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IMPLICATIONS OF HYPERBARIC MEDICINE FOR ANAESTHESIA AND INTENSIVE CARE PART 2

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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, ventilators.

Intensive Care Unit (ICU)

Hyperbaric oxygen therapy may be required for patients who have severe, life threatening diseases that require ICU admission. These include patients who are unconscious (CO intoxication), have respiratory failure (smoke inhalation, CO intoxication), sepsis (clostridial, streptococcal and other soft tissue infections) and gas embolism (secondary to diving, open heart surgery and laparoscopic surgery). Careful consideration needs to be given to transportation, monitoring, airway and cardiovascular manipulations.

TRANSPORT OF THE CRITICALLY ILL

Intensive care patients may require HBO for acute, aggressive soft tissue infections and carbon monoxide intoxication. Recent animal and uncontrolled human data suggest that thermal burns may also benefit from HBO. Trends in plastic surgery dictate early debridement and grafting, hence these patients, often still ventilated and inotrope dependent, may require HBO in the acute phase.

It is well recognised that both inter- and intra-hospital transfers produce unwanted cardiorespiratory instability in critically ill patients. Treatments can mean that patients are away from the ICU for 2-3 hours, which may be repeated two to three times in a 24 hour period. While the absolute duration of time spent away from the ICU may not correlate with post-transfer respiratory performance, ventilatory manipulations, circuitry changes and patient movement may be more significant. Patients who require positive end-expiratory pressure (PEEP) are at increased risk of post transfer respiratory disturbance, which may last for more than 24 hours.[60] This can be minimised by minimising patient movement (e.g. using dedicated hyperbaric beds and/or barouches which do not require excessive patient movement during HBO sessions) and maintaining similar ventilatory parameters to those carried out in the ICU, including PEEP and intermittent mandatory ventilation (IMV). Inotrope infusions can be employed, using dedicated hyperbaric pumps. Skilled maintenance of critical care nursing is probably the most important variable in ensuring transfer stability.

Temperature maintenance can be difficult, especially in those patients who have extensive burns or other losses of skin cover. Pre-transfer temperature stability is essential. Many multiplace units have air conditioning to heat and cool the chamber. This is especially useful during phases of compression and decompression, when the chamber temperature can change several degrees over a few minutes due to adiabatic cooling and heating. These problems are further compounded by transfer through cool corridors and operating rooms.

As with most areas in medicine, the risk-benefit ratio must be assessed when considering treatment of these high risk patients. Potential instability needs to be balanced against the beneficial effects of enhanced tissue oxygenation, reduction in bubble size, stimulation of new blood vessel formation and augmentation of neutrophil function.

POSITIVE END EXPIRATORY PRESSURE

PEEP may be required to increase lung functional residual capacity (FRC) through recruitment, stabilisation and distension of alveoli, with resultant decrease in shunt fraction. Failure to maintain PEEP can lead to prolonged respiratory deterioration.[60] Modern ICU ventilators have internal, integrated valves which use the exhaust valve to produce PEEP. The airway pressure is constantly monitored and adjusted accordingly. External PEEP valves can be classified into either threshold (flow independent) or orificial (flow dependent), although most are hybrids of both.

In a study looking at four external PEEP valves, Youn identified that all valves produced an increase in preset PEEP by between 2 and 4 cm of water (0.2-0.4 kPa) in the hyperbaric environment.[61] The valves tested were; water column (Emerson®), spring type (Siemens®), magnetic type

(Instrumentation Industries®) and floating ball (Boerhinger®). Floating ball types are not adjustable, therefore less versatile in changing clinical circumstances. The water column was least affected by pressure change from 1 (sea level) to 6 bar (100-600 kPa). Awareness of these changes with HBO is noteworthy, as small unexpected increases in PEEP can potentially lead to reduced preload, septal shift, reduced ventricular size, increased dead space and barotrauma. PEEP levels need to be monitored closely and adjusted during different phases of the HBO treatment profile. Some authorities recommend that external PEEP be removed during phases of compression and decompression.[62] Other workers reported "occult PEEP" during positive pressure ventilation during HBO therapy.[63] Accurate airway monitoring and awareness of changes within the HBO environment can prevent many of these problems.

ENDOTRACHEAL TUBE (ETT) CUFFS

Boyle's law dictates that gas filled ETT cuffs will become smaller during periods of increased pressure. This problem can be overcome by routinely filling cuffs with saline or water. The increased mucosal pressure generated by these cuffs needs to be considered, especially with prolonged or frequent treatments. ETTs which use foam filled self inflating cuffs may be useful in this situation, but are not commonly available. A further option is to measure ETT cuff pressure continuously using a manometer and adjusting accordingly.

VENTILATORS

Critically ill patients with acute CO poisoning or fulminant soft tissue infections often require ventilatory support. The characteristics of an ideal hyperbaric ventilator are shown in Table 4.[64] Not surprisingly, no one ventilator, at present available, fulfils all the desired criteria.

