Case reports

Saturation treatment in shore-based chambers for divers with deteriorating cerebro-spinal decompression sickness

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Recompression, saturation treatment, heliox, hyperbaric oxygen therapy, cerebro-spinal decompression sickness, divers, diving

Abstract

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Nearly 4% of all primary recompression treatments in Scotland employ saturation tables (helium/oxygen-oxygen/air or oxygen/air alone). These cases usually involve divers presenting at the surface who then develop deteriorating spinal cord injury with varying degrees of cerebral involvement. Treatment is delivered either through immediate saturation therapy or through conversion of failing or failed primary treatment. The basic principles and delivery protocols of saturation treatment are outlined. A case study from both types of treatment is presented to illustrate the forms of decompression sickness that may require saturation treatment and how the treatments are initiated and evolve.

Introduction

In an analysis of treatment protocols for emergency recompression of divers in shore-based chambers in Scotland, treatments based on saturation were reported for approximately 4% of all cases (range 1–6% across the treatment centres employing saturation).[1] Two forms of saturation therapy are used in Scotland: helium/oxygen-oxygen/air ('heliox') and oxygen/air ('air') saturations. The basic principle of both treatment protocols is that entering saturation extends greatly the duration of treatment at pressure in circumstances where the alternative is returning to the normobaric situation before the presenting condition has improved or even stabilised.[2] The heliox tables have the further advantage of being able to increase the depth of compression with reduced oxygen partial pressures and there may be benefits in some cases of using pressures greater than 284 kPa.[3] In addition, some hyperbaric clinicians believe there are therapeutic advantages to 'heliox' over oxygen alone, though this is supported only by anecdotal evidence. The present account outlines methods and considerations for delivering the two types of saturation treatment. Two case reports are presented that required saturation treatment, one heliox and one air. These selected examples demonstrate the types of presentation that result in saturation.

Methods

The oxygen/air saturation treatments delivered have followed the US Navy treatment table 7 (USN 7) protocols; these are outlined in detail elsewhere.[4–6] The delivery of USN 7 at Dunstaffnage was dependent on the continuous presence of a doctor trained in diving medicine and a team of 4–6 external chamber operators. USN 7s were run with only one internal attendant; extra attendants were locked in and out when necessary using standard air decompression schedules. Saturation treatment was not embarked upon if there were too few external operators available to support the treatment or where there were too many internal attendants already inside the chamber.

The helium/oxygen-oxygen/air saturation treatments began with an initial compression to 284 kPa (18 metres' sea water, msw) on air. Where there was no response from the patient, the doctor in charge would complete any medical interventions before further compression on 2% O_2 in He to the depth of relief (ranging from 405 to 608 kPa, 30 to 50 msw). The 2/98 O_2/He mix was used for pressurisation only; the oxygen content of the therapeutic gases delivered ppO_2 levels of 2.0 bar or less and was determined by the depth of relief; chamber atmosphere oxygen levels were 41 kPa (0.4 bar) at storage depth and 51 kPa (0.5 bar) during the decompression. An extra nurse was locked in at this time with the added requirement of an additional nurse externally for logistic support. Each 24 h period required two life support technicians externally. A minimum period of 24 h was required at the chosen storage depth to ensure saturation; during this period, treatment gas sessions were delivered following the USN 7 format (repeated cycles of four 25-minute treatment gas with 5 min air break session for 2 h, then air only for 2 h).

Decompression on the helium saturations follows the USN 7 format but allowing for entry into decompression from deeper initial storage depths. Decompression from storage depth to 253 kPa (15 msw) was at a rate of 60 $min.m^{-1}$; from 15 msw to surface was at a rate of 80 $min.m^{-1}$. Decompression was suspended between midnight and 0600 each day. Permission was obtained from the subjects of both case reports for inclusion in this account.

Case reports

CASE 1: AIR SATURATION TREATMENT FOR SPINAL DCS

A 41-year-old male with no past medical history of note, except a possible allergy to the stinging jellyfish *Cyanea capillata* (Linnaeus, 1758) for which he carried self-administrating adrenaline, was diving in the Sound of Mull on the west coast of Scotland. He reported that he had been using tables to control his decompression though there was some doubt as to the reliability of this. His dive computer became 'lost' during transfer; all depths and times reported below are, therefore, recollections of the patient who had obvious signs of confusion, backed up only in part by information from his diving companions.

