.8 ATA pressure; bubble volume is immediately reduced by two-thirds.¹⁰ Hyperoxia corrects tissue hypoxia and, by minimising blood

nitrogen, maximises the diffusion gradient from the embolised gas to circulating plasma, thus optimising off-gassing. Furthermore, HBOT has anti-oedema and anti-inflammatory effects in acute injury, in particular inhibiting neutrophil adhesion to blood vessels, thus reducing reperfusion injury. The therapy is associated with significant improvement in the majority of patients with AGE.^{9,11,12} Of note, HBOT should be initiated early once the patient is stabilised—the UHMS recommends 100% oxygen at 2.8 ATA, with treatment repeated until symptoms completely resolve or there is no further improvement, typically after no more than five to ten treatments.^{2,13}

Acute carbon monoxide poisoning

Hyperbaric oxygen therapy has been used in a variety of acute poisoning settings, including those caused by CO, methylene chloride, hydrogen sulphide, and carbon tetrachloride; gas embolism resulting from hydrogen peroxide ingestion; and methaemoglobinaemia.¹⁴ This article focuses on

CO poisoning, as it remains a major cause of non-medicinal poisoning death¹⁵ and often results in persistent or delayed neurological sequelae.¹⁶

The pathophysiology of CO poisoning is complex and readers are referred to excellent reviews by Weaver¹⁶ and Roderique et al¹⁷ for details. In brief, CO causes tissue hypoxia by forming carboxyhaemoglobin (COHb) and shifting the oxyhaemoglobin dissociation curve to the left. It also binds to various haem proteins, impairs mitochondrial function, causes release of nitric oxide and free radicals, and triggers inflammation through a myriad of mechanisms independent of hypoxia.¹⁶⁻¹⁹

Oxygen therapy is the standard treatment. It works by reversing hypoxia, competing with CO for haemoglobin binding, and shortening the half-life of COHb (from 320 min in room air to about 70 min with 100% oxygen at 1 ATA); HBOT further reduces its half-life to 20 min (100% at 2.5 ATA), and increases the amount of dissolved oxygen in the plasma.²⁰ Recent studies have shown that HBOT also restores mitochondrial function,²¹

TABLE 4. Summary of randomised controlled trials comparing hyperbaric oxygen and normobaric oxygen for carbon monoxide poisoning

