The Editor's Offering

This issue starts off with an unexpected finding from Western Australia. In a group of 36 divers with decompression related illness, nine, who presented with neurological or cerebral symptoms were shown to have a right-to-left shunt, seven with a definite patent foramen ovale (PFO) and two who showed a late burst of bubbles, while a tenth diver with cerebral symptoms refused the injection needed for the investigation. Bob Wong and David Wright's patients had an incidence of PFO caused decompression sickness of about that of PFO at post-mortem. A recent paper on divers with neurological decompression sickness investigated for PFOs claims "that most cases of spinal decompression illness divers are associated with a right-to-left shunt".[1]

These two papers reinforce the reasoning behind the paper presented by Jürg Wendling at the 2000 Annual Scientific Meeting discussing the feasibility of screening budding divers for PFO. It would seem be that divers with cerebral symptoms and with spinal signs and symptoms should be investigated by the treating hyperbaric facility. Although cardiologists consider contrast echocardiography a safe procedure, two of Wong and Wright's patients (20%) developed cerebral symptoms after the procedure and had to be recompressed.

Christian Donatsch spent his time as a Club Med doctor in the Maldives conducting a survey of symptoms which can occur with decompression illness or from other causes in order to assess the incidence of subclinical decompression illness. He was able to conclude that recreational diving at Club Med in the Maldives is not associated with more of these symptoms than windsurfing or sitting on the beach. The reason is that the shallow diving in warm water very seldom reaches anywhere near the no-stop limits.

Carl Edmonds, in Diving Doctor's Diary, gives an update of pulmonary oedema, dyspnoea and diving. Both his illustrative cases were medicos and both were on beta-blockers. Just another reminder that the sea can be dangerous to those who are not cardiologically fit for exercise.

There are no medical book reviews in this issue but all three books are recommended, one for diving safety, one for a look into 18th Century barrel diving and the third has special interest for Australians, being set in Broome in 1912 when there was an failed attempt by the Federal Government to apply the White Australia policy to the pearling industry.

The papers from last year's Fiji meeting, besides PFOs, discuss the assessment of fitness to return to diving after a decompression accident and a variety of other topics. David Elliott lays out a logical method of dealing with this problem. Rees Jones, a New Zealand pathologist whose practice covers the waters north of Auckland, reports on the only two deaths since the middle 80s to reach him from the Poor Knights Islands. Both died from arterial gas embolism without any rapid ascent. Paul Thomas, Clifford Ng and Michael Bennett describe how they produced a correction curve which allows Wright peak flow meter readings in a hyperbaric chamber to provide useful information. Malcolm Le May reminds us that eyes first developed in aquatic animals and discusses the various modifications that have developed across the animal world. Robyn Walker discusses the long term effects of diving.

Two of the articles reprinted from other Journals come from off shore islands, Tasmania and Orkney. David Smart and his colleagues presented an update on the health of Tasmanian fish farm divers to the Royal Society of Tasmania, which was published in 1999, which the Journal has been allowed to reproduce. It is gratifying to read that the situation in Tasmania is no longer the disaster story we published in 1990 by David Smart and Peter McCartney, whose hyperbaric unit was almost under siege by bent, untrained, fish farm divers. By 1998 the majority of the divers were trained to the appropriate Australian standard for occupational divers. The reduction in decompression illness cases was such that it is was an order of magnitude less that the figures for the Australian pearl diving industry in 1995 and Scottish fish farm industry in 1992.

The Orkney islands surround Scarpa Flow where the scuttled ships of the Imperial German Navy are dived regularly by recreational divers. Heriot-Watt University has a recompression chamber in Stromness where Alister Wallbank, their Deputy Diving Officer is based. Among his jobs is treating bent divers in the University chamber. He found time to conduct a research project for the University and Health and Safety Executive to find the most useful colours and shape for Diver Emergency Surface Location Devices. Through the kindness of the Editor of DIVER we reprint his Diverised version of the report in this issue.

