ORIGINAL PAPERS

IMPLICATIONS OF HYPERBARIC MEDICINE FOR ANAESTHESIA AND INTENSIVE CARE PART 1

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Summary

Hyperbaric medicine is becoming increasingly accepted as an important adjunctive therapy for many diseases. There are important considerations for anaesthesia and intensive care when interfacing with hyperbaric medicine. These include awareness of the indications for hyperbaric oxygen (HBO), physiological changes associated with HBO, potential complications and drug interactions. Awareness of these considerations will aid in the safe management of patients across these specialties.

Key Words

Anaesthesia, equipment, hyperbaric facilities, hyperbaric oxygen, hyperbaric research, medical conditions and problems, physiology, treatment and ventilators.

Background

Man's exposure to hyperbaric environments (breath hold diving) dates from at least 4,500 BC, and it is known that breath hold diving for sponge was a common and lucrative profession in Ancient Greece. Alexander the Great was reported to have descended in a glass diving vessel in 332 BC during the Battle of Tyre, one of the earliest recorded diving bells.[1] However the deliberate exposure to pressure for medicinal, non-diving purposes, was first described by Henshaw in 1664, when he constructed his *Domicilium*. This English clergyman and physician constructed a pressure vessel which could produce both increased (hyperbaric) or decreased (hypobaric) pressure to treat a variety of maladies using air as the breathing medium. Despite a belief that hypobaric exposure "cured" chronic diseases whilst hyperbaric pressure was better for acute diseases, scientific evidence was lacking.

Hyperbaric medicine's first contact with surgery and anaesthesia came with Fontaine's development of a mobile hyperbaric operating room.[2] Twenty-seven procedures were performed under moderate hyperbaric conditions using nitrous oxide as the sole anaesthetic agent. In addition the "normal" postoperative cyanosis was not seen due to the slightly higher partial pressure of oxygen in air at 1.25-1.3 atmospheres absolute (ATA). The diving equivalent of 1 ATA is 1 bar and the SI equivalent is 101 kPa. The father of pressure physiology, Frenchman Paul Bert, further developed hyperbaric nitrous oxide anaesthesia in the late 19th century, describing this in the treatise *La Pression Barometrique*.[3]

Hyperbaric air therapy continued to spread throughout Europe, Canada and the US as a panacea for a wide variety of illnesses. The complete loss of confidence in this type of treatment came with the public denigration of hyperbaric medicine in the North American medical community. Orville J. Cunningham, from Kansas City, used a hyperbaric chamber to treat the victims of the Spanish 'flu epidemic during World War I.[4] While its value in providing increased systemic oxygenation in pneumonias may have had some scientific basis, subsequent treatment for diabetes, hypertension, syphilis and cancers was at best naive and at worst fraudulent. Indeed, Cunningham postulated that cancers, diabetes and some arthritides were due to anaerobic micoorganisms, hence the efficacy of hyperbaric air. A grateful patient, who owned a large bearings firm, constructed a six storey, seventy-two room spherical hyperbaric chamber, complete with carpets and grand piano. Lack of scientific data to prove efficacy and minimal exposure to the greater medical community led an American Medical Association investigation bureau to conclude that hyperbaric medicine was "tinctured much more strongly with economics than with scientific medicine.[5] A hiatus remained in hyperbaric medicine until the middle of the 20th century.

Although Priestley discovered oxygen in 1776, it was not until 1937 that Benkhe and Shaw used hyperbaric oxygen (HBO), rather than air, to treat decompression illness (DCI). In the subsequent 30 years, carbon monoxide poisoning,[6] radiotherapy,[7] clostridial soft tissue infections[8] and osteomyelitis[9] were all treated with HBO. Once again surgeons were at the forefront in the development of hyperbaric medicine. Boerema, a Dutch cardiac surgeon, demonstrated that pigs which were exsanguinated, and had their blood volume replaced with saline to a haematocrit of 4%, could live with no problems for up to 15 minutes when subjected to 100% oxygen at 3 |atmospheres.[10] In the days prior to cardiopulmonary bypass, Boerema was able to perform complex cardiac operations, e.g. repair of Tetralogy of Fallot, under circulatory arrest and hypothermia. It was not clear however whether the real benefit from HBO was gained during the period of circulatory standstill, prolonging arrest time, or from enhancing oxygenation during the post-reperfusion phase.