Study	Sample size	Study design	Intervention	Key results	Issues
Raphael et al, ²⁷ 1989	629	RCT, unblinded	If no LOC, 2-h HBOT (2.0 ATA) + 4-h NBO vs 6-h NBO; if LOC, 1 HBOT session vs 2 HBOT sessions	No difference in recovery between groups at 1 month	Lack of objective assessment of neurological sequelae; many cases received HBOT >6 h after poisoning; suboptimal pressure (2 ATA)
Thom et al, ²⁸ 1995	65	RCT, unblinded	HBOT (2.8 ATA for 30 mins, then 2 ATA O ₂ for 90 mins) vs NBO till symptom resolution (mean, 4.2 h)	No DNS in HBOT group vs 23% in NBO group (P<0.05); NNT=4.3	Mild-to-moderate CO poisoning presented within 6 h; excluded LOC or cardiac compromise; small sample size; lack of sample size calculation
Mathieu et al, ²⁹ 1996	575	RCT, unblinded	HBOT (2.5 ATA) for 90 mins vs 12-h NBO	Significant difference in neurological symptoms at 3 months (HBOT: 9% vs NBO: 15%) but not at 1 month, 6 months, or 12 months	Abstract only; unclear bias in randomisation, allocation concealment and selective reporting; no report of dropouts
Scheinkestel et al, ³⁰ 1999	191	RCT, double-blind sham therapy	3 Daily 1-h HBOT sessions (2.8 ATA) vs 3 days of NBO (100% O ₂ at 1 ATA sham dives). Both groups received continuous high-flow O ₂ for 3 days. 3 Additional courses of original treatment for patients with 'poor outcome'	HBOT group had a significantly worse outcome in the learning test; DNS restricted to HBOT group	Large number of suicide attempts; delayed HBOT for >6 h; high lost-to-follow-up rate; continuous high-flow O ₂ not accepted as standard practice
Weaver et al, ³¹ 2002	152	RCT, double-blind sham therapy	3 HBOT sessions (3 ATA for 1 h, then 2 ATA for 1 h in session 1, 2 ATA for 2 h in sessions 2 and 3) in 24-h vs 3-sham chamber sessions (100% O ₂ at 1 ATA in session 1 and normal air at 1 ATA in sessions 2 and 3)	Less-frequent cognitive sequelae (25% vs 46%) at 6 weeks, 6 months, and 12 months	Excluded if >24 h after CO exposure; NBO group had a higher prevalence of cerebellar signs at baseline; trial stopped after the third interim analysis; apparent change in the primary outcome
Annane et al, ³² 2011	385	RCT, unblinded	Trial A (n=179; patients with transient LOC): 1 HBOT session (2.0 ATA) + 4-h NBO vs 6-h NBO Trial B (n=170; patients with initial coma): 2 HBOT sessions + 4-h NBO vs 1 HBOT session + 4-h NBO	Trial A: no difference in 'complete recovery' at 1 month (58% vs 61%) Trial B: 'complete recovery' rate 47% with 2 HBOT sessions vs 68% with 1 HBOT sessions at 1 month	HBOT at 2 ATA only; excluded suicide attempts and non-domestic CO poisoning; lack of objective assessment of neurological sequelae; outcome assessment at 1 month only; premature trial termination because of harm and fertility in the interim analysis

Abbreviations: ATA = atmosphere absolute; CO = carbon monoxide; DNS = delayed neurological sequelae; HBOT = hyperbaric oxygen therapy; LOC = loss of consciousness; NBO = normobaric oxygen; NNT = number needed to treat; O₂ = oxygen; RCT = randomised controlled trial

reduces brain lipid peroxidation,²² and inhibits the CO-induced inflammatory response by inhibiting $\beta 2$ integrin-mediated neutrophil adhesion to brain microvasculature and by inhibiting lymphocyte sensitisation to myelin basic protein.²³⁻²⁵

Hyperbaric oxygen therapy was first used for CO poisoning in 1960²⁶ but has remained controversial owing to the conflicting results of RCTs of its effect on delayed neurological sequelae. These are summarised in Table 4.²⁷⁻³² A Cochrane review in 2011 that involved six RCTs and 1361 participants showed that HBOT does not have a significant benefit in a pooled random-effects meta-analysis (odds ratio for neurological deficits, 0.78; 95% confidence interval, 0.54-1.12). The reviewers cautioned that the “significant methodologic and statistical heterogeneity” and “design or analysis flaws” of the included trials warrant cautious interpretation of the results.³³ The American College of Emergency Physicians, on the basis of a systematic literature review, stated that “It remains unclear whether [hyperbaric oxygen therapy] is superior to normobaric oxygen therapy for improving long-term neurocognitive outcomes” in CO-poisoned patients.³⁴ Nonetheless, a recent population-based retrospective cohort study in Taiwan involving 7278 patients showed that HBOT was associated with reduced mortality in patients with CO poisoning after adjusting for covariates, especially in those who were younger than 20 years and those with acute respiratory failure.³⁵ These findings add weight to the argument in support of HBOT use in CO poisoning.

