1988;18 (2):53-60
2 McAniff JJ. *United States underwater diving fatality statistics 1970-79.* Washington DC: US Department of Commerce, NOAA, Undersea Research Program, 1981
3 McAniff JJ. *United States underwater diving fatality statistics 1986-87.* Report number URI-SSR-89-20, University of Rhode Island, National Underwater Accident Data Center. 1988
4 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 1. The human factor. *SPUMS J* 1989; 19 (3): 94-104
5 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 2. The environmental factor. *SPUMS J* 1990; 20 (1): 2-4
6 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 3. The equipment factor. *SPUMS J* 1991; 21 (1): 2-4
7 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine 3rd Edition*. Oxford: Butterworth/ Heinemann, 1992: 354-361
8 Lourey CJ. The cardiac reflexes revisited. *SPUMS J* 1981; 11 (Supp): 11-16
9 Kizer KW. Dysbarism in paradise. *Hawaii Med J* 1980; 39 (5):109-116
10 Farm FP, Hayashi EM and Beckman EL. *Diving and decompression sickness treatment practices among Hawaii's diving fishermen.* Sea Grant Technical Paper 86-01, University of Hawaii. 1986
11 Erde A and Edmonds C. Decompression sickness - a clinical series. *J Occup Med* 1975; 17; 324-328

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A PRELIMINARY REPORT ON A PROSPECTIVE RANDOMIZED, DOUBLE-BLIND, CONTROLLED STUDY OF OXYGEN AND OXYGEN-HELIUM IN THE TREATMENT OF AIR-DIVING DECOMPRESSION ILLNESS

Alison Drewry and Des Gorman

Abstract

The treatment of Australasian recreational divers with decompression illness using the United States Navy recompression algorithms has a high failure rate. Oxygen-helium gas mixtures may have some advantages over oxygen alone in such therapy, and consequently, a prospective randomized double-blind controlled study of oxygen and oxygen-helium in the treatment of air-based decompression illness has been initiated at the Royal New Zealand Naval Hospital in Auckland. Thirty patients have been studied in the first 4 months of 1992.

Introduction

The treatment of recreational air-divers with decompression illness (DCI) in Australasia is largely based on the "minimal recompression oxygen" tables promulgated by the United States Navy (USN) in 1965.[1] Although the USN, both initially and still, reports high resolution rates with the use of these treatments in its own naval divers,[2,3] this is not the current experience in injured recreational divers in Australasia. Failure rates (incomplete resolution of symptoms and signs) vary between 37% in Melbourne (1991; 100 divers),[4] 32% (neuropsychiatric sequelae) and 48% (abnormal EEG recordings) in Sydney (1987; 87 divers),[5] 54% in Auckland (1990; 125 divers)[6] and 54% in Adelaide (1988; 64 divers).[7] These failure rates do not vary significantly between facilities and the total number of patients treated and surveyed is large. It is also noteworthy that these failure rates exceed those reported in 1964 for both the 30 and 50 msw oxygen-nitrogen (air) recompression treatment tables.[8] Although these injured USN divers and their follow-up are not directly comparable with the nature and assessment of contemporary injured Australasian recreational divers,[4,5] the "high" failure rates reported in 1964[8] were used to justify the development of the 1965 alternatives (those in current use)[1] The same arguments then, used to introduce these "minimal recompression oxygen" tables can now be used to justify the development and testing of alternative therapies, at least for the treatment of recreational divers in Australasia.

The "minimum recompression oxygen" tables are a compromise between ambient pressure and oxygen toxicity, however the use of 2.8 bar inspired oxygen tension is nevertheless toxic to the injured brain.[9] An alternative is to use oxygen-helium mixtures at the same or greater ambient pressures, but such that the inspired oxygen tension is kept between 1 and 2 bar. The ideal inspired oxygen tension for treatment of DCI in vivo is 2 bar[10], but the optimal dose of oxygen to inhibit bubble-induced polymorphonuclear leucocyte (PMNL) accumulation (see below) has not yet been determined.[11] Although some studies of cardiopulmonary decompression illness in dogs and guinea pigs have failed to demonstrate any advantage,[12,13] oxygen-helium breathing has resulted in faster shrinkage of air bubbles in rat adipose tissue [14] and spinal cord white matter[15] than when either air or oxygen are breathed. This is explained by net gas flux being determined by both gas solubility and diffusion. Importantly, there is no evidence that oxygen-helium breathing causes air bubbles to grow in aqueous tissues such as

skeletal muscle and tendon; instead, such bubbles have been shown to shrink.[16] There is also anecdotal clinical support for oxygen-helium treatment of DCI which develops following air diving.[17]