The first, uneventful dive was at approximately 1800 on the Friday evening to a maximum depth of 23 msw for a total time of 25 min. The following day, after a surface interval of 19.5 h, he carried out a second dive to a maximum depth of 28 msw; after 10 min he started ascending. At approximately 20 min dive time, buoyancy control problems resulted in a rapid ascent to 6 msw. An over-correction of his buoyancy caused him to descend again to 28 msw. Gaining control of his buoyancy he ascended again, stopping at 9 msw and 6 msw for 10 min each. He reported some panic during these stops because of the presence of numbers of stinging jellyfish before surfacing at 1250 after a total dive time of approximately 50 min.

Following a short swim back to the dive boat he felt unwell and required assistance to exit the water. Within 10 min of surfacing he developed loss of sensation in his legs; this deteriorated quickly and he was unable to move his legs against gravity on making shore less than 10 min later. At 1320 he was seen by the local doctor who confirmed paraplegia and commenced him on high-flow oxygen although failed to establish intravenous (IV) access. He was evacuated urgently to the Dunstaffnage Hyperbaric Unit (DHU) using the Oban lifeboat with an experienced diving doctor on board.

On arrival at the DHU at 1505, he was conscious, fully orientated in time and space and demonstrated a complete paraplegia with a sensory level to touch and pin prick at his waist. Additionally there was marked weakness to flexion and extension of his arms with weakness of grip of the left hand being worse than the right. Cranial nerve examination and serial sevens were normal. Prior to recompression IV access was established and he received two litres of normal saline over the next two hours.

Recompression was started at 1630 using the Royal Navy treatment table 62 (RN 62) protocol. After three standard oxygen/air cycles and one oxygen extension at 284 kPa (18 msw) there were no discernable objective or subjective signs of improvement. Given the serious nature of his condition at this stage, the treatment was converted at 1745 to a USN 7 at 287 kPa (60 feet sea water, fsw). In addition, he was given dexamethasone 80 mg intravenously.

Within 30 minutes, he reported some return of sensation with small movements to his toes; he passed 750 ml of urine. By 2000, he was able to lift his legs to 90 degrees against gravity. At this point, the table protocol prescribes a 2 h air break; mid-way through this break, he was able to stand unsteadily unsupported with his eyes open but could not walk. Following completion of the next 2 h oxygen session (7.5 h under pressure) he had return of normal power in his arms and could just walk, though with a broad-based gait and very unsteady Romberg test.

During the next set of oxygen cycles, he started to develop a cough but on completion at 0400 (day 2; 11.5 h under pressure) he could walk slowly with more confidence, and a less broad-based gait. The 0600 assessment prior to commencement of further oxygen showed improved gait and demonstrated careful, slow heel-toe walking; he could heel-shin slide while standing on one leg. Unfortunately at this stage (0615), oxygen breathing had to be stopped because of pronounced coughing and retrosternal chest pain. He had received over 1650 units of pulmonary toxic dosage (UPTD). Intravenous fluids were suspended having received 5 L and he was taking oral fluids well.

His condition remained stable on air, and the ascent from 287 kPa was commenced following 14.5 h at that depth. During the first few hours of ascent he continued to have an irritant cough and he described a number of pains in his back (which he associated with an old injury) and lower leg (attributed to a previous fracture). By 1700 (day 2, 24.5 h under pressure) he was at 200 kPa (32 fsw); his condition was essentially unchanged, he described his head feeling less clear and simple mental arithmetic challenges showed some impairment. He was given a further dose of dexamethasone and, as his chest symptoms had lessened, he was recommenced on oxygen-breathing cycles. These were continued until 2300 (day 2) but suspended overnight to permit sleep. The following morning (day 3), after a good sleep, neurological examination was unchanged, and he surfaced at 1930 (day 3; 51 h under pressure) and was transferred to the Oban hospital for post-recompression monitoring.

During his hospital stay, he underwent psychometric examination in which he showed major loss of semantic memory (vocabulary), and was slightly confabulatory, with perseveration that raised suspicion of a recent compromise of the cerebral circulation. He was discharged on day seven with normal motor power, slight ataxia on walking, a small area of sensory loss on his right foot and no ongoing discomfort or pain. He was advised not to dive again, which we believe he has adhered to.