Limitation of space and access mandates the use of small, compact ventilators. As with all hyperbaric appliances, electrical powered equipment presents a possible ignition source, which is of greater importance in the 100% oxygen environment of the monoplace chamber than in the multiplace facility. Maintenance of the same ventilatory parameters as carried out in the intensive care unit provides continuity of care and also promotes cardiorespiratory stability. Patients who are PEEP dependent also benefit from a ventilator which provides this modality in an accurate and minimally variable way. There are many methods employed in weaning patients from ventilation. It is useful if these can be employed in the hyperbaric unit for two reasons. Firstly, patient weaning may be delayed by continued HBO therapy if conventional IPPV is the only method available, leading to possible airway and systemic problems. Secondly, the work of breathing increases at depth due to increased gas density.

TABLE 4

DESIRED CHARACTERISTICS OF A HYPERBARIC VENTILATOR
Modified from Moon[63]

Small, compact.
No electrical requirements.
No flammable lubricants.
Wide range of minute volume with varying tidal volumes.
Constant inspiratory-expiratory time (I-E) ratio.
Minimum work of breathing with continuous positive airway pressure (CPAP) and T piece modes.
Weaning modes, pressure support synchronised intermittent mandatory ventilation (SIMV).
Constant positive end-expiratory pressure (PEEP).
Wide bore circuitry.
Powered by compressed air or chamber environment gas.
Driving gas vented to outside chamber.
Continuous monitoring of tidal volume, frequency, minute volume, peak and mean air pressure, PEEP, I-E ratio.
Control and display panels unaffected by pressure.
Similar to other ventilators in the Intensive Care Unit (ICU) and High Dependency Unit (HDU).

Patients who have been weaned onto a T-piece or pressure support ventilation may not ventilate adequately during HBO treatment if the only option is self ventilation via a modified BIBS circuit or head tent. Therefore continuous positive airway pressure, pressure support and synchronised intermittent mandatory ventilation are useful options to have in a hyperbaric ventilator.[65]

Ideally the driving gas should be that of the chamber environment, so that vented gas does not contaminate the chamber atmosphere. This would most commonly be air, which is safer from a fire perspective than oxygen. Finally, from an educational and safety perspective, ventilators similar to those used in other acute care areas e.g. intensive care and high dependency, are easy to use and reduce potential operator error because they are familiar.

There is a wide range of ventilators used in Australian and New Zealand Hyperbaric Medicine Units. These include Hyperlog (Dräger), Bird, Oxylog (Dräger), Oxford Mk II (Penlon). The hyperbaric literature has several studies which have looked at the efficacy of various breathing circuits and ventilators at depths from 2 to 31 bar (200-3,100 kPa).[64-78]

Early work from Lamy's group in Belgium identified problems with ventilators in the hyperbaric

environment.[69] The Celog 03 was limited by a lack of external controls once the patient was pressurised, whilst the Assistor was a fragile ventilator which required frequent interventions to alter inspiratory flow, expiratory pause and cycling pressure limit. It also had a low initiating trigger pressure, causing excessive cycling. The Logic 03 required several modifications to allow it to function at test pressures up to 3 bar. Using a test lung, it was found that both delivered tidal and minute volume decreased with increasing pressure, whilst the respiratory rate increased. The ventilatory pattern could be easily adjusted by a single alteration of the respiratory rate.

The Emerson, Urgency, IMV, and modified Mark 2 Bird were tested for performance using a test lung up to 6 bar (600 kPa). [70] While the Emerson (pneumatically powered piston) had no changes to preset parameters up to 6 bar, the Urgency and IMV Birds (pneumatic flow cartridges and venturi systems) failed at pressures greater than 3 bar and the Modified Mark 2 would not work above 4 bar. In addition respiratory rate increased as a function of more rapid pressurisation of the timing circuit. As the Emerson has bellows lubricated with mineral oil, this would have required further modification to allow it to meet accepted safety standards.

The Oxford ventilator (Penlon®) was evaluated using both air (up to 6 bar) and oxy-helium (up to 31 bar {3,100 kPa}) as driving gases.[71] Characteristics of ventilation were virtually unaffected up to 6 bar in air, but oxy-helium gas led to an increase in respiratory rate secondary to the less dense medium. Luckily, as the inspiratory and expiratory valves were identical, the set I-E ratios were maintained over a wide range of treatment pressures. A commonly used intensive care ventilator, the Monaghan 225 was reviewed by Moon to pressures of up to 6 bar. [64] There was no significant change in delivered tidal or minute volume, but respiratory rate decreased to almost half at 3 bar compared with 1 bar (sea level) and maximum minute volume decreased from 50 l/min to 18 l/min. Increasing both inspiratory flow rate and circuitry bore size can minimise this effect. The Pneupac range of ventilators have been assessed for both monoplace (Pneupac Variant HB) and multiplace (Pneupac HC) environment.[73,74] Reduction in flow through the flow limiting needle valve in the chamber produced a reduction in preset minute volume in both models.

Youn also looked at the Penlon Oxford using a test lung simulating pathological lung conditions utilising resistors and adjustable compliance springs.[75] Respiratory rate decreased with increasing resistance and decreasing compliance. With moderate resistance and compliance of 15 ml/cm H_2O, auto-PEEP [62,79] generated was 0, 2, 6 and 25 cm H_2O (0, 0.2, 0.6 and 2.5 kPa); peak airway pressure was 45, 50, 58 and 65 cm H_2O (4.5, 5, 5.8 and 6.5 kPa) at 1, 2, 3 and 6 bar respectively. Similar preliminary studies have been carried out with the Bird Avian ventilator.[68] The popular Siemens® Series has been used successfully in several centres for HBO patients. Some advocate a degree of modification to separate the power supply from the pneumatics,[76] whilst others report it to work effectively and safely using all its modalities under pressure (CPAP, PS, SIMV).

