Extreme survival: A serious technical diving accident

Article · March 2005

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Extreme survival: a serious technical diving accident

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Key words
Decompression illness, technical diving, rebreathers/closed cicuit, hypercapnia, case reports

Abstract

(Trytko B, Mitchell S. Extreme survival: a serious technical diving accident. *SPUMS J*. 2005; 35: 23-7.)
A 34-year-old technical diver involved in a dive to 105 metres' sea water incorrectly assembled his rebreather and suffered carbon dioxide toxicity at depth. He developed anxiety, confusion and dyspnoea resulting in a rapid ascent with loss of consciousness. In the process, he omitted 51 minutes of decompression time. His subsequent presentation with life-threatening decompression illness and salt-water aspiration, and the management of these problems are discussed.

A note on terminology: *In this paper we follow Francis and Mitchell[1] in utilising the term 'decompression sickness' (DCS) to refer specifically to the consequences of bubble formation from dissolved inert gas, and the term 'decompression illness' (DCI) to refer to the broad spectrum of bubble-induced symptoms that may arise from DCS and arterial gas embolism.*

Introduction

Perhaps the most significant recent trend in recreational diving is the emergence of so-called 'technical diving' methods, adopted to extend underwater duration and/or to facilitate dives deeper than the conventional 'recreational limit' of 40 metres' sea water (msw). There is debate over which diving activities and methods qualify for the 'technical' sobriquet, but one proposal is that it includes diving that involves special techniques, decompression procedures and utilisation of gases other than air, or equipment other than single-cylinder, open-circuit scuba.[2] This definition embraces equipment applications such as multiple-cylinder configurations or rebreathers, and techniques such as decompression diving, nitrox and mixed-gas diving. Such diving requires additional training as well as significant investment in equipment and logistics. Technical divers often operate in hazardous, open ocean environments, and to depths previously encountered only in the context of commercial or military practice.

Clearly there are hazards associated with such activity. The decompression protocols utilised by deep technical divers can only be considered experimental and there is an undefined risk of DCS that may be significant even in dives that are conducted according to plan. Moreover, accurate calculations and obsessive attention to detail in the preparation of dive plans and equipment are essential. Mistakes will inevitably occur and may result in disaster in an environment that leaves little room for error. It seems inevitable that as technical diving becomes more prevalent we will see an increase in the number of divers with severe manifestations of DCS.

This paper describes a technical diving incident. We report this case because it is illustrative of some unique hazards of deep technical diving, and of the particularly severe DCI that diving physicians are likely to encounter more frequently in this group.

Case report

Diver X is a 34-year-old, experienced technical diver who had completed more than 2000 dives previously, including many to depths greater than 60 msw using mixed gases. Indeed, he is one of Australia's most experienced recreational mixed-gas divers. He was fit and had never suffered DCI.

Diver X was diving 10 kilometres offshore with three other divers and three support crew. The dive was to an irregularly contoured reef, with the depth varying between 95 and 110 msw according to the depth sounder. The plan was to spend 12 minutes at a maximum depth of 110 msw (including descent time) followed by staged decompression over 75 minutes.

Diver X was using a Biomarine Mark 15 closed-circuit rebreather (CCR). The diluent cylinder was filled with trimix 10/57 (10% oxygen, 57% helium, balance nitrogen) and the PO_2 setpoint was 130 kPa (1.3 ATA). In addition, he carried two 2.64 m^3 aluminium cylinders of 'bailout' gas in a sling arrangement. One contained air and the other nitrox 50 (50% oxygen, balance nitrogen). The system was set up for air to be introduced to the CCR loop as an alternative diluent so that nitrox could be used during decompression.

The divers were to enter the water staggered in two groups of two, with a five-minute interval between them. Diver X was the second diver of the first pair into the water and

descended without difficulty to 105 msw via the anchor line. During the next three minutes at depth, Diver X was noted to adjust his rebreather. At approximately eight minutes he indicated to his buddy that there was a problem with his CCR but was otherwise well and began an ascent after communicating he was all right to do so without assistance. He later recalled feeling dyspnoeic and anxious. At this time the next pair was descending via the anchor line.

On initiation of his ascent, Diver X was observed by his buddy to appear calm and in control. The first diver of the next pair formed the same opinion after an exchange of signals at 80 msw. However, on passing the second diver of the second group, who was some 15 msw further up the anchor line, it was obvious that Diver X was having problems. He did not respond appropriately to signals and would not accept a bailout regulator when it was offered. The second diver aborted his descent and attempted to control his and Diver X's ascent toward the surface. Unfortunately, at 24 msw, the buoyancy of Diver X's drysuit and equipment made this difficult. In addition, the second diver was passing his decompression ceiling and the difficult decision was made to release Diver X for an uncontrolled buoyant ascent.