The third reprinted paper, also from DIVER, is the story of a disaster avoided by pure luck. Had the two divers had read Alister Wallbank's paper they would not have been lost. But their incident was 30 years ago but even then sensible divers doing a drift dive used a surface marker buoy which they did not.

Reference

1 Wilmshurst P and Bryson P. Relationship between the clinical features of neurological decompression illness and its causes. *Clinical Science* 2000; 99: 65-75

MODES OF PRESENTATION OF PATENT FORAMEN OVALE IN TEN DIVERS

Robert Wong and David Wright

Key Words

Accidents, cardiovascular, case reports, cerebral arterial gas embolism, decompression sickness, medical conditions and problems.

Summary

A series of divers treated for DCS at the Fremantle Hospital Hyperbaric Unit during 2000 showed a high prevalence (28%) of patent foramen ovale, which is consistent with the autopsy findings in Hagen's study.[1] We acknowledge that our series may be considered too small to be a representative sample.

We recommend that the presence of a PFO should be considered in any diver who presents with predominantly cerebral features of DCS and that such cases should be investigated for PFO. In our series there were 2 cases (cases 2 and 7) who had been treated for such symptoms in the past but had not been investigated at that time. It is possible that a large number of cases in other centres are also not investigated.

Since this paper was accepted for publication, we have detected PFO in four divers out of a total of 30 cases of DCS seen between 2001/1/1 and 2001/4/20.

Introduction

Forty-one divers were seen in our Department during the year 2000 for review and/or management. Five patients did not have decompression sickness, leaving 36 with decompression sickness. Ten (28%) were diagnosed as having a patent foramen ovale (PFO) which had given rise to cerebral arterial gas embolism (CAGE). Twenty (55%) were diagnosed as having decompression sickness (DCS) and six (17%) as CAGE. This report describes the presentation, treatment, and investigation of the ten patients with PFO and CAGE.

Their diving experience varied from beginners learning to scuba dive to experienced divers who had logged over 3000 dives or had 20 years experience. Apart from one commercial diver in this series, all were recreational divers.

The profiles that precipitated neurological symptoms were not necessarily benign; four were to depths of 30 m, one was to 41 m, and all these were provocative. While some recalled performing a forceful Valsalva manoeuvre during the dive, this was not a constant feature.

A high index of suspicion is required when a diver presents with neurological (particularly cerebral) symptoms after a dive. Previous diving experience does not preclude this. Echocardiography to exclude the presence of a PFO is recommended.

Clinical Cases

Case 1

A 33-year-old male was diving in the Swan River to catch crabs and fish. His profile was 15 m for 30 minutes. He surfaced with no safety stops to take his bearings, then descended to 10 m to follow the riverbed to the surface. As he left the water, he experienced a rapid onset of weakness. This progressed over two minutes to affect all his limbs, initially his right leg, then left leg, right arm and left arm consecutively, such that he required assistance from his dive buddy to get up to the riverbank. He also developed complete blindness and felt confused. On his way to hospital by ambulance, he was given high flow oxygen, during which his vision and lower limb strength gradually returned.

At initial assessment in the emergency department, all he complained of was a mild headache and weakness in the right leg. High flow oxygen by non-rebreathing mask was continued. Physical examination revealed a slight weakness of right hip flexors, hip extensors, knee flexion and knee extension; upper limb strength was normal. There was no sensory deficit. Visual testing and fundoscopy were normal. His Sharpened Romberg score was 45 seconds. On review and on the basis of the dive profile, it was thought that DCS was unlikely, and he was discharged and was advised to report to the Hyperbaric Department for review.

Later in the day, he represented to the Emergency Department with symptoms of vagueness, mild dysphasia, and myalgias affecting his shoulders and thighs, intermittent mild headache, and ongoing concern about his collapse earlier in the day. Physical examination was unremarkable. A diagnosis of possible Transient Ischaemic Attack was made, and he was discharged and advised to see his General Practitioner for review. His sister, a nurse, said that he probably had a mild "stroke" and he attempted to seek admission to a private hospital without success.