As a direct result of Boerema's work, Professor Johnstone initiated the building of a hyperbaric facility at Prince Henry Hospital in Sydney in 1964. In an era of rapidly developing technology, extracorporeal oxygenation,

developed by Gibbon in 1953 and in wider use by the mid 60s, replaced HBO for cardiac operations. The requirement for surgery in a hyperbaric operating suite was soon relegated to that of historical interest only. Yet pioneering work by Dr Ian Unsworth allowed the unit to continue with new direction. The first recorded hyperbaric exposure in an Australian hospital occurred on 13th July 1970 at Prince Henry Hospital, with patient treatments taking place in early 1971 for carbon monoxide intoxication and gas gangrene. Over the next 25 years, formal units, mostly multiplace chambers, were formed in most Australian capital cities. New Zealand also developed hyperbaric centres on both North and South Islands.

TABLE 1

AUSTRALIAN AND NEW ZEALAND HOSPITAL HYPERBARIC MEDICINE UNITS IN 1996

State or Island	Town	Hospital	Address
South Australia	Adelaide	Royal Adelaide Hospital	North Terrace, Adelaide 5000
Northern Territory	Darwin	Royal Darwin Hospital	Rocklands Drive, Tiwi 0810
Western Australia	Fremantle	Fremantle Hospital	PO Box 480, Fremantle 6160
Tasmania	Hobart	Royal Hobart Hospital	PO Box 1061, Hobart 7001
Victoria	Melbourne	Alfred Hospital	Commercial Road, Prahran 3181
New South Wales	Sydney	Prince of Wales Hospital	High Street, Randwick 2031
New South Wales	Sydney	HMAS Penguin	RCC Facility, Balmoral 2088
Queensland	Townsville	Townsville General Hospital	Eyre St Townsville 4810
North Island New Zealand	Auckland	HMNZS Philomel	Naval Base, Devonport, Auckland
South Island New Zealand	Christchurch	Christchurch Hospital	Private Bag 4710, Christchurch

In Australia and New Zealand Specialist training in hyperbaric medicine is at present not structured. Historically, doctors with experience in diving and military medicine were at the forefront of hyperbaric medicine. Over the past years the majority of hospital hyperbaric facilities have been staffed by specialists in anaesthesia, intensive care or occupational and emergency medicine who have a sideline interest in hyperbaric medicine. Recently, independent units, with full time staffing, have allowed hyperbaric medicine to evolve into a distinct medical speciality; yet further work is needed to enhance the profile of hyperbaric medicine in the medical community. A recent review of physicians at a large United States teaching hospital showed that the majority of doctors never received any training in hyperbaric medicine in their undergraduate training. More importantly, these doctors may have had patients in their specialty who could have benefited from HBO treatment.[11] The Australian and New Zealand College of Anaesthetists (ANZCA) has approved a provisional fellow position in hyperbaric medicine to fulfil FANZCA requirements at the Royal Adelaide Hospital. Such positions have also been granted from the College of Emergency Medicine to Fremantle Hospital and the Royal Australasian College of Physicians at Alfred Hospital, Melbourne. Additionally, special interest group status is in the process of being sought from ANZCA.

Hyperbaric Medicine is not at present a registrable speciality in Australia, unlike in North America and Europe. The Diploma in Diving and Hyperbaric Medicine (DipDHM) is currently granted to doctors who have completed and passed a two week course in Diving and Hyperbaric Medicine, worked in a recognised Hyperbaric Unit for the equivalent of six months full time and submitted a thesis on one aspect of diving or hyperbaric medicine. This is submitted to SPUMS (South Pacific Underwater Medicine Society) for review by assessors. There are plans by the ANZHMG (Australian and New Zealand Hyperbaric Medicine Group) to develop a formal training program for hyperbaric medicine, but this is some time away. The growth of this field is reflected by the increasing number of national and international meetings devoted to hyperbaric medicine, along with national and international societies devoted to development and importantly controlled research into this area (Table 2). Hopefully the days of Orville J. Cunningham will never reappear!