Diver X surfaced face down and unresponsive 20 metres from the boat and was seen immediately. He was retrieved onto the boat deck and was noted to be apnoeic, apparently pulseless and to have red froth at his mouth. CPR was commenced with 100% oxygen from an Oxy-Viva™. After 1½ minutes he regained consciousness and complained of dyspnoea and lower-limb paralysis. In a very fortunate sequence of events, the nearest aeromedevac service was both close to the site and in a high state of readiness. Approximately 20 minutes after surfacing Diver X was retrieved by helicopter to the emergency department of the nearest major teaching hospital where he arrived approximately 40 minutes after leaving the water.

On arrival he was receiving assistance with respiration from a paramedic on a Laerdal self-inflating resuscitation bag with 100% oxygen. He was otherwise alert and complaining of dyspnoea, severe back pain, numbness with paralysis below the level of his ribcage and progressive weakness of his arms and neck. A presumptive diagnosis of severe DCS with the 'chokes', probable pulmonary barotrauma, arterial gas embolism, and salt-water aspiration was made. On-call hyperbaric unit staff were contacted as the accident occurred outside of usual operational hours.

On examination he was noted to have marked cutis marmorata, tachypnoea, chest signs consistent with aspiration, sinus tachycardia of 152 on ECG, unpalpable peripheral pulses and lower-limb arreflexia with marked lower-limb weakness. A supine chest X-ray demonstrated increased interstitial markings consistent with aspiration, but no pneumothorax or mediastinal gas. Arterial blood gases showed a metabolic acidosis, severe haemoconcentration, and a coagulopathy (Table 1).

Diver X's condition progressively deteriorated, the patient exhibiting increasing tachypnoea, dyspnoea and bulbar weakness at which point the decision was made to intubate. Ongoing resuscitation consisted of large volumes of crystalloid and colloid. One and a half litres of colloid and three litres of crystalloid were given in the first half hour. A lignocaine bolus of 1 mg/kg was administered soon after intravenous access was obtained and continued as an infusion of 4 mg/min for one hour and 2 mg/min for the next 47 hours. Arterial and central venous lines were inserted for haemodynamic monitoring and intermittent use of vasopressors. He was transferred for compression immediately the chamber was made operational.

Initial compression was to 283 kPa (2.8 ATA) – the maximum pressure capability at the facility – whilst ventilating with 100% oxygen. In view of the significant aspiration it was necessary to maintain sedation and paralysis for optimal ventilation. This prevented any assessment of clinical response to treatment, and a decision was therefore made to treat with an extended and modified US Navy treatment table 6 (Navy Department 1993). Two extensions were made at 283 kPa and the rate of ascent from 283 kPa to 192 kPa was halved with a stop at 242 kPa for five minutes. Total treatment time was six hours and 22 minutes. A further two and a half litres of colloid and three litres of saline were administered during the treatment. Endpoints for fluid resuscitation were haematocrit and urine output.

Table 1
Haematological and biochemical parameters on Day 1

Time	**1045**	**1245**	**1520**	**2140**
FiO_2 Intubated	1.0	1.0 @283 kPa	1.0 @283 kPa	0.4
pH	7.24	7.20	7.26	7.29
PaO_2 mmHg	113	317	490	86.5
SBE $mmol.l^{-1}$ (N -2.0–2.0)	-12.5	-7.5	-8.2	-6.9
Lactate $mmol.l^{-1}$ (N 0.5–1.6)	5.0	3.1	1.7	3.3
Hb $g.l^{-1}$ (N 115–165)	254	229	179	148
INR (N 0.8–1.1)	2.0			1.6
APTT secs (N 27–36)	105			51

FiO_2 – fractional inspired oxygen
SBE – standard base excess (37°C) at CO_2 = 40 mmHg
INR – international normalised ratio
APTT – activated partial thromboplastin time
N – normal range

Following compression Diver X was transferred to the intensive care unit where further invasive haemodynamic monitoring, consisting of a peripherally inserted cardiac output monitoring device (PiCCO™), was instituted in view of continued instability. Haemodynamic parameters suggested fluid overload and significant capillary leak prompting the administration of diuretics with subsequent improvement and weaning of inotropic support.

