incident the diver was experienced in ditch-and-recovery from his work as a scuba instructor. If the evidence is correct that there was no failure of his air supply, there must have been another, not identified, reason for his actions. While there was entrapment of his buoyancy vest's belt in the water jet-gun this machine had been turned off about the time he died. Possibly he misjudged the danger of his situation and inhaled water while he was attempting to ditch his equipment.

Acknowledgements

This report would not be possible without the support of many people. In particular the on-going support of the Justice/Law/Attorney General's Department in every State, continues to be an essential element of this safety project.

Project Stickybeak Divedata Databank

The objective of this on-going project is to identify factors which influence the safety of divers, whether recreational or commercial divers. Reports are requested concerning incidents of all types and severity, particularly where there has been a successful outcome. MEDICAL CONFI-DENTIALITY is given to every communication received. Address for correspondence:-

> Dr D Walker P O Box 120, Narrabeen New South Wales 2101 Australia.

DEVELOPMENT AND USE OF THE OXYGEN-BREATHING MINIMAL-RECOMPRESSION TREATMENT OF DECOMPRESSION SICKNESS

Geoffrey Gordon

Introduction

When man ventures into a hyperbaric or hypobaric environment, his excursions are occasionally complicated by the development of decompression sickness (DCS) and arterial gas embolism (AGE). To treat the occurrence of these illnesses, recompression therapy is used either according to a standard treatment table or to a depth that brings relief. This approach to treatment is not based on experimental evidence, but on the empirical application of theoretical concepts.^{1,2} Old protocols were superseded when it was felt that the results were unsatisfactory, rather than when case analysis indicated poor treatment outcomes. Analysis of the effectiveness of treatment regimens needs to be conducted if, in any meaningful way, we are going to be able to improve our effective treatment opinions. That a solution will be developed to benefit every case is a naive idea, but have the current "minimal recompression oxygen breathing tables", developed in 1965, filled a void, or are they just another attempt at treating a disease process that is still incompletely understood?

The history of treatment tables

It was not until 1847 that Pol and Watelle³ first recognised that there was a relationship between the onset of DCS symptoms and the depth, bottom time and rate of ascent. This was 28 years after Siebe developed the first practical deep-sea diving outfit and 6 years after the production of the first large capacity compressors that permitted large numbers of men to work at raised ambient pressures. Although this work established recompression as the primary treatment modality, the manner of its application was unclear. It was not until 1878 that Bert⁴ demonstrated that liberation of nitrogen in the form of bubbles was the cause of DCS. He also recommended recompression and went on to expound that treatment with oxygen should be effective. No decompression rates were specified, and as pure oxygen was both scarce and very expensive it was little used. In 1897 Zuntz⁵ utilised oxygen in conjunction with recompression to increase the gradient for nitrogen elimination and hasten bubble resolution. However, due to the development of oxygen toxicity, the use of this adjunct proved unpopular and was not widely used again for many years. Air recompression therapy for DCS was subsequently developed.

In 1937 Behnke and Shaw⁶ re-investigated the use of oxygen in the treatment of DCS. They hoped to utilise the increased gradient for nitrogen elimination to improve treatment outcomes. In experiments using a dog DCS model, they observed that severe cardiopulmonary DCS responded well to recompression to 30 msw regardless of whether air or oxygen was breathed. On subsequent decompression however, those treated on air had recurrences of cardiopulmonary DCS of pretreatment severity. Those treated with oxygen showed a minimal return of symptoms, indicating better inert gas clearance when an oxygen atmosphere was breathed. Yarbrough and Behnke⁷ two years later, documented a 50% recurrence of symptoms in divers treated by recompression to depth of relief plus 10 metres of seawater, the procedures published in the US Navy (USN) Diving Manual of 1924. This protocol probably did not achieve resolution of all the gas in bubble form despite the pressure applied. In an attempt to achieve complete bubble elimination, they empirically developed guidelines limiting recompression depth to 50 msw with a minimum time at this depth of 30 minutes. In modifying the Haldanian type decompression, 100% oxygen was breathed from 18 msw to the surface. The process of gas diffusion from bubbles and tissues was thought to be slow, and so administration of

oxygen at increased pressure was used to accelerate this process. Initial results were encouraging, and attributed to the elimination of gas mixtures during the ascent from 18 msw.