Indications for HBO Treatment

An important rationalisation of the use of HBO came in the report of the committee on hyperbaric oxygenation.[12] This was a consultative document with the major US medical insurance companies (Blue Cross/Blue Shield) and Social Security (Medicare) along with an executive committee from the Undersea and Hyperbaric Medicine Society (UHMS). A list of those diseases which had sound basis for HBO treatment and those which were

TABLE 2

HYPERBARIC MEDICAL AND TECHNICAL ASSOCIATIONS
(in alphabetical order)

Organisations	Countries covered
Associacion Mexicana de Medicina Hiperbarica y Subacuatca, AC (AHMS)	Mexico and Central America
Australia and New Zealand Hyperbaric Medicine Group (ANZHMG)	Australia and New Zealand
Baromedical Nurses Association (BNA)	North America
European Undersea and Biomedical Society (EUBS)	Europe
Hyperbaric Technicians and Nurses Association (HTNA)	Australia and New Zealand
International Congress on Hyperbaric Medicine	International
Japanese Society for Hyperbaric Medicine (JSHM)	Japan
Societe de Physiologie et de Medicine Subacuatiques et Hyperbares de Langue Francaise (MEDSUBHYP)	France
South African Underwater and Hyperbaric Medical Association	South Africa
Undersea and Hyperbaric Medical Society (UHMS)	North America

investigative was formulated. These indications are under continual review and form the basis of practice in the majority of hyperbaric units in Australia and New Zealand.

Conditions commonly treated by HBO include; decompression illness, acute carbon monoxide (CO) poisoning, chronic osteomyelitis, osteoradionecrosis, problem wound healing (e.g. diabetic and/or arteriosclerotic wounds) and necrotising soft tissue infections.[12] There are other indications which would be considered as having considerable potential benefit, such as crush and reperfusion injury and compromised skin flaps. Finally there are occasions when HBO may be used in exceptional circumstances, or which are currently under study (e.g. thermal burns, exceptional blood loss anaemia (Jehovah's witnesses) or soft tissue sporting injuries).

TABLE 3

BENEFICIAL EFFECTS OF HYPERBARIC OXYGEN THERAPY

A **Reduction in bubble size**

B **Hyperoxygenation**
Vasoconstriction
Angiogenesis
Antibacterial (direct and indirect mechanisms, inhibition of toxin production and deactivation of toxins)
Osteoclastic stimulation
Fibroblast stimulation

Physiology of hyperbaric oxygen therapy

HBO is thought to exert beneficial effects through a variety of mechanisms (Table 3). Knowledge of the physiological responses to HBO are essential when dealing with patients who are critically ill, elderly or have significant cardiorespiratory disease. Key questions that need to be asked before any treatment include; can HBO benefit the patient and can it be associated with adverse physiological events? The answers to these questions can alter both anaesthetic and intensive care management of patients undergoing HBO therapy.

HEART RATE RESPONSE

Bradycardia is commonly seen during HBO treatment. Possible mechanisms include; direct pressure effect on pacemaker function, hyperoxia itself, increased work of breathing with dense gases or the effects of dissolved inert gases.[13] Bradycardia is also seen when 100% oxygen is breathed at normal (surface) pressure (1 ATA, 1 bar, 101 kPa).[14] Örnhagen studied hyperbaric exposure of isolated sinus node preparations from mouse, rat, guinea pig, rabbit and dog hearts.[13] All species showed a direct pressure related (up to 150 bar, 15,000 kPa) reduction in beating frequency in isolated pacemaker cells, which was not modulated by adrenergic or cholinergic agents. Studies in intact animal models at lower pressures suggest that this bradycardia may, however be mediated via vagal stimulation, baroreceptor activation following vasoconstriction and increased mean arterial pressure, or as a direct effect on chemoreceptors.[15,16,17,18] Over the course of a hyperbaric exposure the initial bradycardia will become less, but does not tend to return to baseline levels until the treatment is completed.[15] Unexplained tachycardia or even normalisation of heart rate have been

reported in the convulsive and pre-convulsive periods of central nervous system (CNS) oxygen toxicity.[19] In exposures up to pressures of 71 bar (7,100 kPa), which are not used clinically, a pressure induced bradycardia and reductions in P and T wave amplitudes were seen;[20] other conduction changes have been seen in commercial saturation divers.[21]

CARDIAC OUTPUT

Cardiac output has been shown to be reduced when breathing 100% oxygen at surface pressure (1 bar, 100 kPa) and during hyperoxic hyperbaric exposures.[14,23-27] This reduction in cardiac output may be oxygen tension dependent rather than due to the effect of pressure per se. Normoxic exposure to pressures up to 6 bar (600 kPa) using mixtures of helium, oxygen and nitrogen failed to demonstrate any reduction in cardiac output, contrasting with the hyperoxic data.[28]