CASE 2: HELIUM SATURATION TREATMENT FOR SPINAL DCS

A 70-year-old male carried out a shore dive, his first dive for 14 weeks, on the Scottish east coast. This was the deepest and longest of ten dives since having an aortic valve replacement 10 months previously, for which he was on warfarin. The dive commenced at 1448 to a maximum depth of 26.5 msw with a total dive time of 36 min including a one-minute stop at 6 msw. Within 10 min of surfacing and while on the way back to his car, he developed upper back discomfort with bilateral leg weakness and numbness from the thighs distally. He no longer had sufficient strength in his legs and collapsed to the ground with uncontrolled leg jerking. 100% oxygen was commenced and the local emergency services were contacted. Transfer to the Aberdeen Hyperbaric Medical Unit was organised by helicopter with the patient arriving at 1800. During transfer he was described as having weakness of all limbs, though he had some subjective improvement on oxygen.

On admission, examination demonstrated mild upper limb ataxia, bilateral up-going plantar reflexes, absent abdominal reflexes, paraesthesia over the feet, a wide-based, ataxic gait, and a blood pressure of 190/110. All blood haematology and biochemistry were normal, and his INR level was 2.9 (daily target range 2.5–3.0). He was able to pass urine. Upward progression of the paraesthesia in his legs was noted prior to recompression. Subsequently it became known that he had a history of spinal shock following a rugby accident in his 20s.

Intravenous fluids were commenced and he was compressed at 1905 to 284 kPa (18 msw) using the USN 6 protocol. Continued hypertension was noted while under pressure and he became unable to pass urine but declined urinary catheterisation. With the IV fluid resuscitation continuing, his urinary retention progressed, precipitating and then aggravating myoclonic jerking of his legs. This settled when urinary catheterisation was finally permitted but complicated neurological assessment regarding the progression of decompression illness to the extent that it was not really possible. At a pressure of 192 kPa (9 msw) it was again difficult to assess his neurological progress. Although there had been some improvement, walking was with unsteady gait and paraesthesia was present from his thighs distally. He surfaced following a fully extended USN 6 treatment at 0225, at which point examination showed his condition to be about the same as he had been at 18 msw prior to going into retention and worse than prior to recompression. He was returned to the wards for monitoring with a further recompression planned for later that day.

At 0630 (day 2) he was reported to have had a sudden deterioration in his condition, with nausea, and examination found him to have a sensory level at his xiphisternum (T4/5), unable to sit with hip extension power grade three, and finger-nose ataxia . At 0900, he was recompressed on a Comex 30 protocol. Soon after starting 50% oxygen in helium at 405 kPa (30 msw) the patient reported that the deterioration in his condition had halted. At the end of the first hour, considerable improvement in muscle power was noted by the attendant nurse but it was clear that the patient was by no means back to normal. At this stage, the Comex 30 table requires decompression to 344 kPa (24 msw). In view of the substantial and continuing improvement, however, it was decided to convert his treatment to helium saturation and he was further compressed to 507 kPa (40 msw). Treatment at that depth consisted of breathing cycles of 25 min using 35% oxygen/helium with 5 min breaks for four cycles at a time, repeating these cycles after a two-hour air break. In terms of O_2 levels between heliox treatment sessions, the target level was 41 kPa (0.4 bar) at storage depth and 51 kPa (0.5 bar) during the decompression.

While still at 40 msw (507 kPa), he developed a raised blood glucose requiring sliding scale intravenous insulin administration and he was also given low molecular weight heparin (LMWH) after discussion with his cardiac surgeon. His treatment gas cycles were suspended at midnight and recommenced first thing in the morning of day 3. At 1030 on day 3 (having had just over 24 h at 507 kPa) reassessment found him to be stable, able to stand on his own albeit with a shuffling gait. Decompression was started with continuation of the treatment breathing cycles with 50% oxygen in helium at 28 msw (385 kPa) and 100% oxygen at 18 msw (284 kPa). Decompression was completed on day 5 after 78.5 h under pressure. He could walk with an ataxic gait with normal leg power and had a negative Romberg test. He remained catheterised and it became clear on the ward that he was unable to control his anal sphincter.

He was discharged to the rehabilitation unit on day 12 with his LMWH having been changed back to warfarin following a normal transcranial Doppler assessment. He was fully mobile, being able to manage stairs well and was independent but still had some sensory loss distal to his lower abdomen. He remained catheterised and had to use rectal suppositories. Four months after discharge he was essentially back to normal apart from some reported sexual impairment with erectile dysfunction. One year after discharge, he reported being back to normal and had taken up yachting. Eight years after discharge, he reported enjoying good health, being very active, but missing scuba diving.