By 1945 the performance of these new treatment tables "appeared" no better than the results obtained using the US Navy 1924 protocol. As a result of this "apparent" failure, a series of experiments conducted by Van der Aue et al.8 led to the development of formal treatment tables embodying the Yarbrough and Behnke principles. These tables were subsequently promulgated as USN Tables 1, 2, 3 and 4 in the USN Diving Manual of 1945 and remained in use becoming standard in the USN, several foreign Navies and many commercial worldwide for the next 20 years.² It is interesting to note that these widely accepted treatment procedures were based upon a study involving only 33 subjects and that some of the individual treatment tables were based on as few as 6 subjects. The Royal Navy (RN) developed their equivalents in the RN Tables 52, 53, 54 and 55 a few years later.

Problems with the current air treatment tables

In 1947, Van der Aue and his colleagues analysed 113 cases treated with these "new" air tables and found a first treatment failure rate of 5.3%.9 It was clear that these air treatment tables showed promise. Unfortunately these tables were not subjected to further outcome analysis until 1962 when Slark¹⁰ retrospectively reviewed the treatment of 137 cases. In this study the overall first treatment failure rate had risen to 21.5%. Slark considered this unacceptable, and further pondered on the likelihood that the nitrogen uptake occurring during treatment contributed to the observed failure rate. He did see something positive in the air tables however, postulating that the prolonged 9 msw soak was the strength of USN tables 3 and 4. In recommending the development of new tables, Slark foresaw these having a longer time at lower pressures, and further suggested incorporating an even release of pressure during the ascent.

In 1964 Rivera¹¹ reviewed the treatment of 935 cases of DCS, spanning the period 1947 to 1963. The failure rate of tables 1 and 2 during initial treatment was 5.6% and considered satisfactory. These two tables were used to treat pain only DCS. The treatment of serious DCS with tables 3 and 4 had a failure rate during initial treatment of 25%. This overall figure is perhaps a little misleading as the failure rate rose dramatically over the review period, from 16.1% in 1947 to 46% in 1963. This increase was paralleled by an escalation in the number of civilian divers being treated and a concomitant increase in divers presenting following marked departures from accepted diving practice. In 1963, some 66% of the divers treated had developed DCS following such non-standard dives, and this sub-group accounted for almost all of the 46% initial treatment failures. There were no failures using USN tables 3 and 4 on Navy divers, who invariably received early recompression, unlike the civilian divers, who usually presented following long delays.

Goodman and Workman¹² reviewed the cases treated with air tables during 1964, and noted a similar overall failure rate of 25%, with USN tables 3 and 4 again having an initial failure rate of 47%. This group similarly comprised civilian divers with long delays to treatment following nonstandard exposures with omitted decompression.

The construction industry has traditionally utilised air recompression in cases of DCS occurring at the workplace, and have developed their own code of practice. Analysis of the effectiveness of these procedures in the various large projects has not been undertaken in this review, but air recompression has been used in two recent large projects in Hong Kong and Singapore with acceptable results.¹³

These figures, and the increasing number of civilian casualties, necessitated a fresh look at the problem of treatment DCS and the development of more effective recompression tables for what promised to be an epidemic of DCS in the 1970s.

Development of the minimal recompression oxygen tables

Goodman¹² in 1964 defined what he felt to be the fundamental route to DCS symptom remission viz:-

1 Compression to reduce bubble volume and radius in order that the intensity of the tissue reaction be reduced.

2 Termination of the focal ischaemia brought about by the endothelial irritation.

The latter, he surmised, could best be achieved by exploiting the collateral blood supply after hyper-oxygenating the patient, effectively establishing a metabolic detour around the occluded vessels. Hyper-oxygenation would also provide the maximum gradient for the elimination of inert gas from within the bubbles and tissues. Compression has a limited ability to RESOLVE bubbles, so bubble compression was relegated to a position of less than first order significance. The risk of developing DCS from treatment was also essentially eliminated with oxygen therapy.¹²

Goodman and Workman, from the USN Experimental Diving Unit, were given the task of revising the treatment of DCS, and in late 1965 their landmark report¹⁴ was released.