Weaning patients from ventilatory support can be associated with increased work of breathing which may be compounded by increased gas density at depth and work done against the ventilator. Oxorn compared a demand IMV system with a continuous flow device at 2 bar using a test lung.[65] The continuous flow system was associated with an increased work of breathing of 30% compared with a 200% increase with the IMV system over control at ambient pressure (1 bar). This has important implications for maintenance of such modes within the hyperbaric environment.

The Sechrist 500A monoplace ventilator is probably the most common monoplace ventilator in the US.[67] Like the other ventilators described above, there is a marked reduction in tidal volume which occurs in lungs with low compliance. Close monitoring is essential as injudicious increase in respiratory rate and/or tidal volume can predispose to auto-PEEP with its cardiovascular and barotrauma complications.[79] Air breaks can be easily administered to patients within a multiplace chamber, thus minimising CNS and pulmonary toxicity. These can also be carried out in the monoplace chamber in both conscious, cooperative patients and in those who are being mechanically ventilated.[47,80]

MONITORING

Minimum standards of monitoring are well recognised and followed in anaesthesia and intensive care practice, but this is not yet established in hyperbaric medicine.[81] Despite this, most units would recognise the need for a similar spectrum of monitoring as that employed in other acute care areas. Attendant and environmental monitoring are also essential in addition to patient monitoring. Normal clinical observation can be difficult in HBO environments for technical reasons. These include limitation of patient access, noise, decreased ambient lighting and altered sound transmission, making a simple technique such as chest auscultation difficult and unreliable. Specific criteria and standards need to be met for electrical and/or battery operated equipment within the HBO environment.[82] All equipment should have a dual power supply in case of primary source failure and all are required to be waterproof, explosion proof and protected from the chamber's sprinkler system. Finally, all equipment needs to be specifically designed for use in the hyperbaric environment, or tested for that specific purpose. However, the majority of monitors can work effectively with the electrical module on the outside, connected to the patient through dedicated penetrations in the chamber wall.

Pulse oximetry may have a place in hyperbaric practice in selected cases. Hypoxaemia and desaturation can occur even with a patient breathing HBO. A saturation of 97% along with measured PaO_2 of 346 mm Hg (0.45 bar or 45 kPa), on 100% oxygen at 2.5 bar, helped in diagnosing a right main bronchus intubation in a patient with severe rhino-cerebral mucormycosis.[83] Pulse oximetry can also be used to titrate a reduced FiO_2 during air breaks, again in an attempt to avoid CNS and pulmonary oxygen toxicity. This can be verified by performing arterial blood gas analysis using dedicated portable blood gas analysers. These have been shown to be accurate in both hypo- and hyperbaric environments.[84] An alternative method is to send a sample to a machine on the outside of the chamber. This is limited by rapid release of dissolved oxygen and the requirement for the machine to be calibrated within the hyperbaric range of partial pressure of oxygen.

The Anaesthetic Incident Monitoring Study recognised the limitations of routine electrocardiogram (ECG) monitoring,[85] however several conditions which present for emergency care have important cardiac complications. These include myocardial ischaemia and infarction with acute CO poisoning, atrial fibrillation and other supraventricular dysrhythmias with sepsis, and a multitude of ECG changes secondary to metabolic, endocrine or pharmacological reasons. Continuous ECG monitoring is recommended in some cases of DCI which present with rhythm problems, e.g. ventricular ectopic beats[86] and during adjunctive lignocaine therapy, which has been reported to be of benefit in refractory cases of DCI.

End-tidal carbon dioxide ($ETCO_2$) monitoring is mandatory for all intubated patients, yet is used infrequently during HBO therapy. The most common types are main stream and side stream capnographs. In a study comparing the accuracy of these at 1 and 3 bar, it was suggested that the main stream machines were less accurate than side stream models at pressure.[87] However, modification to the chamber of the side stream analyser, necessitating addition of a flowmeter and dump valve for the exiting gases to the outside, limits its feasibility. A relatively new chemical indicator, Easycap (Fenem) incorporated into an ETT connector, which changes colour, breath by breath, can provide inexpensive capnometry within the chamber.[88] It has been used during HBO treatment, but is limited by lack of alarms and difficulty in viewing the indicator from outside the chamber. Disconnection from the ventilator can be missed in an area where there is significant background noise and capnography is not universally used. The Ohmeda volume monitor was shown to work effectively and accurately in the hyperbaric environment up to pressures of 6 bar, and proved an essential monitor for use with critically ill patients.[89]

The use of advanced monitoring techniques, e.g. pulmonary artery (PA) catheterisation, are relatively easy to perform within a multiplace chamber with the appropriate technical alterations to the chamber. Apart from ensuring that the PA balloon is not inflated during decompression, because of the potential for PA rupture, there are no specific difficulties. This contrasts to their use in the monoplace chamber, where complex engineering is required to modify the chamber.[90] In addition, rapid removal of the patient is difficult due to limitation of mobility and attachment of monitoring. This limits the efficacy of such a monitor within a monoplace chamber.