Having been told that DCS was unlikely, he did not report to the Hyperbaric Department for review. He went to work despite feeling unwell, and he presented to the Emergency Department of another hospital five days later. Again he was advised to report for assessment by a Hyperbaric Physician.

Six days after his dive he was seen at our Department and the same history was elicited. During the intervening days, he had noticed poor concentration and forgetfulness, particularly at work, intermittent dizziness and feeling vague. A diagnosis of CAGE was made and he was recompressed, following which, he felt more alert and noticed improved strength. He received further oxygen treatments and felt subjectively back to normal.

This man had been a regular scuba diver for over 20 years, and had never experienced any symptoms. A neurological consultation was sought, and the opinion was that an arterial gas embolism affecting the posterior cerebral circulation was most likely. A transthoracic echocardiogram (TTE) was performed with agitated saline contrast that demonstrated a small communication at atrial level, probably a PFO. This man has accepted advice not to dive again. At follow up after three months he was well with no apparent sequelae.

Case 2

A 32-year-old male with 17 years diving experience, and who had logged approximately 400 dives, was referred to our Department two days after diving with a diagnosis of probable DCS. On this occasion, he had made a single dive for crayfish to 15 m, with an average depth of 10 –11 m for 43 minutes. A safety stop was made at 6 m for four minutes. He experienced a sinus squeeze during the descent with right eye pain and had performed a forceful Valsalva. The sinus pain eased during ascent to 10 m.

On completion of his dive, he felt well. While standing on the deck of the dive boat about two minutes after surfacing, he experienced a sudden onset of fatigue and had just wanted to lie down. He tried to remove his diving equipment but experienced difficulty. He sat down and noted loss of power in his arms and legs. He also noted dysphasia and a feeling of disorientation. There was no loss of consciousness, but he experienced transient visual disturbance with a black and white worm-like pattern across his visual fields.

The dive master recognised that the diver was experiencing difficulties, laid him down and gave oxygen. A basic field assessment showed weakness of all limbs and neck muscles. Oxygen was continued for approximately 40 minutes. An ambulance met the dive boat to transport the patient to hospital, by which time his visual changes and weakness had resolved, although he remained lethargic. He was assessed in an Emergency Department, where, unfortunately, some of the key points in the history were missed (visual changes and weakness). Nonetheless, a diagnosis of DCS was made. This diver has a past history of DCS, and had been seen earlier with an almost identical presentation, which following a dive which had required a forced Valsalva for middle ear equalisation. This incident had been treated successfully with recompression, but no investigations were performed.

When assessed in our Department two days later, this man had continual headache with some difficulty in concentration and recall, even of information such as his own telephone number. On examination, a left grade three middle ear barotrauma was noted, but there were no abnormal neurological findings. Sharpened Romberg score was 60 seconds on the second attempt. A diagnosis of CAGE was made. Despite his 17 years of diving experience, the mode of presentation and his dive profile made the diagnosis of PFO highly probable.

He was recompressed. At the completion of the treatments, he felt well apart from intermittent occipital headache. He was referred for TTE to determine whether or not a PFO was present, but unfortunately he decided not to proceed with this because of a needle phobia. Although this case was not confirmed by TTE, he sensibly decided to give up diving.

Case 3

A 44-year-old male presented with vague symptoms of clouded thought, mild apraxia, fatigue, and a reduced ability to concentrate. He last dived two days before his referral and had been scuba diving for two years and had logged 120 dives.

Three months before presentation, he had dived on a wreck to 30 m, for which he was paired with an inexperienced diver. During the final ascent, his buddy descended again to the bridge of the wreck. He was concerned that his buddy might have been affected by nitrogen narcosis and was unaware that they might have exceeded the no-decompression limits. He swam after his buddy to bring him back to the anchor line of the dive boat. This action necessitated an additional decompression time for which he utilised the emergency tank on the anchor line.