Schedule development

Noting, as did Slark¹⁰, that the 9 msw soak was probably the strength of the USN tables 3 and 4, Goodman and Workman conducted a number of trials at this shallower depth. The first provisional format was empirical in its design. Patients were taken to 10 msw breathing 100% oxygen and assessed after 10 minutes. If all symptoms had been relieved, then treatment was continued at this depth as indicated by the dotted line in Figure 1. If relief was not complete in this time, then the patient was taken to 18 msw and decompressed according to the 18 msw schedule shown in Figure 1.

In a few instances, the decompression from 18 msw was interrupted by spending 30 minutes at 9 msw before continuing the decompression to the surface. There were no failures with this provisional format.

Following analysis of the cases so treated, patients were subsequently compressed directly to 18 msw, eliminating the 10 msw trial. This resulted in treatment times of between 100 and 130 minutes.

Retrospective analysis showed that both the full treatment depth and the actual time breathing oxygen, were significantly related to treatment adequacy, leading Goodman and Workman to define a Minimum Adequate Time of 30 minutes oxygen breathing at 18 msw with a total time breathing oxygen of 90 minutes. New schedules were developed reflecting treatment durations of 1.5 and 3 times this minimum adequate time and designated the 2nd provisional format (Figure 2). If relief occurred within 10 minutes at 18 msw, 130 minutes of oxygen breathing was administered (Figure 2 upper). If relief was not complete however, then 285 minutes was spent on oxygen (Figure 2 lower).

This second format also proved very effective, but was further refined to reduce the risk of acute oxygen toxicity by the inclusion of air breaks interrupting the periods of oxygen breathing. The resulting tables are the familiar USN tables 5 and 6 (RN tables 61 and 62).

In the cases receiving adequate treatment, 50 cases in all, there was a 2% failure of initial recompression which compared favourably with the overall air tables initial failure rate of 15%. Of these 50 cases 28 (56%) met the criteria for treatment with USN tables 3 or 4 (serious cases) and these had an initial failure rate of 3.6%, a marked improvement over tables 3 and 4, which historically accounted for 27% of all treatments and had a failure rate of up to 47%. Better results were thus achieved with sicker divers. Oxygen toxicity was not a problem as it had been with Bert in 1878.

In attempting to convince physicians that deeper was not always better they noted that the law of diminishing returns becomes relevant for bubble diameter before it does so for bubble volume (Figure 3). Thus in going from 20 msw to 50 msw little is achieved in diameter reduction, but there is an obligation of additional inert gas uptake as gas mixtures





First Provisional Format (Drawn from data by Goodman and Workman¹⁴)

FIGURE 2



Second Provisional Format Drawn from data by Goodman and Workman¹⁴)





Effect of compression on the pN_2 gradient from a bubble using air and 100% oxygen (Redrawn from Pilmanis²⁷)

Volume and diameter changes under varying overpressures

must be breathed below 20 msw.

The question as to whether the treatment failures occurring after recompression on the air tables was due to a relapse of DCS or whether they represented freshly provoked DCS due to additional inert gas uptake, has never been satisfactorily answered. What is known though, is that the gradient for inert gas (nitrogen) elimination while the patient is breathing oxygen at the maximum safe pressure is large. At 18 msw the partial pressure (pp) of nitrogen in the bubble is approximately 2,105 mm Hg while the pp of nitrogen in the blood of a patient breathing 100% oxygen will be 0 mm Hg, creating a massive gradient for nitrogen egress and avoiding the potential problems of bubble growth due to additional inert gas uptake. Breathing 100% oxygen results in a nitrogen elimination curve with the partial pressure gradient of nitrogen between the tissues and the bubbles increasing with time as the oxygen is breathed, driving the nitrogen out of the bubbles, reducing their size until they collapse. In contrast, during treatment with air, the gradient for nitrogen elimination decreases with time and there is additional nitrogen uptake, nitrogen that sooner or later must be off-loaded (Figure 4).