Cardiac output is determined mainly by heart rate, preload, contractility and afterload. The heart rate response to HBO is usually bradycardia. HBO may affect preload as shown by an increase in haematocrit (Hct), possibly secondary to accumulation of interstitial transudate.[29,30] This may be mediated by the release of adrenalin, atrial natiuretic peptide and endothelin causing an increase in vascular permeability and leakage of fluid and albumin.[31] An increase in Hct of over 40% in a 6 hour study at 3 bar (300 kPa) was demonstrated by Amin,[30] an effect which may be increased in patients who have other causes of fluid loss, e.g. sepsis, burns, unhumidified ventilation, nausea, vomiting or impaired consciousness. Such haemoconcentration needs to be considered when assessing fluid requirements in the peri-treatment period. It can be worsened by repeated fasting for surgical debridements and interruptions of fluid administration during transportation.

Myocardial contractility has been shown to increase[32] or decrease[33,34] during HBO therapy. Myocardial contractility and left ventricular pressure have been shown to increase during exposures to 5 bar (500 kPa), even when the partial pressure of oxygen was maintained at the same level as before compression.[35]

There are few human studies which examine the haemodynamic responses to HBO at clinically relevant pressures. Pisarello[36] noted in a volunteer study that cardiac output decreased significantly during continuous oxygen exposure of 2-3 bar (200-300 kPa), but this effect recovered during the latter part of the hyperbaric exposure. A standard hyperbaric treatment profile with intermittent air breaks was examined by Pelaia.[37] Cardiac output, heart rate and stroke volume were all significantly reduced, while mean arterial pressure increased compared with that at 1 ATA. Cardiac output and stroke volume reduction rapidly reverted to control levels after cessation of HBO, but no change was noted during air breaks. Reduced cardiac output probably relates primarily to the combination of heart rate reduction and increase in systemic vascular resistance. However reduction in coronary blood flow[15] in combination with increased indices of contractility[35] may lead to negative supply/demand ratio with resultant cardiac ischaemia and loss of function.

PERIPHERAL CIRCULATORY RESPONSES

The peripheral circulatory response to 1 ATA oxygen in anaesthetised dogs was examined by Plewes.[14] Compared to an air control group, both heart rate and cardiac output fell by 14% and 7% respectively. There was no change in total peripheral resistance, however regional tissue beds showed a variable response to oxygen administration. Renal outer cortical and juxtamedullary blood flow fell by 20%, while there were no changes in overall splanchnic blood flow. Total cerebral blood flow was unchanged, but mesencephalon, vermis and hippocampal flows differed significantly from control values. Retinal blood flow was most markedly reduced, by 27% from the air control. Oxygen delivery may be significantly altered due to the balance of increased oxygen solubility combined with reduced tissue blood flow. Since this is calculated as the product of cardiac output and arteriovenous (A-V) oxygen difference, it may be unchanged in acute hyperoxia. In dogs at normal atmospheric pressure (1 bar, 100 kPa) the total oxygen delivery was 106 ml/min on room air compared with 103 ml/min when breathing 100% oxygen at 1 bar. The 20% increase in renal vascular resistance and reduction in renal blood flow seen at 1 bar (100 kPa) oxygen[14] was also demonstrated to occur in canine studies at pressures up to 4 bar (400 kPa).[38]

In unanaesthetised rats at 5 bar (500 kPa), heart rate and cardiac output fell by 21% and 14% respectively.[15] Organ blood flow, measured with the microsphere technique, fell in most organs, but was maintained at control levels in the kidney, liver and adrenals. In comparison with other studies oxygen delivery was significantly increased to the kidneys at 5 bar (500 kPa), from 1.26 ml O_2/min/g to 1.67 ml O_2/min/g. Importantly, in this study, right and left ventricular blood flow fell by 41 and 47% respectively with no change in myocardial performance, suggesting that myocardial ischaemia may be a possibility at hyperbaric pressures.

Studies looking at haemodynamic modifications during HBO have tended to focus on healthy control subjects. However, Muhvich examined regional blood flows in an antibiotic-treated septic rat model.[39] There was an overall reduction in renal, adrenal and myocardial blood flow from 1 bar to 2 bar. These alterations continued for 20 minutes after conclusion of the compression. Interestingly, there was no difference between control and “septic” rats. This may question the model, which even at 1 bar did not show any haemodynamic difference between the two groups.