DEFIBRILLATION AND PACEMAKERS

Problems associated with defibrillation inside a chamber include fire and explosion along with equipment malfunction, e.g. cathode ray screen implosion. Prerequisites for generating a fire or explosion include a source of flammable material, source of combustion and oxygen. Static electricity generated by clothing is eliminated by the use of cotton materials in the chamber. In addition, potential sources of electrical discharge, e.g. batteries, brush-motors and lighters, are prohibited. Fires have been reported in both monoplace and, less commonly, multiplace facilities.[91] There have been concerns about spark generation from defibrillation. This is especially valid in monoplace chambers where there are 100% oxygen environments; here defibrillation cannot be safely carried out. In this situation the patient needs to be decompressed and resuscitated once well outside the chamber. Multiplace chambers on the other hand, with an upper oxygen limit of 23%, have a much increased safety margin. While altered thoracic impedance may be present at higher partial pressures of oxygen, due to altered blood volume and flow secondary to oxygen-induced vasoconstriction, successful defibrillation has been carried out without complications. Some authorities recommend that, during defibrillation, one inside attendant wears emergency breathing apparatus and readies the fire hose while defibrillation is proceeding.[92] Another method of avoiding the problem is to site the defibrillator outside the chamber with leads extending through the chamber wall dedicated penetrations to specially designed gelled monitoring defibrillation pads.[93] Although the delivered energy was reduced by approximately 9%, this was within the accepted limits of the machine operating specifications. This minimises many of the problems and additionally reduces any potential operator error due to inert gas effects at increased ambient pressure.

Temporary transvenous external pacemakers have been reported to have failed under hyperbaric conditions.[94] Katz tested twenty permanent and eighteen external pacemakers in pressures from 1 to 6.7 bar (100-600 kPa) in 100% oxygen environments.[95] All permanent pacemakers functioned normally up to 6.7 bar, however the external pacemakers failed at 3-4 bar, with completely normal function before failure. Normal function spontaneously returned during decompression. Subsequent animal experiments gave the same results. Therefore, while

patients with implanted pacemakers can safely undergo HBO therapy, emergency patients may require close observation if treatment pressure exceeds 3 bar. Automatic implanted cardiac defibrillators (AICD) can also tolerate pressures up to 6 bar. The use of external transthoracic pacing has not been reported in the hyperbaric literature and limited data exits on the feasibility of intra-aortic balloon pumps within this environment. [96]

FLUID MANAGEMENT

The debate continues as to whether crystalloid or colloid is the optimum fluid for resuscitation. In a series of dog experiments, Gross[97] examined the haemodynamic effects of induced hypovolaemia during HBO therapy and the effects of infused intravenous fluids. Exposure to pressures from 2.8 to 6 bar did not change the haemodynamic responses to shock compared with controls at ambient pressure (1bar). In addition there was no change in the volume of colloid (dextran 70) required to resuscitate the dogs at depth compared with at the surface. The conclusion was drawn were that fluid management during HBO should not differ from that outside the chamber and that HBO per se did not alter the normal homeostatic mechanisms involved with hypovolaemia.

Anaesthesia

BACKGROUND

Anaesthesia in the clinical hyperbaric environment was first reported by Bert in 1879. It became more common in the 1950s and 1960s, but is rarely performed today with possible exceptions being therapeutic lung lavage for pulmonary alveolar proteinosis[98] or emergency surgery for commercial saturation divers who cannot be rapidly decompressed. There is some experimental evidence that anaesthesia per se may have a protective role against the development of pulmonary and central nervous system oxygen toxicity[99] and it may also have a synergistic effect in tumour killing with HBO and radiotherapy.[100]

Boerema described the use of halothane, curare and pethidine in cardiac surgery, but did not expand on techniques or problems. Later workers reported their experiences more specifically and mentioned limitations with hyperbaric anaesthesia.[101-106] In particular, temperature changes, flowmeter problems and vaporiser outputs were discussed. Potential solubility problems and DCI associated with the use of nitrous oxide were considered by Smith some 30 years ago.[103] It is interesting to note the case reports regarding DCI and N_2O in the recent medical literature.[107,108]

Anaesthetists may increasingly have contact with patients who have undergone repeated HBO exposures. The early work investigating the effects of HBO plus radiotherapy[7] on tumour growth is being re-examined today.[100] Patients may require surgery and hence anaesthetics during prolonged HBO courses. This is also true of patients with a history of osteoradionecrosis who have prophylactic HBO treatments (up to 30) before surgery. Debridement, resection and reconstructions are carried out, followed by postoperative HBO. Again the anaesthetist needs to be aware of the potential interactions between HBO and anaesthesia and, in particular, respiratory alterations with prolonged HBO therapy and potential drug interaction with chemotherapeutic agents.

EQUIPMENT

Historically, most of the earliest anaesthetics were nitrous oxide and oxygen with some added volatile agent. Early workers noted that as the pressure increased the output from the flowmeters decreased. This entailed calibrating each flowmeter for individual gases at the intended depth of treatment.

The vapour pressure of a liquid remains constant with variations in ambient pressure. Therefore, theoretically, at increased partial pressure the output of vaporisers should be constant. McDowall demonstrated that Fluotec vaporisers delivered accurately from 2-4% but, at lower settings, tended to over-deliver halothane at high ambient pressure.[102] This has not been repeated with modern vaporisers, but may be irrelevant as total intravenous anaesthesia (TIVA) is probably preferable in the modern hyperbaric medicine setting.