Goodman and Workman¹⁴ saw benefit in the even release of pressure during decompression, rather than the staged ascent of the air tables. This avoidance of sudden pressure reductions was seen to be an important adjunct and had been previously recommended.¹⁰ The time savings of the oxygen tables were of major significance. A USN table 6 (RN table 62) takes 4 hours 45 minutes to complete, while a USN table 4 takes 38 hours 11 minutes. This time saving, combined with a better treatment efficacy, had considerable equipment and personnel advantages, changing what was often a marathon treatment effort into a tolerable and effective therapy.

These new tables met with instant favour when first released in November 1965. But have they stood the test of time, and are they as effective as the first 50 cases treated would suggest?

Effectiveness of the oxygen tables

Following the inclusion of these treatment tables into the USN Diving Manual in 1967, Workman¹⁵ reported on his experience of using these in 150 cases, 40 of whom were civilian divers. First treatment failures in this series was 15%, falling to 5% after the second treatment. In the military divers, initial failure was 7%. Consistently good treatment results were documented and at a reduced time obligation and staff commitment. Workman did concede that these tables would not benefit 100% of divers, especially in view of the increasingly inadequate decompression seen in the civilian divers developing DCS. However, his initial treatment relief with the oxygen tables of 85%, compared with 55% on USN tables 3 and 4, was indeed encouraging.

Three divers not improving at 18 msw in this series, were further compressed to 50 msw with no additional benefit.¹⁵ Bornman¹⁶ in the same year stated that although an increase in pressure could relieve the pain associated with mechanical pressure and ischaemic obstruction, pressure per se would not aid bubble elimination. He concluded that the use of oxygen in the new tables was very important.

Lambertson¹⁷ in noting the initial success with these new tables, concluded that extension of oxygen tolerance at pressure would provide the ultimate improvement in the therapy of DCS. His initial studies showed that the total tolerated oxygen dose at 20 msw could be extended to 20 hours by periodic respite from exposure to these toxic oxygen levels.

In other series, Erde and Edmonds¹⁸ noted an initial treatment failure of 13-15% if the delay was greater than 3 hours while Davis¹⁹ noted a 10% failure rate, many of these occurring following considerable delays before treatment. Melamed and Ohry²⁰ in 35 patients documented a 10% failure of initial therapy, while Hunt²¹ recorded 100% success in the initial treatment of 18 divers (78% civilian) with DCS, the best figures achieved in any study. Bornman's²² experience in treating Navy divers was a 3.6% failure following the institution of early recompression.

Kizer²³ reviewed 50 cases of DCS treated following delays in excess of 12 hours, again most of these divers having marked departures from accepted diving practice. Forty-seven of these patients were treated with USN table 6 (RN 62) half of these treatments being extended. The standard table had an initial failure rate of 20%. In the severe cases, when the table was extended, the failure rate rose to 37%. Overall though, Kizer felt that 92% had complete or substantial recovery following initial treatment. These results are similar to those achieved in the other series, but Kizer noted that the oxygen dose frequently needed to be increased to achieve this result.

Green and Leitch²⁴, reviewing 20 years' experience analysed 179 cases with severe DCS. Fifty-six (30%) were treated with air tables and 123 (70%) with the short oxygen tables. Overall, the oxygen tables performed better, but particularly if the delay to treatment was greater than 12 hours. Some cases presented at between 10 and 17 days after symptom onset and made full recoveries following treatment (Table 1). The USN¹⁶ found the oxygen tables to be superior if the delay to treatment exceeded as little as 5 hours. Looking at the results achieved with the oxygen tables as a separate group, RN table 61 (USN 5) had an 18% relapse rate following initial treatment, while the RN table 62 (USN 6) had a relapse rate of 3%.²⁴ Green and Leitch²⁴ restated that it is inappropriate to treat severe cases on an RN table 61, as this treatment proved ineffective in severe cases and had a high relapse rate. With the oxygen tables firmly established as the mainstay of treatment, they concluded that grounds exist for the removal of the RN table 61 (USN 5) as a treatment option, relying instead on the longer oxygen table. This would reduce treatment errors and improve outcomes, a feeling that has been expressed by others.25

The same authors, but in a different study²⁶, reviewed the treatment of severe DCS that was not responding at 18 msw. In the 24 cases, almost universal ineffectiveness was