He dived regularly on most weekends, but after each of these dives he noted that his ability for sustained concentration was impaired and that he made frequent errors with his written work. He used a dive computer for guidance. After his last dive, he noticed transient sensory change over the right side of his face, but he had no peripheral arthralgias, motor or other sensory symptoms. Physical examination was unremarkable. Although the score of his Folstein's mini-mental test[2] was normal, his performance for numerical and short-term memory tasks was significantly slower, and laboured, than would have been expected. The performance was inconsistent with his career as a mathematician.

In view of the temporal relationship of his symptoms with diving he was given a trial of pressure to 18 m. This resulted in significant subjective improvement of his

cognitive function. He received further oxygen treatments and has remained well since.

Due to the predominantly cerebral nature of his symptoms, he was referred for a TTE. This clearly demonstrated a PFO with right to left shunting in the release phase of the Valsalva manoeuvre.

He was advised to cease scuba diving, however he was a committed and enthusiastic diver who was keen to pursue all options to enable him to continue to dive. He even considered surgical intervention and correction of his PFO if necessary. Trans-oesophageal echocardiogram (TOE) and magnetic resonance imaging (MRI) of the heart showed that the PFO was about 10 mm in size. He elected for a closure of the PFO, which was performed transluminally with an Amplatzer Septal Occluder (AGA Medical Corporation, Golden Valley, Minnesota, USA). There is inadequate data to determine the successes of such procedure at present. He returned to diving three months after the closure of his PFO. Up to January 2001 he had completed ten dives to depths of 30 m and has been free of any symptoms of DCS.

Case 4

A 39-year-old man did a recreational 30 m dive for 30 minutes using a dry suit. The dive itself was uneventful. He was an experienced professional diver with over 3,000 logged dives, including mixed gas diving. Fifteen minutes after surfacing he noted discomfort in his right axilla, a right hemiparesis and numbness in the right leg. He noted his gait was abnormal, feeling unsteady, with his right leg giving way. Initially he attributed his right axillary discomfort to a nicotine patch that he had put on in the morning of his dive.

On presentation to our department three days later, he had persistent right axillary and right leg pain, and mild weakness of right toe extensors. His Sharpened Romberg was unsteady, with a best score of 30 seconds.

He was recompressed, with subjective improvement and resolution of his pain, and of toe extensor weakness. His Sharpened Romberg score improved to greater than 60 seconds.

A TTE was performed that demonstrated a PFO, with minor shunting at rest, and increased shunting in the release phase of the Valsalva manoeuvre. Despite our advice to this man to cease compressed air diving, he has continued with some diving although we do not know the diving profiles that he uses.

Case 5

A 30-year-old female novice diver experienced a mask squeeze on descent during the third open water dive of her scuba course. At 18 m she had considerable sinus pain that forced her to make a rapid, controlled ascent with her instructor. On the surface she noticed blood in her mask and her hearing was notably muffled.

Over the next 24 hours she was unusually fatigued, with impaired concentration, nausea and intermittent headaches. Examination at this time showed evidence of bilateral middle ear barotrauma and bilateral periorbital haematomas. Her Sharpened Romberg score was 10 seconds. Mini-mental status examination score was 27/30, with mild impairment of short-term memory and calculations.

Although there was clear evidence of sinus and middle ear barotrauma, in view of her subtle subjective cognitive impairment, coincidental DCS could not be excluded. She was recompressed, which led to subjective improvement in her mentation, with greater ability to concentrate, and her Sharpened Romberg score improved to greater than 60 seconds.

Four days after completing treatment, a TTE was performed which confirmed the presence of a PFO, with trivial shunting at rest, and with slight augmentation of shunting in the release phase of the Valsalva manoeuvre.

Following the TTE, this patient experienced a relapse of symptoms including headache, impairment of sustained concentration and short-term memory, and dizziness. She was again recompressed until complete resolution of her symptoms. She was advised not to dive.

Case 6

A 32-year-old female diver, who held only an Entry Level Open Water "C" Card, returned to scuba diving after an eight-year absence. She went on a diving holiday and undertook a series of 10 dives outside the DCIEM no-decompression limits (provocative dives) over four days to depths up to 41 m). She began to feel unwell after her second dive with nausea, visual disturbance, difficulty with concentration and short term memory. She also had left shoulder ache and left arm paraesthesiae. Nonetheless, she continued to dive for the four days!