It is now apparent that significant pathology in DCI is due to the "biochemical" effects that bubbles have and not solely to the long-espoused "mechanical", "compressive" or "occlusive" effects. Both DCI evolution and sensitivity in rabbits is dependent upon complement protein activity.[18-20] The brain dysfunction that follow air embolism of the brain in rabbits[21] and dogs[22,23] is largely due to an accumulation of PMNLS and a consequent fall in brain blood flow. Not surprisingly, measures adjuvant to recompression that may ameliorate these "biochemical" effects are being sought; at present, only lignocaine infusion is ready for human trials. Lignocaine may act either by stabilising membranes or by inhibiting PMNL accumulation and its toxicity or both. It is effective in cats and dogs with DCI both prophylactically [24] and therapeutically.[25,26] The benefit of lignocaine may actually be additive to that of hyperbaric oxygen.[26,27] Lignocaine has already been used in humans with DCI.[28]

Consequently, it was decided to start a prospective, randomised, double-blind, controlled study of oxygen-helium in the primary treatment of air-diving DCI, and a secondary study of lignocaine versus a placebo in DCI refractory to recompression (This will be the subject of a further report).

Methods

The trial has been approved by the Royal New Zealand Navy Human Ethics Committee and has begun at the Naval Hospital at Auckland. This facility treats about 40 to 50 recreational divers with DCI annually.[6] When database management and other protocol-specific issues are settled, it is intended to recruit both the Royal Australian Navy School of Underwater Medicine, at HMAS PENGUIN in Sydney, (about 25 to 30 divers a year)[5] and the Royal Adelaide Hospital Hyperbaric Medicine Unit (about 25 to 30 divers annually)[7] into the study, as both of these facilities are capable of oxygen-helium recompression.

All recreational divers with DCI after air diving who present for treatment, regardless of the duration of their signs and symptoms, are compressed to 2.8 bar absolute. All receive intravenous fluids (1 litre of normal saline over 1 hour and then 1 litre 4 hourly), but no chemotherapy. These patients are randomly allocated (Alpha and Bravo cards) to Group Alpha (50/50 oxygen-helium) or Bravo (100% oxygen). The randomisation is stratified into those presenting within 48 hours and those 48 hours or more after the onset of their symptoms. Neither the attendant medical officer nor the patient is made aware of the allocated group. The allocated gases are breathed on arrival at 18 m and for a further 45 minutes. If this produces an 80% or greater improvement (see scoring system below), then a USN Table 6[29] pressure profile is completed, with extensions if the response is less than complete. Group Alpha breathes oxygen-helium instead of 100% oxygen and has no air breaks. Group Bravo has a standard USN Table 6.

However, if the initial period produces a less than 80% improvement, then the patient is compressed to 30 msw and given the pressure profile of USN Table 1A.[29] Group Alpha patients breathe 50/50 oxygen-helium and Group Bravo breathe 50/50 oxygen-nitrogen.