Any air-fluid interface will have potential problems in the hyperbaric environment if not allowed to equalise during compression and decompression. Gravity-fed intravenous giving sets need to be monitored closely to avoid collapse of drip chambers. Infusion pumps, commonly used in the anaesthesia and ICU setting, require to be reviewed and tested prior to any exposure in the hyperbaric environment. The accuracy must also be tested under hyperbaric conditions. The 350 Controller infusion device failed to function at depth due to the retrograde filling of the drip chamber.[109] In contrast the volumetric IMED® pumps, (960 & 928) functioned accurately at pressures from 1 to 6 bar. Syringe drivers, modified to remove flammable grease, are the most effective and efficient pump for use in TIVA. This anaesthetic technique can provide hypnosis, muscle relaxation and analgesia without the need for anaesthetic machines, complex circuitry and scavenging, and importantly avoids environmental pollution with expired hydrocarbons and nitrous oxide.

NITROUS OXIDE

Nitrous oxide anaesthesia was the sole agent used in the early days of hyperbaric anaesthesia. However two recent case reports illustrate a potential problem with nitrous oxide anaesthesia and the development of

decompression illness (DCI). Acott and Gorman reported a patient who developed transient symptoms of DCI following a provocative dive profile.[107] He subsequently underwent a nasal operation, during which nitrous oxide was administered. Symptoms consistent with DCI developed over the following two weeks, when he presented to the hyperbaric unit for recompression therapy. This provided immediate resolution of his symptoms.

The second reported case presented with symptoms of DCI following general anaesthesia (nitrous oxide) for relocation of a shoulder, which had been dislocated a few hours previously during the scuba dive.[108] Again the symptoms responded to HBO. As nitrous oxide has a blood gas partition coefficient 13 times that of nitrogen, any air bubble present in the body exposed to nitrous oxide will rapidly increase in size, causing symptoms of DCI. This has important implications for anaesthesia as in vivo bubbles have been identified in tissues for several weeks after diving.

To this end, a diving history should be sought for any patient presenting for surgery, nitrous oxide should be withheld from anyone who has participated in a dive over the past six weeks and entonox should not be given to any dive-related accident victim.

Miscellaneous

HBO is an accepted treatment in pregnancy, with no apparent detrimental effect to either mother or foetus. Van Hoesen documented the treatment of a mother with accidental CO intoxication.[110] She had a depressed Glasgow Coma Score with an accompanying carboxyhaemoglobin level of 47% (normal range <5%) with an associated foetal tachycardia and poor heart rate variability. The drowsiness and foetal cardiovascular changes rapidly resolved with HBO therapy. The use of HBO in pregnancy has potential adverse effects. These include teratogenicity, retrolental fibroplasia, reduction in placental blood flow and premature closure of the ductus arteriosus. None have been shown to be significant in man despite conflicting animal data. Practically, the acutely ill pregnant patient can be treated in the same way as other hyperbaric patients. Additionally, constant foetal heart and cardiotochogram monitoring should be routinely monitored during HBO treatments.

Finally, patients who have external fixateurs or complex traction devices can be easily accommodated in a multiplace chamber. Greater care needs to be taken in a monoplace chamber, where repeated scratching of the acrylic shell by these metallic devices may weaken the monoplace wall.[22]

Pain management

ACUTE PAIN

Patients who have had recent surgery, e.g. bone grafting in chronic osteomyelitis, debridement in gas gangrene or necrotising soft tissue infections, require analgesia. Patient controlled analgesia (PCA) is an effective method of providing immediate, effective pain relief yet requires a dedicated infusion pump, which may not be suitable for use in the hyperbaric environment. A Bard PCA device was subjected to pressure tests at 1, 2 and 6 bar (100, 200 and 600 kPa).[111] There was no evidence of battery leakage (using alkaline rather than lithium batteries), no alteration in PCA pump display or face plate and it was shown to deliver a test fluid within the specifications of the pump. It was also demonstrated to be clinically effective when used on 3 cases within a multiplace chamber.

The use of battery powered devices in a monoplace chamber is, however, not recommended. The combination of plastic and polymers which are combustible in 100% environments, along with threshold energy levels for combustion (in the vicinity of 1 microjoule), mean that these devices should not be used in monoplace chambers. The pump could be used via a nurse from the outside of a monoplace chamber and was shown to be accurate at 2 bar. Pumps which do not rely on battery power would be an advantage. An infusion pump using an elastomeric reservoir through a flow restrictor was tested with clinically relevant fluids at pressures of 1 and 2.3 bar.[112] All fluids demonstrated a statistically higher volume delivered under hyperbaric conditions compared to control, however these differences were not considered to be clinically relevant. Another variation, utilising a spring loaded infusion pump and flow restrictor, has been described for use in the intensive care setting, however it has not yet been subjected to hyperbaric environmental testing. These pumps might be ideal for use in both monoplace and multiplace environments.