TABLE 1

PER CENTAGE CURE RATES

| < 12 | hrs delay | > 12 hrs delay | Overal |
|---------------|-------------|-------------------------------|--------|
| Air Tables | 50% | 7% | 32% |
| Oxygen Tables | 58% | 58% | 46% |
| (from o | lata by Gre | en and Leitch ²⁴) | |

documented following further compression, usually to 50 msw. Those showing some response had already demonstrated some improvement at 18 msw. They concluded that serious cases of DCS are not materially improved by further compression to a greater depth. Further, 6 cases still deteriorating at 50 msw were no better at 70 msw, reinforcing the belief that compression to a greater depth does not necessarily halt or prevent deterioration. The animal data also supports the doubtful efficacy of increasing the pressure to greater than 18 msw in the treatment of serious DCS.^{1,27}

While most commonly seen in divers and caisson workers, DCS also occurs in aviators during their exposure to sub-atmospheric pressure. In a review of 145 cases of aviators bends, Davis et al²⁸ documented a recurrence rate of 22% in those treated with the air tables, while in contrast, only 1% of those treated with the oxygen tables relapsed. The USAF used modified tables 5 and 6, in that they continued the 20 min oxygen/5 min air sequence at 9 msw, rather than the 60 min oxygen/15 min air as promulgated in the USN tables for this depth. The total time on oxygen remained unaltered. As with diver DCS, the treatment of stricken aviators with the oxygen tables was very successful, even following delays of many hours.

Studies have suggested that treatment at 18 msw for AGE may also be effective especially if a concomitant decompression debt had been incurred. Traditionally, patients at 18 msw on oxygen not showing improvement have been further compressed to 50 msw on air. This makes it difficult to make a true assessment as the effectiveness of RN 62/USN 6 in treating AGE. From the data^{2,15,21,29} it is not possible to say that 50 msw is better than 18 msw. It seems likely that most cases of AGE will do equally as well at 18 msw on oxygen as they will at 50 msw on air. If early recovery does not occur, time seems to be the major factor, regardless of the pressure at which treatment occurs, hence extension at 18 msw is indicated. In the studies reviewed, USN 6A/RN 63 showed the best performance. However following a delay before treatment of greater than 6 hours, proceeding to 50 msw was no more effective than staying at 18 msw. One omission that does stand out is that maximum use was not made of extending the tables at 18 msw as is

permitted.^{30,31} Conclusion

Human beings did not evolve for a marine existence, but they continue to venture into this and other alien realms with attendant morbidity and mortality. Before the 1950s, the divers presenting for treatment of compressed air illness were primarily military or commercial. They presented early and with predominantly "pain only" DCS. This has been entirely reversed since the late 1960s by the large number of less disciplined and unsupervised sport divers who not only present later, but have a preponderance of the more sinister neurological manifestations of DCS.^{11,15,24,26} The treatment protocols used to treat this civilian population in the 1980s have been satisfactory, but it must be realised that they were designed in the military for a totally different population.

Because the therapeutic value of compression therapy is so self evident controlled studies in man have not been done. Davis in 1935 further commended that "No one who has seen the victim of compressed air illness, gravely ill or unconscious, put back into a chamber and brought back to life by the application of air pressure, will forget the extraordinary efficiency of recompression or will be backward in applying it to a subsequent case of illness".³² In contrast to this, Saumarez and his colleagues³³, who were without recompression facilities, successfully treated a case of severe neurological DCS with oxygen at the surface.

Pressure and oxygen have become the cornerstones of therapy and studies have been directed at identifying the optimum pressure, oxygen dose and time that these variables should be applied to achieve maximum effectiveness. Some centres believe that pressure is the all important variable, but the Boyle's Law effect is not nearly as dramatic when viewed from the standpoint of the bubble as opposed to the more usual bubble volume (Figure 3). In an animal model of spinal cord DCS treated with a constant 2.0 bar pp oxygen, recovery was not significantly altered by recompressing deeper than 20 msw.¹ This, and other studies, support the belief that going deeper brings little further benefit. In a similar study, pressure was held constant at 5.0 bar and the pp of oxygen was varied between 1.0 and 3.0 bar.34 Treatment with a pp of oxygen of 2.0 bar (10 msw) achieved the best results suggesting that the optimum treatment of DCS was at 10 msw on 100% oxygen. DCS has been reported following treatment with hyperbaric oxygen³⁵, so potentially at least, oxygen as well as inert gas, can cause a DCS like syndrome. The use of oxygen and pressure in the manner empirically derived by Goodman and Workman¹⁴ is gradually being supported by work from the laboratory. The time over which this "dose of pressure" and "dose of oxygen" needs to be applied is, as yet, less clear. What is clear though, is that oxygen treatment at lower pressures is superior to deeper recompression on air. Although the current oxygen tables have a failure rate of between 4 and 15%, this was achieved in much more seriously afflicted patients than those previously treated on the air tables.