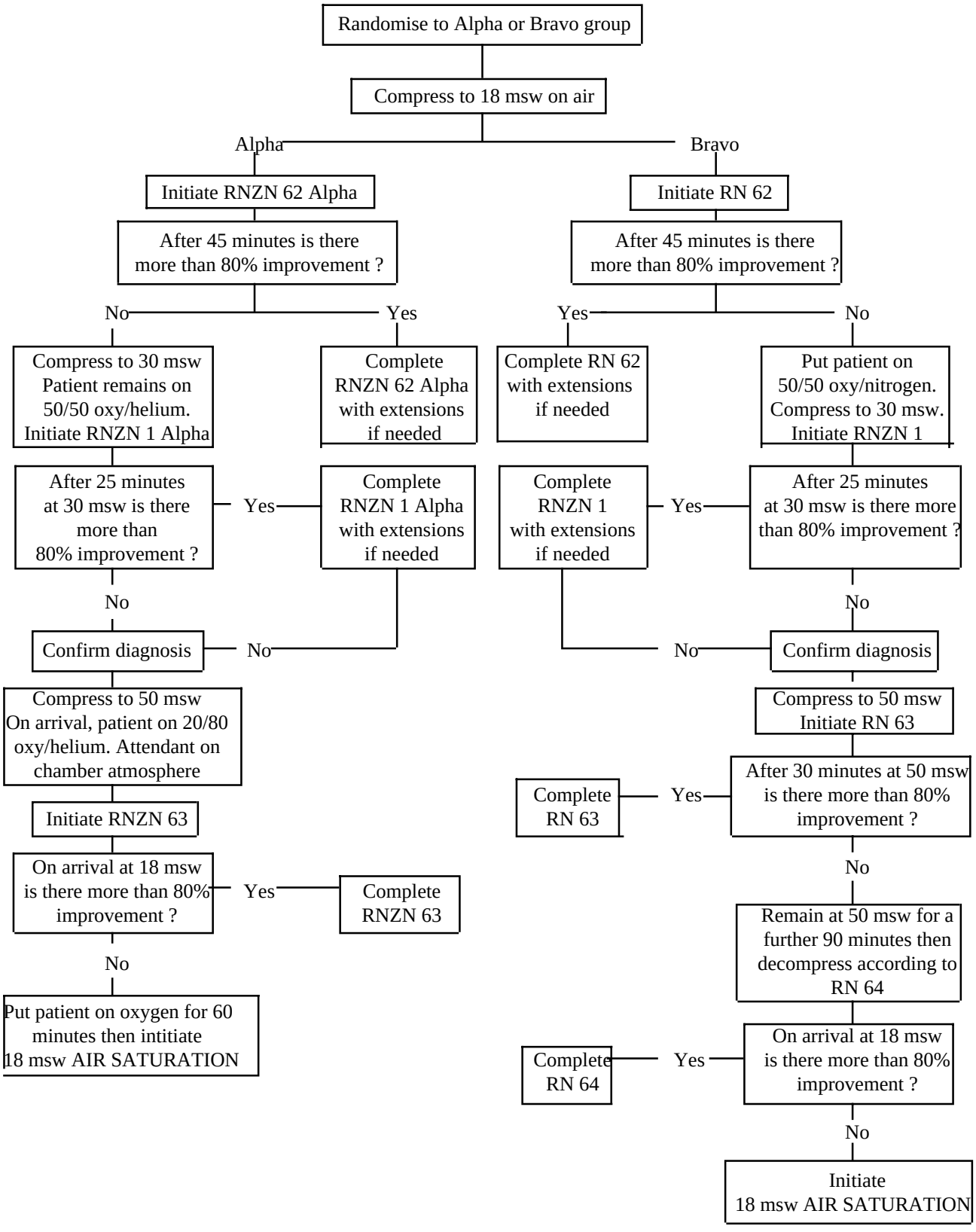
If the response to this is less than 80% improvement, then the patient is compressed to 50 msw and given the pressure profile of USN Table 6A[29] or RN Table 64.[30] Group Bravo have a standard Table USN 6A or RN Table 64, breathing air and oxygen. Group Alpha patients are limited to USN Table 6A and breathe 20/80 oxygen-helium and 50/50 oxygen-helium instead of air and oxygen. Repeat treatments, made necessary by relapsing or persistent problems, are conducted according to the Royal Adelaide Hospital protocol of 18 m for 60 minutes on oxygen with a 30 minute decompression (RAH 18/60/30). Group Alpha have 50/50 oxygen-helium, Group Bravo breathe 100% oxygen. Treatments are given daily until the problems resolve or two successive treatments do not produce any sustained improvement. A treatment algorithm for the study is shown in Figure 1.

Individual informed consent is obtained from each patient. In the event of any patient being unconscious or confused, consent for participation in the trial is sought initially from a first-degree relative. Women who are known or suspected to be pregnant are excluded from the trial.

Response is based on history and examination findings; and in particular, visual analogue scales for pain and sensation,[31] serial-7 performance (time and errors), memory of a Babcock sentence (number of trials), muscle power percentages and sharpened Romberg testing (percentage time stable). Outcome is assessed clinically as this is still the most sensitive measure available.[32] Assessments are made during the first treatment (frequency of compression to 30 and 50 msw and cost), after the first treatment (signs and symptoms), at the time of discharge from hospital (number of treatments, adjuvant care, signs and symptoms), at one and six months and one year later (signs and symptoms).

Data are stored in a dBase IV database and are subject to sequential analysis by an independent blinded observer. The study will be stopped either when the difference in outcome at one month reaches a significance level of 0.05 or group sizes of 50 are exceeded for both groups (and providing the groups are directly comparable). Relative group composition and outcome are tested by t-test (parametric data) and by calculation of Fisher's exact p value. When multiple simultaneous comparisons are made, the Bonferroni correction is applied.[33]

RNZN HYPERBARIC UNIT
TREATMENT FLOW CHARTS



NOTES

All follow up treatments are to be on Table 18:60:30.

- Follow up treatments are to be on the gas they were treated on.

Follow up treatments are to continue until there is no sustained response to two successive treatments.