Ten critically ill patients, who required invasive monitoring, were subjected to hyperbaric oxygen at 2.5 bar (250 kPa). There was an increase in oxygen delivery (DO_2), but no change was noted in oxygen consumption (VO_2) and oxygen extraction ratio, in contrast to the limited human data at 1 bar.[40]

PULMONARY CHANGES WITH HBO

Administration of HBO causes a significant increase in arterial and venous PO_2. Healthy subjects breathing 100% oxygen at 3.4 bar (340 kPa) showed raised PaO_2 (1,721 mm Hg, 2.26 bar or 226 kPa) and PvO_2 (424 mm Hg, 0.55 bar or 55 kPa) levels while $PaCO_2$ levels were marginally raised.[24] This latter effect was presumably secondary to the loss of CO_2 buffering capacity from reduced haemoglobin.

HBO does have effects on both pulmonary mechanics and vascular responses.[41] There is ongoing debate about the effect on alveolar-arterial (A-a) gradients during HBO therapy. Many studies have shown an increased A-a gradient[24,42] of up to 460 mm Hg (0.6 bar or 60 kPa) at 3.0 ATA. Flook,[43] in a porcine model, suggested that the shunt fraction (Q_s/Q_t) could increase to over 25% during hyperbaric exposure to 3 bar (300 kPa). This contrasts with other workers who have failed to show any increase in A-a gradient during HBO.[44] Interpretation of such data is difficult as there are significant differences in the experimental models. Data from uncontrolled human studies suggest that the measured PaO_2 is greater than predicted in patients with significant pulmonary disease. In contrast, patients with normal lungs have lower than predicted PaO_2 levels.[45] This has important implications for HBO therapy, as expected PaO_2 levels may not be achieved with the usual treatment regimens in some patients.

Animal studies have shown that the acute administration of HBO at 2.8 bar (280 kPa) induced significant increases in pulmonary vascular resistance and blunting of normal hypoxic vasoconstriction, while lung mechanics (static lung compliance, wet to dry weight ratio and surface tension) did not change.[29] These changes recovered after breathing air for 24 hours. Hyperbaric oxygenation significantly improves systemic oxygen supply; however this effect may be offset by pre-existing pathology (e.g. chronic airway disease, cardiac failure), disease processes (e.g. sepsis), drug therapy (e.g. vasodilator or vasoconstrictor therapy) or airway management (e.g. positive pressure ventilation and positive end expiratory pressure [PEEP]). Measurement of PaO_2 during treatment sessions are highly recommended to optimise therapeutic goals.

SUMMARY

There are many physiological responses which occur during HBO treatment. These have been studied widely, but interpretation is difficult due to differing animal models used in the studies, presence or absence of anaesthesia, variable pressure, duration and gas mixtures utilised and differing experimental methodology. However, some basic conclusions may be drawn. Heart rate reduction occurs frequently and may be significant in those patients with existing bradycardia or conduction delay. The increase in peripheral vascular resistance, leading to reduction in cardiac output, needs to be considered in those patients with marginal haemodynamic status or who are on vasoactive medications. The onset and offset of these cardiovascular changes can be anticipated, with appropriate vasodilator and vasoconstrictor therapy readily available. Direct invasive monitoring of all critical patients is mandatory, along with the recognition that HBO may not always mean improved tissue oxygen supply. Abolition of hypoxic vasoconstriction, increasing dead space and shunt all reduce theoretical gains from measured oxygenation. As there is potential for organ damage during HBO, ECG, arterial blood pressure, urine output and oxygenation (using pulse oximetry or arterial blood gases) should be measured in all critically ill patients and in those with marginal organ function. Further work is needed to investigate this in controlled human studies at pressures between 2 and 5 bar (200-500 kPa).

Administration of hyperbaric oxygen

In Australia and New Zealand the administration of HBO is most commonly carried out in dedicated multiplace chamber facilities. This contrasts with parts of Europe, United States and Asia where monoplace chambers are more commonly used.

MULTIPLACE CHAMBERS

In multiplace chambers (Fig 1, p 7) oxygen is breathed via a hood or demand flow BIBS (built in breathing system) apparatus. In cases where there is head and neck pathology, e.g. burns or radical surgery, a hood is often preferable. In other instances, e.g patient preference or claustrophobia, BIBS apparatus may be the better option. There are also rare circumstances in patients who have tracheostomies, or other anatomical problems, who require modification to their breathing circuit.[46] The multiplace chamber allows attendants to be present with the patients and so allows patient interventions which would be impossible in a monoplace chamber e.g. intubation, pleurocentesis and intravenous cannulation. It can also use treatment pressures greater than 3 bar (300 kPa), and often up to 6 bar (600 kPa). Other assistance can be easily obtained by pressurising staff to the treatment depth rather than requiring emergency patient decompression. However, multiplace chambers are more expensive to manufacture and maintain, and require attendants for each treatment, thus exposing them to risk, albeit small.