The advent of computer technology has seen the development of much remarkable investigative equipment, and with the realisation that DCS is primarily a disease of the nervous system, follow up investigation has been directed towards assessing the neurologic sequelae of DCS. Gorman, Edwards and Parsons³⁶ in treating 87 cases of DCS achieved a 96% resolution rate at discharge. At 1 week follow up, 47% had abnormal EEGs and 20% had abnormal CT scans. So, is our treatment of DCS as effective as we might think, or has improving technology just reinforced our fears that we really know little about this disease and how to treat it effectively?

The future

Current research is being directed at alternative approaches to the treatment of the difficult cases, i.e. those in which the victim deteriorates while at treatment depth, or those with significant deficits not improving at treatment depth. Previously, further compression to 30 or 50 msw has been advocated. Mixtures of oxygen and an inert gas (helium or nitrogen) are breathed and a saturation type final decompression is adopted for the return to the surface.1,23,26,27 The use of helium has been favoured recently because of the existence of safe decompression tables for heliox diving and because of the decreased incidence of serious DCS noted in heliox divers.^{37,38} Heliox diving however is usually performed by professional divers in saturation, and the relevance of these studies to the treatment of no-decompression sport divers is tenuous. Isolated case reports with sports divers demonstrate that these techniques are occasionally effective, but then failures are not usually reported.^{2,32,37,38}

In contrast, in 1984 the USN began pursuing the option of being able to remain at 18 msw indefinitely when the diver was not responding to standard therapy. Advantages were seen in this, as no additional gas mixtures would be required thus simplifying therapy. This investigation saw fruition in 1989 when treatment table 7 was promulgated in the USN Diving Manual.³⁹ Table 7 is a "heroic measure" for the treatment of the seriously ill diver and is essentially an extension of Table 6 at 18 msw with a saturation type decompression to the surface over 36 hours. The minimal time at 18 msw is 12 hours with solid evidence of continuing benefit required for stays of longer than 18 hours. A maximum of 24 oxygen breathing periods are allowed for. This gives a table length of 48+ hours. The usefulness of this technique is yet to be verified!

In the meantime we shall all continue to search for a solution to the treatment of the non-responding neurological DCS patient.

Conclusions

bles have remained effective in the treatment of a sports diver population that is presenting with increasingly more severe disease. However, as our investigative tools become more sophisticated, previously covert disease will be demonstrated. When the efficacy of these tables is tested against our ability to demonstrate pathology in this minimally affected group of DCS patients they may, as have the air tables in the past, be found wanting. Continued development of investigative tools and the on-going analysis of results is warranted.