She presented three days after her holiday ended. Physical examination was unremarkable. Given her profile and symptoms, she was given recompression therapy, which gave rise to rapid improvement in all of her symptoms during the first treatment.

In view of the predominance of cerebral symptoms, a TTE was performed. This confirmed the presence of a PFO, with minor shunting at rest, but with significantly increased shunting in the release phase of the Valsalva manoeuvre. She was advised not to dive.

Case 7

A 24-year-old male diver on this occasion had undertaken two dives. The first dive was to 21 m for 51 minutes, with a safety stop at 5 m for five minutes. After a 3 hour surface interval, he conducted a second dive to 10.5 m for 48 minutes, with a safety stop at 5 m for five minutes. On leaving the water, he had a headache.

Over the preceding month, he had noted headaches each time he surfaced after a dive. At his request, this man was reviewed because he wanted to know why he has a headache each time he dives. At consultation, he was still experiencing episodes of vagueness and headache from his last dive, but there were no other neurological symptoms. A trial of pressure was given, however there was no improvement, and he became claustrophobic and dizzy in the chamber.

On review of his history, it was noted that three years previously he suffered from pulmonary barotrauma with CAGE and near drowning. On this occasion, he had dived to 60 m when his dive buddies noted that his regulator was out of his mouth and he was not breathing. His buddies brought him to about 15 m and put his regulator in his mouth, inflated his buoyancy compensator and brought him to the surface. He was noted to be cyanosed and apnoeic. After towing him to the dive boat, cardiopulmonary resuscitation was given aboard, to which he responded. He was taken to hospital and was given recompression therapy. Despite the diagnosis, he was permitted to dive again, but was advised not to dive deeper than 30 m because he had suffered nitrogen narcosis at that depth on an earlier dive. No investigations were done other than an initial chest X-ray that supposedly was normal.

In view of his past history, TTE and high resolution CT scanning of the chest were done. The CT chest was normal, but the TTE revealed a PFO with trivial right to left shunting in the release phase of the Valsalva manoeuvre. He has been advised against further compressed air diving.

Case 8

A 33-year-old experienced dive instructor presented with symptoms suggestive of DCS. He had been teaching an enriched air nitrox (EAN) course, and had used a dive computer. He conducted two dives using EAN 32 (O_2 32%) mix. The first dive was to 30 m for 30 minutes with a slow ascent and a safety stop at 5 m for three minutes. During his surface interval of 85 minutes he went for a prolonged swim. His second dive was to 26.5 m for 30 minutes, again with a slow ascent and a safety stop at 5 m. During this second dive, an O-ring on his regulator ruptured on entering the water. He repaired this and then continued diving. On the second day of diving, after a surface interval of 17 hours, he conducted two air dives. The first dive was to 23.7 m for 35 minutes and the second dive was to 23.2 m for 35 minutes. At the end of each of these dives, a slow ascent was made and a safety stop for three minutes at 5 m was made. The surface interval between the dives was 81 minutes. Assessing these dive profiles with DCIEM tables, he had exceeded the no-decompression limits.

Two days after these dives, he felt unwell with dysphoria, nausea and intermittent, migratory paraesthesiae in his right face, forearm and lower leg, and an ache in his right shoulder. He felt that his concentration was poorer.

On physical examination, Sharpened Romberg score was three seconds. There were isolated patches of hypoaesthesia to pain over the right jaw in C1-2 distribution and over the right C5 distribution.

He was recompressed with subjective improvement in cognitive function, reduced shoulder ache and reduction of paraesthesiae. After three further oxygen treatments some symptoms of right shoulder ache and paraesthesiae still remained.

A TTE was performed which showed a PFO with right to left shunting, with trivial shunting at rest and a mild increase in shunting at the peak of the Valsalva manoeuvre.

He was advised against further scuba diving. However, at the time of discharge, he was contemplating further assessment with a view to proceeding to transluminal closure of the PFO.