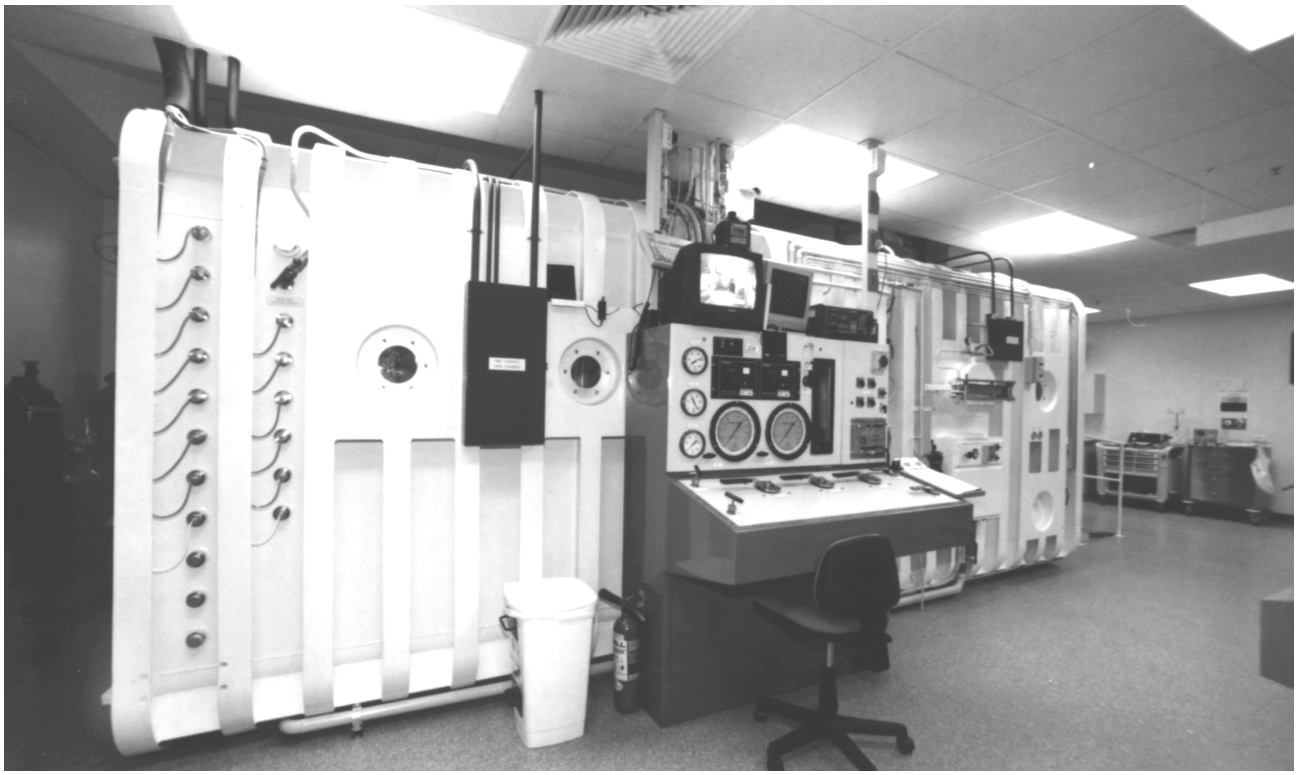

Figure 1. Multiplace chamber. (Royal Adelaide Hyperbaric Medicine Unit).

MONOPLACE CHAMBERS

Monoplace chambers (Fig 2, p 8) allow patients to breathe 100% oxygen from the environment. Air breaks can be given to co-operative patients who are able to use BIBS themselves.[47] They are cheaper than multiplace facilities, easy to use and do not require attendants. Their main drawback is lack of patient access if problems arise. These can be overcome, and some centres routinely treat unstable, critically ill patients in monoplace chambers. Finally, the additional risk of fire and explosion in a 100% compared to 21% oxygen environment, also makes monoplace chambers potentially riskier.

General

ATTENDANTS

It is essential that a trained attendant accompanies each patient during hyperbaric treatment sessions in a multiplace chamber. Ideally, these attendants should be the nurse treating the patient in the intensive care unit (ICU) or ward setting, so that continuity of care is maintained, although practically this may be limited by hospital staffing levels and nurse availability.