Yet, the threshold for HBOT use for CO poisoning varies across different centres³⁶ and uncertainties exist regarding the optimal chamber pressure, number and frequency of sessions, and time window after CO poisoning. In particular, pregnant women pose special challenges as they are at high risk of adverse effects from both CO and HBOT, although information is lacking because they are excluded from most prospective trials. In view of the devastating fetal outcomes of maternal CO poisoning, such as stillbirth and damage to and anatomic malformation of the fetal central nervous system, COHb thresholds for HBOT are often set lower for pregnant patients (COHb, 15%-20%) and HBOT is often considered indicated when there is evidence of fetal distress.¹⁴ Clinical experience in Russia supports the contention that HBOT is safe during pregnancy,^{37,38} but its benefit in averting CO-related adverse fetal outcomes is unclear.¹⁴ In CO-poisoned children, indications for HBOT are similar to those for adults, although they have not been evaluated systematically.³⁹ Studies have reported that HBOT has been used safely in paediatric patients^{39,40} but there are special paediatric considerations. Readers are referred to the review by Liebelt⁴¹ for further information.

Before more convincing evidence is available, clinicians are advised to weigh the benefits, risks, and costs of HBOT carefully on a case-by-case basis when making a decision about HBOT for CO-poisoned patients. It is commonly suggested that HBOT should be reserved for (1) situations where there are indicators of higher-severity CO poisoning, such as loss of consciousness, abnormal neurological signs, cardiovascular dysfunction, or severe acidosis; (2) patients older than 35 years; (3) prolonged exposure (eg, >24 hours) or high COHb level (eg, $\geq 25\%$ ¹⁶). The most recent UHMS guidelines nevertheless recommend that HBOT be considered for all cases of acute symptomatic CO poisoning, given the lack of predictive factors for poor long-term outcomes or for which patients might receive the greatest benefit from HBOT.²

Acute infections

Hyperbaric oxygen therapy is directly bactericidal and/or bacteriostatic to anaerobes, facultative anaerobes, and many aerobes as a result of bacterial intolerance of the excess oxygen radicals induced by HBOT. The therapy improves tissue oxygenation, maximises oxygen-dependent phagocytic function, reduces tissue oedema, and potentiates uptake and/or action of various antimicrobial drugs, including the aminoglycosides, fluoroquinolones, and vancomycin.⁴² The therapy has been used as an adjunct in a variety of life-threatening bacterial infections—in particular, those with associated tissue necrosis such as gas gangrene, necrotising fasciitis, and other necrotising soft tissue infections (NSTIs).

Gas gangrene is a rare but fulminant infection that is most commonly caused by *Clostridium perfringens* and germinates in devitalised and hypoxic tissue. The lethal α -toxin produced by the organism causes rapidly progressing liquefactive myonecrosis. Hyperbaric oxygen therapy is bactericidal to *C perfringens*⁴³ and inhibits toxin production.⁴⁴ Experimental studies and case series support the use of HBOT as an adjunct to surgery and antibiotic therapy for gas gangrene.⁴² Three sessions of HBOT at 3 ATA for 90 min should be given in the first 24 h, followed by twice-daily treatments for the next 2 to 5 days, until infection control is achieved.^{2,45}

Other forms of NSTI, often polymicrobial, are often both life- and limb-threatening. Early HBOT adjunctive to surgery and antibiotic therapy has been associated with improved survival and limb salvage,⁴⁶⁻⁴⁸ although several other small case series have suggested no benefit.^{49,50} In a large case series that involved 1583 NSTI cases in 14 US centres with their own facilities, HBOT was associated with increased survival and fewer complications in the sickest group of patients.⁵¹ In heterogeneous, high-acuity, and relatively uncommon conditions

like NSTI, where there is no RCT support for any particular therapeutic strategy and none is likely, clinicians must base their decision-making on evidence from observational studies supported by interpretation of known pathophysiology and therapeutic mechanisms, as well as from any relevant animal data.⁵² Hyperbaric oxygen therapy is mechanistically attractive and supported by what appear to be good outcomes from experienced centres that routinely use HBOT. It should be considered in patients with serious NSTI, provided that referral for such treatment does not defer aggressive surgery and antibiotic therapy. The recommended hyperbaric oxygen protocol is 2.0 to 2.5 ATA for 90 min twice daily until the infection is controlled.^{2,45}

In addition to necrotising bacterial infections, there are reports supporting HBOT use in treating intracranial abscess, actinomycosis, and mucormycosis in immunocompromised patients.⁴⁵ Diabetic foot infections, refractory osteomyelitis, and certain implant infections are also important indications for HBOT, but they are outside the scope of this article.