Interim results

Thirty recreational divers have been treated for DCI at Royal New Zealand Naval Hospital in the first 4 months of 1992 and have participated in the study. No significant differences are manifest to date and the small groups are not yet directly comparable.

Discussion

The need to test alternative treatment regimens for recreational divers in Australasia has been well established by an unacceptably high local treatment failure rate.[4-7] A potential role for oxygen-helium in lieu of 100% oxygen in recompression is shown in vivo,[14-16] and clinically, although anecdotally.[17] The trial described here should demonstrate any utility for oxygen-helium treatment in injured Australasian air-breathing divers.

The reasons for the difference in outcome between Australasian divers[4-7] and USN divers[2,3] when treated for DCI on USN algorithms[29] are uncertain (perhaps the delay prior to treatment is critical[32]), but are not the subject of and will not be answered by this study.

References

1 Goodman MW and Workman RD. *Minimal recompression oxygen breathing approach to treatment of decompression sickness in divers and aviators.* USN Experimental Diving Unit Research Report 5-65, Washington, DC, 1965.

2 Workman RD. Treatment of bends with oxygen at high pressure. *Aerospace Med* 1968; 39: 1076-1083.

3 Green JSW, Tichenor J and Curley MD. Treatment of type I decompression sickness using the US Navy treatment algorithm. *Undersea Biomed Res* 1989; 16: 465-470.

4 Weinmann M, Tuxen D, Scheinkestel C and Millar I. Decompression illnesses. 18 months experience at the Alfred Hospital Hyperbaric Unit. *SPUMS J* 1991; 21(3): 135-143.

5 Gorman DF, Edmonds CW and Parson DW, et al. Neurologic sequelae of decompression sickness: a clinical report. In: Bove AA, Bachrach AJ, Greenbaum LJ Jr, Eds. *Underwater and hyperbaric physiology IX*. Undersea and Hyperbaric Medical Society, Bethesda, 1987, pp 993-998.

6 Brew SK, Kenny CT, Webb RK and Gorman DF. A factorial analysis of 125 diving accidents treated at HMNZS PHILOMEL. *SPUMS J* 1990; 20(4): 226-230.

7 Gorman DF, Pearce A and Webb RK. Dysbaric illness in South Australia, 1987. *SPUMS J* 1988; 18(3): 95-101.

8 Rivera JC. Decompression sickness among divers: An analysis of 935 cases. *Milit Med* 1964; 129: 31-334.

9 Holbach KH and Caroli A. Oxygen tolerance and the oxygenation state of the injured human brain. In: Trapp WG, Bannister EW, Davidson AAJ and Trapp PA, Eds. *Proceedings of the 5th International Hyperbaric Congress*. Burnaby: Simon Fraser University, 1974, pp 350-361.

10 Leitch DR and Hallenbeck JM. Oxygen in the treatment of spinal cord decompression sickness. *Undersea Biomed Res* 1985; 12: 269-289.

11 Zamboni WA, Roth AC, Russell RC, Suchy H and Kucan J. The effect of hyperbaric oxygen treatment on the microcirculation of ischemic skeletal muscle. *Undersea Biomed Res* 1990; 17(Suppl): 26.

12 Catron PW, Thomas LB, Flyn ET, McDermott JJ and Holt MA. Effects of He-O_2 breathing during experimental decompression sickness following air dives. *Undersea Biomed Res* 1987; 14: 101-111.

13 Lillo RS, MacCallum ME and Pitkin RB. Air vs He-O_2 recompression treatment of decompression sickness in guinea pigs. *Undersea Biomed Res* 1988; 15: 283-300.

14 Hyldegaard O and Madsen J. Influence of heliox, oxygen and N_2O-O_2 breathing on N_2 bubbles in adipose tissue. *Undersea Biomed Res* 1989; 16(3): 185-194.

15 Hyldegaard O, Moller M and Madsen J. Effect of He-O_2, O_2 and N_2O-O_2 breathing on injected bubbles in spinal white matter tissue. *Undersea Biomed Res* 1991; 18: 361-371.

16 Hyldegaard O and Madsen J. Effect of heliox breathing on air bubbles in aqueous tissues after decompression. *Proceedings of the XVII annual meeting on diving and hyperbaric medicine*. European Undersea Biomedical Society, 1991: 75-80.

17 Douglas JDM and Robinson C. Heliox treatment for spinal decompression sickness following air dives. *Undersea Biomed Res* 1987; 15: 283-300.

18 Ward CA, Koheil A, McCulloch D, Johnson WR and Fraser WD. Activation of complement at the plasma-air or serum-air interface of rabbits. *J Appl Physiol* 1986; 60: 1651-1658.