References

- Leitch DR and Hallenbeck JM. Pressure in the treatment of spinal cord decompression sickness. Undersea Biomed Res 1985; 12(3): 291-305
- 2 Shilling CW, Carlston CB and Mathias RA. *The Physician's guide to diving medicine*. New York, Plenum Press, 1984; 286
- 3 Pol B and Watelle TJJ. Memoire sur les effects de la compression de l'air. *Annal d'hygiene publique et de medicine legale (Paris)*, 1854; 1: 241-279
- 4 Bert P. Barometric pressure. 1878. (1943 Translation to English, republished by Undersea Medical Society, Bethesda 1978)
- 5 Zuntz N. Zur Pathogenese und Therapie der Durch Rashe Luftdruckanderungen Erzengten Krankheiten. *Forschr. d. Med Berlin* 1897; 15: 632-639
- 6 Behnke A and Shaw L. The use of oxygen in the treatment of compressed air illness. US Naval Medical Bulletin 1937; 35(1): 61-73
- 7 Yarbrough O and Behnke A. Treatment of compressed air illness utilising oxygen. J Indust Hygiene Toxicol, 1939; 21(6): 213-218
- 8 Van der Aue OE, White WA, Hayter R, Brinton ES, Kellar JR and Behnke A. *Physiologic factors underlying the prevention and treatment of decompression sickness.* USN Experimental Diving Unit, Report 1, Washington DC: USN, 1945
- 9 Van der Aue OE, Duffner GJ and Behnke AR. The treatment of decompression sickness: an analysis of 113 cases. *J Indust HygieneToxicol* 1947; 29: 359-366
- 10 Slark AG. Treatment of 137 cases of decompression sickness. RNPL report 8/62 August 1962
- 11 Rivera JC. Decopression sickness among divers: an analysis of 935 cases. *Milit Med* 1964; 129: 314-334
- 12 Goodman MW. Decompression sickness treated with compression to 2-6 atmospheres absolute. *Aero-space Med* 1964; 35: 1204-1212
- 13 How JYC. Medical support of compressed air tunnelling in the Singapore Mass Rapid Transit project. SPUMS J 1989; 19(4): 155-172
- 14 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: USN, November 1965

- 15 Workman RD. Treatment of bends with oxygen at high pressure. Aerospace Med 1968; 39: 1076-1083
- 16 Bornman RC. Limitations in the treatment of diving and aviation bends by increasing ambient pressure. *Aerospace Med* 1968; 39(10): 1070-1076
- 17 Lambertsen CJ. Concepts for advances in therapy of the bends in undersea and aerospace activity. *Aero-space Med* 1968; 39(10): 1086-1093
- 18 Erde A and Edmonds C. Decompression sickness: a clinical series. J Occup Med 1975; 17(5): 324-328
- 19 Davis JC (Editor). Treatment of decompression accidents among sport scuba divers with delay between onset and compression. UMS Workshop Report No. 34, Bethesda, Maryland: Undersea Medical Society, 1979
- 20 Melamed Y and Ohry A. The treatment and the neurological aspects of diving accidents in Israel. *Paraplegia* 1980; 18: 127-132
- 21 Hart GB. Treatment of decompression illness and air embolism with hyperbaric oxygen. *Aerospace Med* 1974; 45(10): 1190-1193
- 22 Bornman RC. Experience with minimal recompression oxygen breathing treatment of decompression sickness and air embolism. USN Experimental Diving Unit Report 2/67, Washington DC: USN, 1967
- Kizer KW. Delayed treatment of dysbarism. JAMA 1982; 247(18): 2555-2558
- 24 Green RD and Leitch DR. Twenty years of treating decompression sickness. Aviat Space, Environ Med 1987; 58: 362-363
- 25 Pilmanis AA. Treatment for air embolism and decompression sickness. *SPUMS J* 1987; 17(1): 27-32
- 26 Leitch DR and Green RD. Additional pressurisation for the treatment of non-responding cases of serious decompression sickness. *Aerospace and Environ Med* 1985; 56(12): 1139-1143
- 27 Pilmanis AA. Hyperbaric oxygen therapy rationale for the treatment of diving accidents. SPUMS J 1987; 17(1): 20-23
- 28 Davis JC et. al. Altitude decompression sickness: Hyperbaric therapy results in 145 cases. Aviat Environ Med 1977; 48(8): 722-730
- 29 Leitch DR and Green RD. Pulmonary barotraume in divers and the treatment of CAGE. Aviat Space, Environ Med 1986; 57(10): 931-938
- 30 USN Diving Manual 1985. Best Publishing Co., 1985; section 8: 21-50
- 31 *RN Diving Manual.* BR 2806 HMSO: London, 1987; section 5: 15-26
- 32 Edwards C, Lowry C and Pennefather J. *Diving and subaquatic medicine*. 2nd edition. Sydney, Diving Medical Centre, 1981; 160-161
- 33 Saumarez C, Bolt JF and Gregory RJ. Neurological decompression sickness treated without recompression. *Brit Med J* 1973; 2: 151-152
- 34 Leitch DR and Hallenbeck JM. Oxygen in the treatment of spinal cord decompression sickness. *Under*-