However, six weeks after discharge he did a shore dive to 8.9 m in calm water with a total dive time of 52 minutes. He developed minor symptoms of DCS in a similar distribution to his initial presentation. These symptoms resolved with further recompression treatment and he decided not to dive again.

Case 9

A 29-year-old man was on his initial scuba course. During his first ocean dive he experienced difficulties with middle ear equalisation on descent. However, he managed to equalise by ascending slightly and performed a very forceful Valsalva manoeuvre. He continued his dive to 18 m for 21 minutes, then made a slow ascent, with safety stops at 6 m for six minutes and 2 m for four minutes.

On surfacing, he immediately felt nauseated and vomited. He attempted to climb onto the dive boat, but felt he was unable to co-ordinate his right leg. This was followed by paralysis of his right arm. He also noticed dimming of vision, although he did not go blind, with only vague blue shapes visible. His speech was jumbled. One of the diving instructors assisted him onto the boat and removed his gear, laid him flat and gave oxygen.

Within five minutes, power returned to his right arm and after 15 minutes his vision returned to normal. He was immediately evacuated to hospital. On arrival he only complained of a mild headache. On examination, there was a small patch of diminished sensation to temperature over the dorsum of his left hand and lateral left foot, and bilateral middle ear barotraumas were noted. He was maintained in the supine posture.

He was recompressed on oxygen. During treatment his headache cleared, sensory changes resolved and subjectively he felt much improved. On reaching 9 m, a Sharpened Romberg was performed with a score of 5 seconds. Towards the end of the treatment table, this score improved to 40 seconds. Two further oxygen treatments were given with full resolution of symptoms.

His presentation is highly suggestive of CAGE with a PFO. A TTE was performed but the result was inconclusive, with bubbles appearing in the left atrium after a delay without any clear intracardiac shunt. Further assessment with TOE is being undertaken. He has been advised not to dive.

Case 10

A 39-year-old diver, with 12 years of experience presented two days after a dive on EAN to 4 m for 30 minutes. The dive was uneventful. Over the following 24 hours he experienced light-headedness and arthralgias in both forearms with associated paraesthesia. Neurological examination revealed generalised hyper-reflexia, with non-sustained clonus of the left knee jerk. Hyperbaric oxygen therapy resulted in full resolution of symptoms.

One year before this dive he had been treated at an interstate recompression facility for DCS following a provocative dive to 31 m with omitted decompression stops. He had delayed presenting for treatment for two weeks because of personal circumstances. Hyper-reflexia and clonus had been noted at that time, and the diver said they had been worse then than on this occasion. He had a brain MRI performed at the treating hospital which apparently identified eight brain lesions consistent with cerebral gas embolism.

In view of his cerebral symptoms a saline contrast transthoracic echocardiogram was performed. This was inconclusive, as there was delayed appearance of bubbles in the left atrium after injection of saline contrast, without an apparent intracardiac shunt. However, forty minutes after the TTE, he had a relapse of neurological symptoms, with onset of dizziness, return of paraesthesiae and arthralgia in his hands. He had further recompression treatments with incomplete resolution of symptoms.

A TOE had been considered to confirm the presence of a PFO, but he understandably declined further investigation. He was advised against further diving and sensibly decided to cease diving.

Discussion

A PFO is the most common persistent cardiac abnormality of foetal origin. Normally, the thin left-sided septum primum is pushed against the thicker septum secundum by the higher left atrial pressure thus preventing right to left shunting. A PFO is a dynamic structure with variations in the size of the opening, the size and direction of the septal tunnel and the amount of redundant interatrial tissue.[3] A large PFO may permit right to left shunting under physiological conditions,[4] whereas a smaller PFO may only be flow patent during transient periods, such as with sudden changes in intrathoracic pressure or in right heart compliance, when the right atrial pressure exceeds left atrial pressure.