The risk of DCI in the patient undergoing hyperbaric oxygen therapy is rare. This is because patients usually breathe 100% oxygen for the duration of the treatment, apart from intermittent air breaks which minimise the risk of both CNS and pulmonary oxygen toxicity. DCI may become a problem in those treatment tables which use air as the breathing medium at depth, although these tables are rarely used nowadays. Attendants however do not breathe oxygen routinely at depth. Formal recommendations are made for some schedules used for diving problems (e.g. US Navy, Comex and other recompression schedules), but these are subject to many alterations. Moreover, the majority of HBO treatments are not standardised.[48] Finally, rates of compression, and more importantly decompression, are not uniform, thus potentially making the generation of in vivo bubbles possible with resulting symptoms and signs of DCI.

Anderson reviewed figures for DCI in 62 medical personnel who underwent 1,516 compressions.[49] Barotrauma was the most common adverse effect, affecting 47% of medical staff, though none required myringotomy or grommet insertion. Symptoms of DCI (extremity pain, pulmonary signs, retrosternal discomfort and dysaesthesias) occurred in 9 treatments (0.6%). Interestingly, there were also three episodes of homonymous hemianopia, one of which lasted for 10 weeks. Retinal field defects and cotton wool spots have been described in an attendant who breathed 100% oxygen at 2 bar (200 kPa).[50] Retinal vessels demonstrate marked vasoconstriction during hyperoxic exposure, an effect which shows great variability between individuals and in the same individual on different days. A more recent report from Baltimore reviewed 25,164 exposures.[51] The overall incidence rate of DCI in

Figure 2. Monoplace chamber. (photo courtesy of Dick Clarke, Carolina Hyperbarics).

attendants was 0.076%. Broken down by treatment pressure the incidence rate was as follows; 2.0 bar, 0.04%; 2.5 bar, 0.08%; 2.8 bar, 0.14%; 4.7 bar 1.68% and finally 6 bar, 5.71%. Figures from 1985 to 1995 at the Royal Adelaide Hospital Hyperbaric Unit indicate a similar rate. In 5,792 chamber runs, there were 4 reported cases of DCI, an incidence of 0.07%.

Inert gas narcosis (IGN) is well recognised in the diving literature as a consequence of exposure to inert gases (usually nitrogen) at depth with resulting neurological dysfunction. It has been suggested that there may be subtle neurocognitive effects of nitrogen at a partial pressure of 1 bar (100 kPa).[52] The relevance of IGN has been largely overlooked in the clinical hyperbaric literature. Attendants looking after critically ill patients require normal cognitive and psychomotor activity. Observation, interpretation and institution of corrective strategies may be affected by IGN. "Slight mania and euphoria" was self reported in 11 attendants out of 1,516 exposures at pressures above 2.5 bar (250 kPa).[49] Inert gas narcosis is a function of both depth and partial pressure of inert gas. Some hyperbaric units perform test treatments to acclimatise their attendants, making them aware of the effects of IGN by getting them to perform calculations and simple tasks at depth. There is some evidence from the diving literature that repeated exposure to depth "protects" against IGN. The presence of a trained nurse and physician outside the chamber also aids in the monitoring of both patient and attendant via close circuit television or direct observation.

BAROTRAUMA

Middle ear damage due to failure of equalisation is the most frequent complication of hyperbaric treatment. While it can be prevented in conscious patients who can auto-inflate their middle ear space, fluid retention, inflammation or Eustachian tube dysfunction can make this problematic.

Active auto-inflation during pressurisation led to acute severe hypotension (<40 mm Hg) in a patient with air embolism following a mediastinoscopy.[53] This patient had pulmonary artery occlusion, with the presumed mechanism of hypotension being reduced venous return following the Valsalva manoeuvre. An inadvertent breath hold by a study subject, produced hypotension (a drop from 120 mm Hg to 60 mm Hg) during decompression.[54] Again the proposed mechanism is that of reduced venous return from increasing intrathoracic pressure. Moreover, excessive equalisation can lead to rupture of the round or oval windows, resulting in tinnitus, deafness and vertigo. Equalisation problems are increased in the unconscious or sedated patient, when the first indication of a problem may be an unexplained tachycardia on compression which resolves suddenly after perforation of the tympanic membrane or bleeding into the middle ear cavity. There are two approaches to this latter situation. Firstly, emergency tympanotomy, with or without insertion of grommets. The second approach is to tolerate the middle ear bleeding, which will obliterate the air space and allow future compressions

to take place. However this may lead to problems with hearing impairment or infection, especially in the septic, immuno-compromised patient. The availability of ENT assistance and urgency of treatment often dictates which pathway is followed.