sea Biomed Res 1985; 12(3): 269-289

- 35 Donald KW. Oxygen bends. J Appl Physiol 1955; 7: 639-644
- 36 Gorman DF, Edwards CW and Parsons DW. Neurologic Sequelae of DCS: A clinical report. Underwater and Hyperbaric Physiol IX. 1987; UHMS: 993-998
- 37 McIver NKI. Treatment of compressed air decompression accidents. *J R Soc Med* 1989; 82(2): 74-79
- 38 Douglas JDM and Robinson C. Heliox treatment for spinal DCS following air dives. Undersea Biomed Res 1988; 15: 315-319
- 39 USN Diving manual 1989. Washington DC: US government printing office, Section 8: 29-39

Dr Geoffrey S Gordon, BHB, MB, ChB, FFARACS, is an anaesthetist at the Townsville General Hospital, Townsville, Queensland 4810, Australia.

HIGH RISK DIVING TASMANIA'S AQUACULTURE INDUSTRY

David Smart and Peter McCartney

Despite its cool climate and waters Tasmania has a large population of commercial and sports divers working in, and enjoying the waters of its 3,200 km coast line. Of a total population of 446,500 (1986 figures)¹, over one percent are active divers (Table 1).

Diving accidents in Tasmania requiring hospital admission or recompression therapy occur on average fifteen to twenty times per year. These patients are treated at the Royal Hobart Hospital (RHH) Recompression Chamber by staff of the Hyperbaric Unit, which is linked to the Department and Anaesthesia. There has been little change in the number of accidents over the years 1985 to 1988. The 1985-1988 population treated at RHH consisted mostly of abalone divers and a small number of recreational divers. In 1988 the first of a new population of divers presented for treatment. These divers were employed in the aquaculture industry. From April 1988 to October 1989, eighteen divers from all sources were admitted to hospital for diving related illness and twelve were treated in the recompression chamber. Sixteen of the eighteen divers had been using hookah apparatus and nine divers were employed in the aquaculture industry.

This industry is now a major export earner for the state of Tasmania and by October 1989 according to the Department of Sea Fisheries more than 270 people were employed at 35 separate ventures. At the time of writing

TABLE 1

DIVERS IN TASMANIA

| Registered by the Department of Sea Fisheri | ies | |
|---|------|--|
| Non Commercial Diving Licence | | |
| Commercial Abalone Licence | 125 | |
| Commercial Diving Licence | | |
| (including police divers) | 229 | |
| Unregistered divers (estimates) | | |
| Unlicenced Amateur Divers | 3000 | |
| Sea Urchin and Periwinkle Divers | 400 | |
| Aquaculture divers | 50 | |

approximately 40-50 were divers. During 1990 further growth is expected in the industry to over 400 employees. From its humble beginnings in 1986 the industry now produces Atlantic Salmon of world export quality. The salmon are "farmed" from smolt (50 g size) to adult size in floating circular pens approximately 20 m in diameter (Plate 1, page 151) supporting a cylindrical net approximately 8 m deep. Feed is released automatically at regular intervals to the fish. There can be as many as 5,000 fish per pen. Larger operations manage 40 pens or more. Surrounding the inner pens is a coarser mesh perimeter net (Plate 2, page 151) of up to 250 m by 500 m to prevent predators such as seals attacking the salmon. Divers in the industry are required to maintain these nets and pens and to remove diseased or dead fish from the pens. They are also required to inspect and maintain mooring lines. In some leases the perimeter nets and mooring lines extend to depths of 40 m. Divers contribute significantly to the quality of the salmon when it is finally ready for marketing.

This paper examines the diving practices of the industry, based on information gained from divers treated at RHH, in the hope of reducing the number of diving accidents in the future. One of the authors (DS) was privileged to visit one of the larger fish farm leases at Tassal, Dover, and witnessed its impressive operation at first hand.

Information gained

Nine aquaculture divers were treated at RHH. The majority (5) were aged between 21 and 30. Two were between 31 and 40. There was one in the 10-20 group and one aged between 41 and 50. Eight were male and one female.

Of concern is that only 2 divers had had appropriate training, i.e. specifically for the industry. Even more disturbing is the fact that 4 of the divers had had no formal training while the other three had only had formal training for using scuba as a recreational diver. Of these one had