A full discussion of factors affecting flow patency of a PFO is beyond the scope of this paper, however it should be recognised that intra-subject variability of flow patency occurs. Wilmshurst et al demonstrated that the size and patency of a shunt may not be reproducible from one contrast injection to another.[5]

The true prevalence of PFO in the normal population is not known. However, since the post-mortem findings of Hagen et al, the prevalence of PFO has been accepted as 30%.[1] The overall incidence of this study was quoted as 27.3%, but it progressively declined with increasing age from 34.3% during the first three decades to 25.4% during the fourth through eighth decades and to 20.2% during the ninth & tenth decades. Furthermore, the study indicated that the size of the PFO tended to increase with age, from a mean of 3.4 mm in the second decade to 5.8 mm in the tenth decade.

In our series DCS coexistent with PFO was diagnosed in 28% of the 36 divers who presented in the year 2000. The high clinical prevalence of PFO in our series is close to the incidence of autopsy demonstrated PFO reported by Hagen et al.[1] This may be due to our aggressive investigation of divers who present with cerebral symptoms or it may reflect bias due to our small sample size. Had we been more vigilant previously and considered PFO as a cause of the DCS, more cases might have been identified.

Lechat et al used contrast transthoracic echocardiography (TTE) to study ischaemic stroke patients (comparing them with an age-matched control group) and found the prevalence of Patent Foramen Ovale (PFO) in adults younger than 55 to be in the vicinity of 10-40% of the population.[6]

In another study using contrast transoesophageal echocardiography (TEE), Fisher et al showed the prevalence to be 9.2%.[7] It was also shown that the prevalence in patients aged 40-49 was greater than those aged 70 -79 years (12.96% cf 6.15%). This trend is consistent with the report of Hagen et al.[1] The natural history of an individual's PFO with ageing has not been elucidated.

Whatever the true prevalence is, it appears, from the studies, that in the younger age group PFO is more common and that the size is smaller. What is the significance of this? Does that mean it is more common for younger divers to have Type 2 DCS because PFO is more prevalent? Or is it more likely to occur in older divers because the diameter of the PFO is larger? In our small series, most divers were in the fourth decade (seven out of ten), two in the third decade and one in the fifth decade.

It is also known that a number of divers, after diving safely for many years and logging in excess of 1,000 dives suddenly become hit by neurological DCS.[8] This has also been our experience illustrated by Cases 1, 2, 4, 8 and 10. It is possible that a minimal PFO may become flow-patent with ageing.

The presence of PFO seems to be a risk factor for the development of DCS in divers. Moon et al quoted a high prevalence of 61% in a subset of 18 divers with shunting who showed signs and symptoms of serious DCS.[9]

Germonpré et al. also found that overall prevalence of PFO in DCS was 59.5%.[10] They demonstrated that divers with cerebral DCS had a significantly higher prevalence of PFO than did control divers without PFO. In contrast, the prevalence of PFO in their divers with spinal DCS was not significantly different from that of the control population.

Since the presence of PFO predisposes a diver to serious DCS, what are the risks? Is it worth screening divers for PFO if it is accepted that potentially 30% of the population have a PFO?

Bove's analysis shows that the risks of developing Type II DCS, assuming a prevalence of 30% PFO, is in the vicinity of 2.28/10,000 dives, which he did not believe warranted routine screening by echocardiography.[11]

In contrast , a study by Knauth et al. found multiple brain lesions in divers who had never experienced Type II DCS, which they concluded was most likely a consequence of subclinical arterial gas embolism.[12] This study involved 87 divers who each had a minimum of 160 dives. The prevalence of multiple lesions was higher in the 25 divers with a PFO than in the 62 divers without. They also found that a statistically significant correlation between PFO of high haemodynamic relevance and the presence of multiple brain lesions on MRI. Haemodynamic relevance was classified as low if fewer than 20 air microbubble signals occurred after a Valsalva manoeuvre during transcranial Doppler ultrasonography and high if 20 or more signals occurred. In view of this study, prospective screening of divers for PFO of high haemodynamic relevance might appear to be justified.