19 Ward CA, McCulloch D and Fraser WD. Relation between complement activation and susceptibility to decompression sickness. *J Appl Physiol* 1987; 62: 1160-1166.

20 Ward CA, McCulloch D, Yee D. Stanga D and Fraser WD. Complement activation involvement in decompression sickness of rabbits. *Undersea Biomed Res* 1990; 18: 51-66.

21 Helps SC and Gorman DF. Air embolism of the brain in rabbits pretreated with mechlorethamine. *Stroke* 1991; 22: 351-354.

22 Hallenbeck JM, Dutka AJ and Tanishima T, et al. Polymorphonuclear leucocyte accumulation in brain regions with low blood flow during the early postischemic period. *Stroke* 1986; 17: 246-253.

23 Dutka AJ, Kochanek PM and Hallenbeck JM. Influ-

ence of granulocytopenia on canine cerebral ischemia induced by air embolism. *Stroke* 1989; 20: 390-395.
24 Evans DE, Kobrine AI and LeGrys DC, et al. Protective effect of lidocaine in acute cerebral ischaemia induced by air embolism. *J Neurosurg* 1984; 60: 257-263.
25 Evans DE, Catron PW and McDermott JJ, et al. Therapeutic effect of lidocaine in experimental cerebral ischaemia induced by air embolism . *J Neurosurg* 1989; 70: 97-102.
26 Dutka AJ. Therapy for dysbaric central nervous system ischaemia; adjuncts to recompression. In: Bennett PB and Moon RE, Eds. *Diving accident management.* Bethesda MD, UHMS, 1990, pp 222-234.
27 McDermott JJ, Dutka AJ, Evans DE and Flynn ET. Treatment of experimental cerebral air embolism with lidocaine and hyperbaric oxygen. *Undersea Biomed Res* 1990; 17: 525-534.
28 Drewry A and Gorman DF. Lidocaine as an adjunct to hyperbaric therapy in decompression illness: A case report. *Undersea Biomed Res* 1992; 19: in press.
29 *USN Diving Manual*. NAVSEA 0994-LP-001-9010.
30 BR 2806. *Diving Manual* D/DNW 102/4/31.
31 Scott J and Huskisson EC. Graphic representation of pain. *Pain* 1976; 2: 175-184.
32 Moon RE and Gorman DF. Treatment of the decompression disorders. In: Bennett PB and Elliott DH. *The physiology and medicine of diving. 4th edition.* Balliere-Tindall, London, 1992, in press.
33 Wallenstein S, Zucker CL and Fleiss JL. Some statistical methods useful in circulation research. *Circ Res* 1980; 47: 1-9.

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NATURAL OBSERVATIONS OF DIVING INCIDENTS

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Introduction

Safety has always been a concern for the recreational diving industry. While the major accident rate is very low compared to other sporting groups,[1,2] the consequences of an underwater accident can often be more serious than injuries on land. At a time when there is an increased willingness for the public to sue sports coaches and administrators for any alleged breach in their "duties of care", [3,4] the diving industry needs to look carefully at cost effective methods of improving safety. While formal legislation[5] or industry Codes of Practice[6] may provide frameworks for safety, other informal methods of preventing accidents should also be examined for their potential contribution.

The term "diving incident" has been used by Acott and his colleagues[7] to describe "An error by a diver, or a failure of his or her equipment to function properly. The error or failure could have led to more serious consequences, had it not been detected or corrected in time". An incident has the makings of an accident, where things actually do go wrong, but does not necessarily lead to an accident.

While the term "diving incident" is relatively new, scuba instructors have appreciated the importance of early recognition in accident prevention for some time.[8,9] Reviews of diving accidents consistently highlight some common factors contributing to the accident scenarios. These include medical and psychological factors, dangerous environmental conditions and equipment difficulties.[10-14]

Some of these problems may be difficult for a dive supervisor to overcome (e.g. undetected faulty personal equipment), whereas other problems might be prevented with detailed dive briefings and greater awareness of areas where accidents are likely to occur. For example, after reviewing 264 Japanese diver fatalities Mano and Shibayama[15] concluded that poor diving technique and reckless diving were the main causes of fatalities. They also noted that many accidents they investigated could be predicted on the basis of their non-existent or inadequate dive planning, and only a few accidents occurred that could not have been prevented.

The Diving Incidents approach to accident prevention suggested by Acott and his associates[7] focuses attention on those areas of activity where problems are likely to arise. In their pilot study they asked divers to record any incidents they had observed during a dive on a questionnaire report form. Of the total of 69 incidents reported, 36 occurred during the dive itself. A further 15 incidents occurred during preparation for the dive, and another five during entry.