Acute crush injuries and severe anaemia

Crush injury is a spectrum of injury ranging from minor contusions to limb-threatening damage. The energy of trauma can cause damage to multiple tissues. Damage to the microvasculature causes self-perpetuating fluid transudation, tissue oedema, interstitial bleeding, stasis, tissue hypoperfusion, and hypoxia. Compartment syndrome occurs when the tissue fluid pressure within a skeletal muscle compartment exceeds the capillary perfusion pressure to the muscle and nerves in the compartment. Hyperbaric oxygen therapy works by interrupting the oedema-ischaemia vicious cycle. It induces inflow vasoconstriction and reduces tissue oedema, thus improving microcirculatory blood flow. It also improves tissue oxygen delivery, which is essential in multiple oxygen-dependent host responses to trauma and infection, mitigates reperfusion injury, and enhances wound healing.⁵³

The use of HBOT in crush injury is supported by one small RCT that showed the effectiveness of HBOT in improving wound healing and reducing repetitive surgery, especially in older patients with Gustillo grade III soft-tissue injuries.⁵⁴ A systematic review of nine studies involving 150 patients with crush injury showed that HBOT is likely to be beneficial if administered early.⁵⁵ A larger and more rigorous RCT on open tibial fractures with severe associated soft-tissue injury will be published in the near future.⁵⁶ For compartment syndrome, no RCT has been published, but the use of HBOT is supported by animal studies and small case series.⁵³ The treatment regimen varies depending on the type

of injury, ranging from 2 to 2.4 ATA for 90 min for two or more treatments a day to 120 min for a single daily treatment.^{2,53}

There are situations in which blood transfusion is not possible for major blood loss owing to religious or practical reasons. Hyperbaric oxygen therapy can compensate for haemoglobin deficiency by increasing the amount of dissolved oxygen in the plasma to a level sufficient to maintain tissue oxygen delivery, even in the total absence of red blood cells. Prolonged, continuous HBOT cannot, however, be used to maintain life for the multiple days necessary for autologous replacement of red blood cells, as pulmonary oxygen toxicity becomes an intolerable and eventually fatal side-effect. Literature reports suggest the potential to use intermittent HBOT as a short-term measure to relieve hypoxic symptoms in patients with otherwise intolerably low haemoglobin levels while waiting for red blood cells to regenerate or in patients with limited compatible donor options while waiting for compatible blood products to be delivered.⁵⁷⁻⁶⁰

Past and future development of a hyperbaric oxygen therapy service in Hong Kong

The year 1994 witnessed a major development of HBOT in Hong Kong. Previously, HBOT was provided by the British Royal Navy at the HMS Tamar base and was confined to diving-related conditions. The Recompression Treatment Centre was commissioned in 1994 by the Hong Kong Government on Stonecutters Island and has become the major HBOT provider since then. The facility, comprising a three-compartment multiplace chamber, is managed by the Fire Services Department under the medical supervision of the Occupational Medicine Division of the Department of Health. Nonetheless, it is primarily used during diver training by the Fire Services Department. Medical use is mainly for emergency DCI treatment and CO poisoning cases referred from public hospitals, and many elective sessions are devoted to radionecrosis treatment.⁶¹ Its remote location and lack of back-up critical care facilities, however, render it unsuitable for critically ill patients because of the risks inherent in patient transportation. Furthermore, lack of properly trained local hyperbaric physicians and expertise as well as a lack of human resources and training opportunities hinder the development of HBOT in Hong Kong. Low awareness among physicians and patients makes the referral for this treatment even less frequent. Although HBOT is also provided by a private centre in Hong Kong, patient access remains very limited.