Discussion

The present account summarises the theoretical basis for saturation recompression treatment, outlines the methodology of delivery and gives two case studies as example of how saturation can be employed. All cases where saturation has been used in Scotland have involved patients with intractable or deteriorating neurological problems. In some cases, saturation has been preceded by serious relapse following initial treatment; all cases entering saturation will either have failed to respond to the initial stages of shorter

treatment tables or have been predicted to fail. Both types of saturation described here ('heliox' and 'air') have defused difficult clinical situations and negated subsequent relapse using well-tried and safe decompression schedules.

Whether saturation treatment is more beneficial to the final outcome than other treatments is the subject of ongoing appraisal in Scotland. Nearly all USN 7 treatments carried out have resulted in pulmonary oxygen toxicity for the patient, which may affect the optimum therapeutic effect through impaired lung function.[6] USN 7 use is considered to have little benefit *per se* apart from providing more time at depth (maintaining some bubble compression) to permit use and possible outcome of adjunctive measures. The Scottish Chamber Network treatment algorithm now has two options: (1) stabilisation using standard or extended RN 62/USN 6 treatment followed by immediate helicopter transfer on surfacing to Aberdeen (1–1.5 h transfer) for subsequent treatment, probably using heliox saturation; or (2) treatment using 30 msw (405 kPa) 50:50 heliox tables, such as the Comex 30, which have been used previously for treating spinal cord decompression injury.[7,8]

Heliox saturation treatments provide many more options to the treating centre in terms of enhanced flexibility for selecting treatment depth (added bubble compression) and duration (without concomitant oxygen toxicity). However, in these cases, as well as providing additional time at depth for adjunctive therapy, there are the possible added therapeutic benefits of helium breathing mixtures.[9–11] The use of heliox at pressure possibly causes shrinkage of nitrogen bubbles in a number of tissues, though after an initial expansion.[9,10] Heliox use may give some spinal cord function protection and cause some air bubble shrinkage in white matter compared with an initial growth using oxygen.[11] However, some mathematical models suggest that helium may prolong existence of bubbles in spinal and fatty tissues with isobaric counter-diffusion during the switch from high-pressure air to heliox.[12] Animal models imply that heliox is not beneficial in the treatment of respiratory decompression sickness.[13,14] Animal models of helium use in decompression illness, however, generally study isobaric administration of helium gas mixtures. In clinical practice, heliox combined with increased ambient pressure should limit or remove the risks of bubble growth and the treatment has been described as beneficial in man.[15] Oxygen acts as a vasoconstrictor and whereas the RN 62/USN 6 treatments use 2.8 bar ppO_2 (284 kPa) at the maximum treatment pressure, heliox treatments employ ppO_2 levels of 2.0 bar or less at absolute pressures of 4 bar (405 kPa) or more. Perfusion-dependent nitrogen elimination decreases secondary to vasoconstriction induced by increasing oxygen pressures.[16]

Saturation treatments should only be considered where the treating facilities are adequate and the patient has significant neurological injury.[5] Vestibular DCI may not be an indication, for example, but in cases of severe spinal cord illness where deterioration is continuing at the key treatment depth, saturation provides an additional option. However, saturation treatments do carry risks to both the patient and the internal attendant that need to be balanced against the potential outcome. By its very nature, saturation prevents accelerated bail-out procedures that may be necessary in case of events such as external fire or patient mortality. If compression to depths of greater than 18 msw is thought necessary, however, the use of heliox avoids effects such as nitrogen narcosis and pulmonary oxygen toxicity in attendants while they attempt to conduct the complex care necessary for a seriously injured diver. The internal attendant(s) should be well hydrated at all times, encouraged to exercise/walk frequently during the treatment and adhere to some form of post-treatment health observation procedure. The patient will have elevated risks of deep vein thrombosis, pulmonary embolism and stress peptic ulceration. Para-/quadriplegic patients will require frequent turning; some psychological effects may be evident in patients that are becoming more aware of their condition and likely outcome in addition to contending with physical impairments such as bladder and bowel incontinence. In addition to maintaining maximum carbon dioxide levels in the chamber to below 0.5% surface equivalent (0.5 kPa), atmospheric oxygen depletion can become important at shallower depths in the treatment and must be monitored and adjusted accordingly.

Although some studies have described the need for additional compression treatment following saturation,[13] in most cases completion of a saturation table will conclude the compression phase of recompression therapy. Where saturation delivers an improved outcome, the treatment cost must be measured against the alternative long-term expenditure on patients with significantly impaired cerebro-spinal outcome.

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