As he stabilised sedation was withdrawn to assess neurological response. By late evening he was rousable to verbal stimuli and appeared to have regained all motor movement. He was extubated the next day and although requiring modest levels of inspired oxygen, he was otherwise normal on examination with no obvious neurological deficits. He remained in intensive care for a further 36 hours for monitoring while lignocaine was continued, and was treated with two further compressions to 242 kPa, for 90 minutes with 10-minute ascent time, on consecutive days in view of minor leg pain that was intermittent and cramping in nature. There was no evidence of other pathology.

Ongoing issues requiring a prolonged hospital stay were hypoxia secondary to aspiration and urinary retention. The hypoxia slowly resolved over the next five days and required no further treatment. Urinary retention was noted after discharge from intensive care when the first attempt at catheter removal was made. In view of a past history of urethral stricture he was reviewed by the urologists who performed various investigations and concluded a probable decompression-related aetiology.

Resolution of inability to void occurred over the next few weeks and required no further intervention although he has had urology follow up throughout. However, he has had ongoing problems with hesitancy, constipation and pain in the sacral distribution. These are improving with time. This is despite full clinical neurological assessment suggesting complete recovery otherwise. No further neurological investigations were performed at the time in view of clinical recovery and the perception that further management would not be altered by an abnormal result.

He was formally assessed and reviewed in the Department of Diving and Hyperbaric Medicine four weeks after being discharged, at which time he was advised against diving in future and to return if there were further issues.

Diver X has subsequently, over 12 months, made a full return to his professional life and, rather controversially, to technical diving also. He has completed greater than 50 mixed-gas dives (deepest 100 msw) since the accident with no problems so far as reported to the authors (personal communication, Diver X, December 2004).

Discussion

THE DIVE AND THE CAUSE OF THE ACCIDENT

This accident was subsequently concluded to have been caused by CO_2 toxicity. CO_2 toxicity is a recognized hazard of both open-circuit and rebreather diving, though in rebreather diving there are potential causes other than hypoventilation, which is the main contributor in open-circuit dives. Problems such as exhausted scrubber material, incorrectly packed scrubber canisters with 'channelling' of gas around the material, and over-breathing the scrubber with consequent 'breakthrough' of unscrubbed CO_2 are all potential causes. CO_2 toxicity produces dyspnoea and headache early; and delirium, reduced consciousness, and finally unconsciousness as levels rise.[3]

In Diver X's case, the cause was idiosyncratic to his particular CCR. Assembly of the Mk15 rebreather prior to each use includes folding a rubber flange into place on the CO_2 scrubber–counterlung assembly known as the 'centre section'. This flange establishes the gas flow path through the CO_2 scrubber. If it is not correctly placed the flow may bypass the scrubber allowing CO_2 to build to toxic levels. Diver X had not folded the flange correctly during his preparations for this dive.

It is interesting that Diver X did not notice any problems until he reached the bottom at 105 msw. There are several potential reasons for this. First, it is usually recommended, but not universally practised, that a rebreather is breathed for five minutes prior to entering the water in order to unmask any problems such as the one described above. He did not conduct a significant pre-breathe at the surface prior to the dive. Second, the long descent on a deep dive involves significant physical exertion and any dyspnoea would almost certainly be attributed to that. Third, during a descent to 100 msw (1,114.3 kPa, 11 ATA) the CCR would have added 11 times the surface counterlung volume of uncontaminated diluent gas to the loop. This would have helped dilute the CO_2; an advantage that would have abruptly ceased on arrival at the bottom.

It is also notable that Diver X did not switch to open-circuit bailout when it became obvious to him that there must be a problem with the breathing gas in his CCR loop. In this regard, he even refused the assisting diver's offer of an open-circuit regulator. In reflecting on this later, Diver X observed that he felt so short of breath that he perceived he would drown if the CCR mouthpiece was removed, and he could not bring himself to do it. This is an important observation. Many CCR divers carry open-circuit bailout whose use will require a mouthpiece swap, and Diver X's experience suggests that assumptions about the ease of such swaps under circumstances of CO_2 toxicity may be flawed. This forms a strong argument for the use of combined rebreather/open-circuit mouthpieces where activation of a lever or similar can effect the swap without the mouthpiece being removed. For

completeness, it must be noted that Diver X acknowledges air to be an inappropriate choice of bailout gas for this very deep dive, though he maintains that this was not a factor in his failure to use it.