CHRONIC PAIN

The observation that pain perception in patients with peripheral vascular disease was reduced by HBO led Tufano to study its effect in patients with acute and chronic vascular disease.[113] Subjective visual analogue pain scores were measured, along with plasma ACTH and endorphin levels. Both groups showed resolution of pain; this might be predicted in the acute traumatic ischaemia group, but less so in the chronic pain group. In addition ACTH decreased in both groups after HBO compared with before HBO. Plasma endorphins were reduced after HBO compared with before HBO values, more so in the chronic group. Animal experiments with Cu^{+2} as the oxidative agent produced oxidation of opioid receptor SH groups to SS

disulfide bridges.[114] This may lead to a stronger link between endogenous opioids and receptor sites causing a fall in observed endorphins and ACTH. As HBO also causes oxidation of these receptor SH groups, a similar mechanism for the effect of HBO on analgesia can be postulated. Other refractory chronic pain conditions, such as reflex sympathetic dystrophy, have also anectdotally responded to HBO.[115] Interestingly, Boerema in his report on the early use of HBO commented in passing on its effect on pain, "..the preliminary results are promising, the pain decreased considerably..".[116] Further study in this area is needed.

Drug interaction with HBO

Inter- and intra-patient variability in drug response is well recognised. What is less well understood is the compounding effect of HBO on drug pharmacokinetics and pharmacodynamics. This has important implications for patients requiring sedation, anaesthesia and intensive care within the hyperbaric environment. In addition, it must be emphasised that the pressure and oxygen parameters also change during HBO, thus any pathophysiological effects are in constant flux. Difficulty is encountered in extrapolating much of the present research data which is deep diving oriented (20-30 ATA) to normal clinical ranges (2-6 ATA).

ANAESTHETIC AGENTS

Johnson and Flager first demonstrated pressure reversal of anaesthesia in tadpoles anaesthetised with ethanol and urethrane.[117] Various animal models were subsequently examined, looking at mechanisms of general anaesthesia. With increasing pressure, there was a concomitant increase in anaesthetic requirement, inhalational agents being affected less than intravenous ones.[118] In most studies however, pressures far above those clinically relevant were investigated.

Intravenous thiopentone and ketamine were administered to guinea pigs who underwent exposure to 1, 20 and 31 bar (100, 2,00 and 3,100 kPa) breathing a mixture of helium and oxygen. Increasing pressure led to an increased dose requirement for inducing anaesthesia (2-3 fold), whilst shortening the duration of anaesthesia (up to 50% reduction).[119] Kramer tried to identify the aetiology of antagonism of pentobarbital anaesthesia, looking at drug pharmacokinetics and pharmacodynamics. Dogs were exposed to clinically relevant pressures of up to 6 bar and given intravenous pentobarbital. There was no significant effect on elimination half life, volume of distribution or plasma clearance, suggesting that changes in drug disposition, i.e. pharmacokinetics, did not play a role in reversal of anaesthesia.[120] This study design was repeated with pethidine and aminophylline, again showing no change in drug pharmacokinetics at 1, 2.8 and 6 bar (100, 280 and 600 kPa).[121,122] Dundas reported on a study from the Royal Navy which demonstrated that the dose of Althesin (alphaxalone/alphadalone) was increased by 30-34% at 300 m pressure (31 bar or 3,100 kPa)).[123] Although there tends to be an increased requirement for anaesthetic agents at pressures of 2-6 bar, little data exits for this clinical area.

SEDATIVES

The effect of HBO on the antihistamine clemastine fumarate showed that at 6.1 bar, breathing air, there were no significant CNS depressant or cardiovascular effects.[124] As these medications are in common use in the population for allergic phenomena, it is reassuring to note that hyperbaric therapy seems to be devoid of important interactions. This may have implications for chamber attendants who are on concomitant medication. The sedative effects of alcohol have also been antagonised in murine models, but at 12 bar in heliox environments.[125]

MUSCLE RELAXANTS AND SYNAPTIC TRANSMISSION

Muscle relaxants may be used in the management of the critically ill patient, although this is becoming less common. They may be advocated in certain cases, e.g. poor patient compliance with ventilation and minimisation of sedation, but consideration needs to be given to masking potential hyperoxic seizures. Direct EEG monitoring can be used in this situation, however a more practical approach is to monitor neuromuscular function using a peripheral nerve stimulator, minimising the degree of block or by using an isolated limb technique.

Pressure associated alterations in muscle contraction were first recorded by Regnard in 1891. Subsequent studies have suggested that there are important interactions between neuromuscular physiology and hyperbaric environments. Increasing pressure alone (137 bar or 13,700 kPa)) enhanced twitch tension, but did not change the electromyogram (EMG) response to phrenic nerve stimulation.[126] In the same study, using d-tubocurare and suxamethonium, EMG was depressed but twitch tension was enhanced, with non depolarising relaxant block being enhanced to a greater degree than the depolarising block. It has been suggested that pressure decreases acetylcholine release, but there is enhancement of some aspect of muscle excitation-contraction coupling. Although the net effect in this study was antagonism of neuromuscular block, the relative importance of pressure effects on neuromuscular transmission and excitation-contraction coupling are still unclear. What is even less clear are these effects at lower pressures, as there have been anecdotal reports of increasing relaxant requirements at 2-3 bar.