One of the most important questions to address in developing an HBOT service in Hong

Kong is its service need in our locality. Current data from the Recompression Treatment Centre (200-300 sessions for 20-30 patients per year) offer limited insight since access to the service is limited. To assess the need accurately, it would be necessary to estimate the number of patients in Hong Kong in whom HBOT is indicated and the proportion of persons with each condition who might benefit from HBOT. Doing so would depend on multiple factors including considering alternative treatments available for each condition, cost and financing, and doctor and patient beliefs and acceptance. Unfortunately, treatment thresholds for each of the many possible indications are not widely agreed on, or even researched, and the appropriate criteria for referral vary widely in practice, between nations and even between locations within nations. It must also be acknowledged that the referral rates for HBOT are strongly influenced by its availability, integration into the health care system, and the reputation of individual facilities and their clinical leaders.

One way of estimating the need for HBOT service is to study the data at health system level on the number of chambers and activity level in other countries. Internationally, Australia offers a good example. There, the eight major HBOT facilities are evenly distributed, with one major government hospital-based facility in each of its seven states and territory capital cities, excluding Canberra, plus one in Townsville, the major infrastructure city serving North Queensland and the Great Barrier Reef tourism zone. In addition, there are four active private facilities and several more being planned. Each of the major hospital-based facilities operates a large multiplace chamber, most commonly of 'triple lock' (three compartment) design, that can be configured to provide critical care as well as ambulatory care. Most facilities also operate one or more monoplace chambers to provide flexibility and allow efficient staff utilisation.

All government hospital-based facilities in Australia are integrated with major academic tertiary hospitals. The indications and thresholds for HBOT in Australia are conservative by international standards and can therefore be seen as a good guide to what would be a reasonable aim for Hong Kong. With 12 facilities serving a population of approximately 24 million in Australia, each facility provides 2000 to 5000 treatment sessions every year. While emergency patients receive one to several treatment sessions, non-emergency patients typically receive 20 to 40 treatment sessions spanning 4 to 8 weeks. The workload is in the range of 2000 to 5000 HBOT sessions for 100 to 350 patients per centre per year, with two to three scheduled 2-h sessions per day and six to ten non-emergency patients per scheduled session.

Hong Kong has a population of 7 million and

there exists a need for at least one HBOT hospital-based facility, especially for critically ill patients. In 2010, a task force was set up in the Hospital Authority to review the development of HBOT. Approval was finally given in 2014 to establish the first hospital-based HBOT centre at the Pamela Youde Nethersole Eastern Hospital. This new centre will be managed by the Accident and Emergency Department in collaboration with the Intensive Care Unit. The facility has been designed to be close to the resuscitation room of the Accident and Emergency Department and the service will be operationally supported by the Intensive Care Unit. With the establishment of the first public hospital-based HBOT, it is expected that it will be possible to offer additional treatment options for conditions where HBOT is indicated. For instance, DCI and CO-poisoned patients can be referred to the Pamela Youde Nethersole Eastern Hospital for screening for suitability for HBOT and management. Life-threatening infections, such as gas gangrene and NSTI, and limb-threatening crush injuries can be managed with HBOT as an adjunct to conventional therapy. It is also expected that HBOT will be made available to many patients with chronic conditions such as non-healing diabetic wounds, compromised skin flaps and grafts, and radionecrosis in the out-patient setting. The hospital-based HBOT centre will also provide more opportunity for local training and research.

Conclusion

With the opening of the first hospital-based HBOT centre in Hong Kong in 2018, management of conditions such as DCI, CO poisoning, NSTI, and acute crush injuries may change dramatically, in terms of treatment choices as well as the logistics of patient transfer between hospitals. Involvement of emergency physicians as facilitators, modulators, and coordinators in this treatment will also widen the scope of HBOT application and strengthen collaboration with other disciplines.

Declaration

The authors have no conflicts of interest to disclose.

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