Theoretically, in patients who have an untreated or undiagnosed pneumothorax, HBO can be commenced before pleurocentesis is carried out. In many instances these pneumothoraces will resolve with 100% oxygen and compression alone.[55] In practice, unless there are exceptional circumstances, definitive management and stabilisation should be carried out before HBO. Chest tube insertion and connection to a Heimlich valve is the treatment of choice. Emergency pleurocentesis can be carried out in a multiplace chamber, although conditions may be cramped. It is not a viable option in a monoplace chamber; here a pneumothorax could be a life threatening situation, if decompression to ambient pressure was not possible before cardiac arrest occurs from a tension pneumothorax. However there have been no reports of such fatalities.

Other possible complications include sinus and dental barotrauma. Ventilatory impairment can occur in patients who have large amounts of intestinal, and especially gastric, air. Failure to relieve gastric distension has resulted in gastric rupture during decompression after a diving accident,[56] but has not as yet been reported following clinical HBO treatment. Nasogastric intubation should however be considered in patients who are sedated and ventilated, have had expired air ventilation or have a history of oesophageal anti-reflux surgery.

Skilled technical staff are required to prevent the ultimate barotraumatic insult, the uncontrolled decompression. Luckily this is rare, but is inevitably associated with mortality, often multiple.[57] As with other areas of modern medicine, treatment and crisis management algorithms can help prevent these disasters.

OXYGEN TOXICITY

Oxygen at high percentage or partial pressure has been demonstrated to have adverse effects on many body organ systems. The mechanisms of toxicity probably involve the production of reactive oxygen species (oxygen free radicals) which overcome the body's natural defence mechanisms. These highly reactive molecules include superoxide, hydrogen peroxide and hydroxyl ions. These molecules are produced in small quantities normally in the body, and can be dealt with by a variety of host defences which include avoidance of the univalent pathway, breakdown by enzymes (e.g. superoxide dismutase, catalase, glutathione peroxidase) and provision of natural anti-oxidants (e.g. vitamin A and C). However, under certain conditions, the defences can be overwhelmed, leading to alteration in membrane function, enzyme inactivation and subsequent loss of cellular and organ function. An in-house review carried out in the Royal Adelaide Hospital showed an overall incidence of CNS oxygen toxicity of 1.5 per 1000 treatments. This figure doubled in treatments at 2.8 bar or greater.

The incidence and severity of pulmonary and CNS oxygen toxicity can be reduced during treatments by the use of intermittent periods of lowered oxygen partial pressure (air breaks) during hyperoxia. Even brief (5 minute) air breaks can substantially extend the limits of oxygen toxicity. Hendricks showed that, using the decrease in vital capacity (VC) as an indicator of pulmonary oxygen toxicity, air breaks more than halved the decrease in VC seen with continuous hyperoxia.[58]

Experimental animal models have looked at various pharmacological methods of preventing the respiratory or CNS effects of hyperbaric oxygen. Strategies have included; vitamins A and E, prostacyclin analogues, intracellular hydroxyl ion scavengers (dimethylsulphoxide, dimethylthiourea, mannitol), chelating agents (desferrioxamine), lipid anti-oxidants (butyrated hydroxytoluene), arachidonic acid pathway modifiers (non-steroidal analgesic agents, magnesium), leukotriene inhibitors, inhibitors of nitric oxide synthetase and other synthetic analogues of natural defence mechanisms (catalase, superoxide dismutase), however none have been proven to be reliably effective in man nor have anticonvulsants, given for prophylaxis of CNS complications, been effective

HBO AND TUMOUR GROWTH

Finally, the question regarding cancer promoting effects of HBO needs to be addressed, for both patients and attendants. Theoretical risks of HBO therapy include; nourishing the tumour, immunosuppression and generation of oxygen free radicals, which are implicated in the genesis of some cancers. A review of the HBO literature has gone some way to answer this question.[59] Eleven animal, twelve human and one combined study were reviewed. Only two animal and three clinical human studies suggested a pro-cancer effect with HBO; all of these could be criticised on methodology and analytical technique. The conclusions drawn from available data suggest that HBO is not associated with an increase in tumour growth or metastases.

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