SEVERITY OF DCI AND ITS TREATMENT

Cases of severe multisystem DCI like this are rare. Although haematologic changes such as haemoconcentration and coagulopathy are reported from animal models of severe DCS, it is unusual to see these phenomena in humans.[4] Nevertheless, the severity of Diver X's case is not surprising given the circumstances of the dive. For an eight-minute bottom time at 105 msw, and utilising the gases specified earlier, the Proplanner™ decompression planner prescribes the decompression algorithm in Table 2. Even if we assume Diver X's ascent was conducted at the correct rate (9 m/min), it is obvious that he has omitted a very significant decompression obligation. Such situations do not arise in mainstream recreational diving, and it does seem likely that the increasing number of deep technical divers (and dives) will result in increasing numbers of similar cases.

Diver X enjoyed a remarkable recovery from very severe, progressive, multisystem DCI whose natural history untreated was probably towards death or permanent disability. It can therefore be surmised that his treatment was appropriate. Whilst it is impossible to draw conclusions about the efficacy of individual components of that treatment regimen, it is reasonable to at least speculate on the potential benefits of some of the circumstances and therapeutic strategies.

Table 2
Decompression for dive to 105 msw for 8 minutes prescribed by the Proplanner™ decompression calculator (nominal safety factor setting, PO_2 setpoint = 1.3). Ascent to the first stop and between stops is at 9 msw.min^{-1}. Travel time is in addition to the stop times shown

Stop depth (m)	Stop time (min)	Gas management
69	2	Diluent = trimix 10/57
51	2	Diluent = trimix 10/57
42	2	Diluent = trimix 10/57
33	1	Diluent = trimix 10/57
30	1	Change diluent to air
27	1	Diluent = air
24	1	Diluent = air
21	1	Diluent = air
18	1	Diluent = air
15	1	Diluent = air
12	3	Diluent = air
9	7	Diluent = air
6	4	Flush loop with 100% oxygen
4.5	24	100% oxygen
Total	**51**	

The contribution of Diver X's rapid evacuation to a definitive treatment facility cannot be underestimated. A longer evacuation or management at a lower-level facility without intensive care expertise may well have had disastrous consequences in this case in view of the requirement for endotracheal intubation and mechanical ventilation, invasive haemodynamic monitoring, serial haematological investigations, vasopressor support, and aggressive fluid resuscitation in order to stabilise him for recompression treatment. Such intervention is not available at many hyperbaric facilities. This, of course, is not to say that seriously ill DCI patients should not be managed at lower-level hyperbaric units in the absence of more comprehensive facilities. However, recompression of itself is not likely to stabilise the physiological derangements apparent 40 minutes after surfacing in Diver X's case, and outcome in such cases is likely to be poor if comprehensive care is not available.

The optimum recompression treatment for life-threatening multisystem DCI following massive omitted decompression is not known. Perhaps the only claim that can be made with some confidence is that it should occur as soon as possible, but even that is not (and probably never will be) definitively proven. Arguments can be made for and against the use of deep recompression treatments and heliox mixtures.[5] At the present time neither of these options are well supported by data, and the US Navy table 6 (involving administration of oxygen at 280 kPa) is the mainstay of treatment for more severe DCI.[5] In the present case, where treatment pressure was limited to 280 kPa and the only available treatment gas was oxygen, a table 6 was the logical choice. The table 6 was extended in a conventional fashion,[6] in keeping with the severity of the presentation and the inability to monitor clinical progress in a sedated, intubated and ventilated patient. The reduction of the treatment table ascent rate was imposed in view of anecdotal reports of deterioration during ascent in other cases that followed massive omitted decompression.[7]

The rationale for the use of lignocaine in the treatment algorithm is well described elsewhere.[8] Although use in DCS (arising from bubble formation in tissues or venous blood as distinct from arterial gas embolism) is speculative at best, this does receive qualified support from the UHMS Adjuvant Treatments Committee.[9]

Conclusions

As with many illnesses, prevention of DCS is better than cure. It is important that divers who are pushing technical diving boundaries are well trained, highly disciplined and vigilant to minimise the incidence of these episodes. Although the evolution of technical diving equipment may reduce failures and errors over time, there is risk of error in any system in which humans are involved. To reduce this possibility, technical diving training agencies should place great emphasis on maintenance of high course standards, and the impeccable credentials of their instructors.

As technical diving gains popularity and acceptance it behooves us as clinicians to be prepared to treat life-threatening DCI with a broad armamentarium of clinical interventions. This includes aggressive resuscitation in the initial presentation. There are some locations with on-site chambers and those who would argue that the best treatment for this condition is immediate recompression, but this option is not usually available. However, during a progressively deteriorating and life-threatening presentation, as in this case, compression alone is unlikely to be adequate and needs to be supplemented by scrupulous management of the 'ABCs'. It is imperative that this initial management be expeditiously instituted, before the initial compression if necessary.

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