ANTIHYPERGLYCAEMICS

Diabetic patients are commonly treated with HBO for chronic non healing wounds, acute soft tissue infections

and chronic osteomyelitis. Blood sugar control can be unstable during HBO treatment, and hypoglycaemia is well documented. This may be related to reduction in plasma glucagon[127] or possibly increased sensitivity to insulin. In addition to hypoglycaemia, pre-hyperbaric administration of glucose may decrease the incidence of hyperoxic seizures.[128] Recent cases within the author's institution have shown that hypoglycaemia can be rapid and unpredictable, with blood sugars falling from 12 mmol to less than 2.0 mmol in the space of one hour's HBO therapy. Testing of blood within the chamber may not be accurate, leading to overestimation of actual blood sugar.[129] Due to the glucose oxidase mechanism involved in the reagent strips, it has also been suggested that blood removed from patients and passed outside may also over read correct values.[130] Whilst accurate measurement is problematic, clinical hypoglycaemia, manifest by confusion, agitation and loss of consciousness, is real[131] and often occurs in previously well controlled diabetics.

CHEMOTHERAPEUTIC AGENTS

Cancer patients may present for HBO for incidental reasons or primarily related to their tumour e.g. osteoradionecrosis. Bleomycin, an antitumour antibiotic, is used for squamous cell cancer, lymphoma and testicular cancer, but has significant pulmonary toxicity when associated with high concentrations of oxygen (PiO_2 >228 mm Hg = 30.4 kPa = 0.3 bar).[132] Patients are particularly prone to pulmonary toxicity (pulmonary oedema, pulmonary fibrosis and adult respiratory distress syndrome) if they have pre-existing lung pathology or bleomycin therapy within the previous 1-2 months. These problems may not be seen with the newer antitumour agent, peploycin. Cisplatinum, when studied in animal models, inhibited wound healing by blocking fibroblast production and collagen synthesis, whilst doxorubicin was associated with an increased mortality in animals co-treated with HBO.[133,134]

MISCELLANEOUS

Commonly used medications including aspirin, paracetamol, caffeine, diphenhydramine and dimenhydrinate were evaluated for learning and performance tasks during hyperbaric exposure to 2.8, 5 and 7 bar (280, 500 and 700 kPa).[135] Subtle cognitive deficits were demonstrated with the antihistamine, diphenhydramine, and even less with caffeine and dimenhydrinate. There were no effects with the simple analgesics. Performance evaluation showed no difference with any of these drugs at any pressure. This has important implications for patients and also for attendants who may be required to monitor, interpret and act appropriately under hyperbaric conditions.

The unpredictablity of drug behaviour at depth was demonstrated in rats given amphetamine and chlordiazepoxide at 10 bar breathing air. Both agents produced dose-related accentuation of some parameters at depth, however these were not predictable from the effects of the agents at ambient pressure.[136] Walsh, using amphetamine in a rat model at 7.1 bar, demonstrated a synergistic effect with pressure, a result not consistent with inert gas narcosis but with over stimulation of the CNS at depth.[137]

In summary, many of the studies looking at the effect of HBO on drugs have been in vitro or in vivo animal studies. Few have looked at interactions at clinically relevant pressures. However it would appear that drug effects are unpredictable compared with their effects at sea level (1 ATA, 1 bar or 100 kPa).

Surgery in the hyperbaric environment.

The earliest reports of surgery under hyperbaric conditions came from Fontaine who performed 27 procedures in an air hyperbaric environment, pressurised between 1.25 and 1.33 bar. Nitrous oxide was the anaesthetic agent employed, but while a portable operating room was constructed, a formal fixed surgical facility was never produced. HBO was subsequently employed for rational (reduction of hernias, production of bloodless operating fields) and completely irrational procedures (reduction of dislocated hips!). The first true fully equipped hyperbaric surgical facility was built by Boerema in 1959 in the grounds of the Wilhelmina Hospital in Amsterdam.[116] The operating area covered 24 square metres and was constructed in a cylindrical shape from metal. Landmark studies into HBO therapy were carried out in this facility. Duration of circulatory arrest could be increased using mild hypothermia and HBO compared with hypothermia and normobaric pressure.[138] Exsanguinated pigs, made normovolaemic with colloid solution and pressurised to 3 ATA, remained alive and showed no signs of myocardial ischaemia.[10] Subsequently Boerema successfully performed open heart surgery for procedures such as Tetralogy of Fallot with HBO. There was continued debate as to the lack of controlled data, even in those cases which appeared to have a sound physiological basis. When Bernhard's review of 86 operations performed with HBO was analysed it appeared that, in the group of infants with Tetralogy of Fallot, there was an increased mortality rate compared to those children operated upon under normobaric conditions.[139] The routine use of surgery within a hyperbaric environment was short lived due to the subsequent development of extracorporeal circulation.

The potentially beneficial effects on enhanced tissue oxygenation were investigated in the 1970s. Pulmonary embolectomy, using adjunctive HBO, was carried out successfully in a patient with marked desaturation and acidosis. It was suggested that this form of therapy might be beneficial to reduce the requirement for

cardiopulmonary bypass.[140] Minimisation of extremity hypoxia and spinal cord ischaemia during abdominal and thoracic aortic procedures were examined experimentally, but never gained clinical acceptance.[141] Similarly the potential advantage of preventing cerebral ischaemia during carotid endarterectomy and minimising graft rejection during transplantation were investigated, but never proved clinically effective.[141,142] In other hyperbaric areas, e.g. saturation diving complexes, emergencies such as acute appendicitis are generally treated conservatively, avoiding the need for surgery and anaesthesia at high pressure.[143] Broad spectrum antibiotics may prevent the need for surgery and anaesthesia, along with other problems of altered physiology of deep diving (450 m, 46 bar or 4,600 kPa)). These include infection from pseudomonas species, common in saturation habitats, immunosuppression and thrombocytopaenia.[144] Today, apart from surgery on saturation divers, the only other potential indication for surgery in HBO is in the treatment of pulmonary alveolar proteinosis. This procedure, designed to wash protein casts from the pulmonary tree, is associated with significant desaturation. Selective lung ventilation and washout in the hyperbaric environment can prevent such desaturation and allow effective lavage to be undertaken.[98]

Conclusions

HBO is established as an important adjunct for a variety of medical and surgical procedures. There are important cardiovascular and respiratory changes which occur that have important implications for the anaesthetist and intensivist. This type of therapy is not without problems, which include DCI, inert gas narcosis and barotrauma for the attendants. Patients are also at risk from pneumothorax, arterial embolism, ear and sinus barotrauma. Severely ill patients who require intensive care management pose a particular problem. HBO may be associated with major and prolonged physiological shifts, which can be prevented by close monitoring of cardiovascular and respiratory parameters, using dedicated hyperbaric ventilators and being aware of the limitations and efficacy of emergency procedures such as cardiac pacing. Although pressure reversal effects are well documented with general anaesthetics, drug effects are not well known within clinical hyperbaric pressure ranges of 2-6 bar. There are important interactions between anaesthesia, hyperbaric medicine and intensive care which need to be identified and considered. Knowledge of such can improve treatment efficacy whilst reducing potential incidents and resulting morbidity. Further research is needed to investigate the effects of anaesthesia and intensive care during exposures to hyperbaric pressures between 2 and 6 bar.

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The above paper is Part 2 of the thesis submitted for the Diploma of Diving and Hyperbaric Medicine which was awarded to Dr Kluger in 1996. At that time he was working in the Hyperbaric Medicine Unit at the Royal Adelaide Hospital, Adelaide, South Australia. Part 1 was published in the March issue of the Journal (SPUMS J 1997; 27 (2): 2-11).

AN ESSENTIAL RESEARCH PROJECT: LIVING WITH THE "AMERICANS WITH DISABILITIES" ACT

Douglas Walker

Key Words
Fitness to dive, legal, medical conditions and problems, research, safety.

The Americans with Disabilities Act[1] (AWDA) is one sign of the profound changes in community attitudes towards any perceived discrimination which prevents a person from either obtaining training or employment, or undertaking any other activity, solely on the grounds of some condition (physical, mental or behavioural) they may have. This Act has been presented by politicians as a caring measure in defence of the civil rights of such persons but, unfortunately, is equally likely to have results far beyond those imagined or desired by its creators.

It is very likely that this act will be manipulated by a few individuals, intent on obtaining a financial gain,[2] using the inevitable loopholes which will exist in this, as in all, legislation. There will be claims of unfair or unjustified restrictions imposed because of medical advice and common work-safety beliefs. Regrettably there will be some who may suffer an injury because they are allowed to undertake activities which their "disability" renders less safe for them to undertake than for those not so affected.

There is, however, a potentially positive aspect to implementation of this Act (AWDA). The enforced employment of such persons will make employers consider designing much needed improvements in the work environment. In the recreational industries, those having a responsibility for the safety of participants will be in the same situation. If these improvements are carried over they should reduce the risk levels for all, and so benefit everyone exposed to such work or recreational environments.[3]

The diving community will undoubtedly be significantly affected by this Act, at first in the United States but ultimately world wide, because of the direct and indirect influence of the major diver training organisations.[4-6] These organisations originated in the USA and still dictate the content of training programs of their many overseas dependencies. The effect may well be delayed initially in America through the writing of effective "disclaimer" contracts.[7-9] However what one lawyer devises is usually eventually circumvented by another. The "American disease", of litigation at the least excuse, is spreading to other countries where such disclaimers of liability are far less protective.

The instructor organisations appear to have made a rod for their own backs by their strict enforcement of the rule that no changes can be made by instructors to the written training programs. This rigidity may be welcomed by their insurers and legal advisers as providing a convenient justification for all actions which rigidly follow these hitherto unquestioned protocols. However there is a down side. It appears that the organisations fear to modify their training protocols in line with incident and morbidity reports. It has been suggested that the reason is that any changes could later be represented, in court, as an admission that some parts of the present training programs were either inadequate or contained errors.

This paper is not to discuss the training programs of the instructor organisations beyond stating that there is obviously scope for discussion on the correctness of awarding the somewhat misleading title of "advanced diver" to some divers after they have made only nine scuba dives. In my opinion, the conclusions reached by the UMS (now UHMS) and SPUMS workshops,[10,11] about the necessity for including a practice of shared air ascents in basic training, were reached without a sufficient regard to incident and morbidity data, which was available and should have been more fully considered. These workshop decisions will be a delight to any litigant's lawyer, as there was obviously an acceptance by those involved that to run out of air is a situation which is so common and unavoidable that it should be accepted. This training module is justified to reduce the very obvious dangers of running out of air. The presumption in AWDA is that predictable risk factors must be removed, not accepted, so it could be cited to back a claim that the avoidance of such out-of-air situations should be the focus of diver training. This would reduce the risks that diver inexperience and low-air situations pose.

However it is the medical involvement in assessment of medical fitness to dive problems which is the primary interest here. Doctors first became interested in pressure-