Nonetheless, prospective screening for a PFO has disadvantages. The additional cost of this examination, currently about AUD$280, would be an additional financial burden for prospective divers. There is a significant false negative rate for detection of PFO by contrast TTE. While a positive TTE would require no further confirmation, a negative study may require investigation using contrast TOE.[13] Contrast TOE is more sensitive but is more invasive and usually requires sedation. Contrast transcranial Doppler is less invasive and appears to be of similar sensitivity in detecting PFO to TOE but gives no structural information about the heart.[14]

There is a small but definite risk of transient neurological events associated with the use of contrast echocardiography. The 1982 Contrast Committee of the American Society of Echocardiography reported 28 transient neurological side effects from about 41,000 investigations.[15] This was noted in our cases 5 and 10 and has also been documented by Wilmshurst.[3] A report by Lee and Ginzton described a patient with Atrial Septal Defect (ASD) who developed gross neurological symptoms after contrast echocardiography.[16] Nonetheless, we feel that this investigation is recommended following an episode of DCS with cerebral features, particularly for counselling of divers about future diving.

With an increasing number of reported cases of neurological DCS from breath-hold divers, and in view of the prevalence of PFO, one should also consider that some of these divers will have PFO, and echocardiography screening should be arranged.[17-19]

Counselling of Divers with PFO

Once PFO has been diagnosed, some divers are prepared to accept medical advice to either cease diving, or to dive conservatively. Others may pursue all options in order to continue their passion for diving. Our advice given to divers with PFO follows:

1 Explain the significance of PFO.
2 Explain that the presence of PFO produces a 2.5 times increased risk for developing serious neurological DCS.[10]
3 Advise them to take up an alternative sport.
4 If the diver insists on diving, we advise that he or she should dive conservatively, with no deep dives, no decompression dives, no repetitive dives, use a slow rate of ascent and do routine safety stops.[20,21] Of our two patients who continued to dive without having their PFO closed, Case 4 has not presented

with another episode of DCS during the seven months after he was treated. However Case 8 developed DCS after a very benign dive profile. Johnston et al.[22] have described a military diver with extensive diving experience, who was found to have an ASD during investigation of a cardiac murmur detected during his routine medical examination. This diver continued to dive more conservatively after detection of the ASD without any episodes of DCS.

5) Silent bubbles can be present in central venous blood as long as two hours after a deep dive.[23] Therefore it is prudent to avoid activities post-dive that would elevate intrathoracic pressure, such as orally inflating a buoyancy compensator, a forceful Valsalva, or heaving on an anchor line, that could allow bubbles to traverse the PFO to the left heart.

Repair of PFO

Surgical closure of PFO is feasible but, more recently, there is a technique of percutaneous closure of PFO.[3,24] There is currently insufficient information on the use of this technique for closure of PFO in divers. Nonetheless, Wilmshurst et al described the closure of a PFO with a 30 mm inverted adjustable device to permit two commercial divers to return to their occupation. While one was successful with no evidence of residual shunt, the other diver had a small, persistent shunt. Both were allowed to return to diving.[24]

Some divers might wish to pursue this option, but the current level of experience is limited to a few case reports. Case 3 independently sought interventional treatment of his PFO and appears to have made a safe return to diving. We do not recommend divers with PFO who have had an episode of neurological DCS undergo this procedure until more information on the efficacy and safety of this treatment is available.

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SUBCLINICAL DECOMPRESSION ILLNESS IN RECREATIONAL SCUBA DIVERS

Christian N Donatsch

Key Words

Decompression illness, recreational diving, research.

Abstract

This study was designed to determine if there is any evidence suggesting that recreational scuba divers diving within the commonly "accepted norms" (PADI Tables) present any signs of decompression illness. Decompression illness (DCI) is usually only diagnosed when divers have significant symptoms, such as paralysis, paraesthesia, severe rash, pruritus, etc., which lead them to consult a doctor. Divers usually neglect fatigue, headache, itchiness, and slight disturbances of gait which can be the first symptoms of DCI. This study attempted to determine if any of these sub-clinical forms of DCI were present after normal dives and their incidence. The study was performed in the Republic of Maldives over a 2 month period on a group of 28 divers and a control group of 9 non-divers. A questionnaire was submitted to every volunteer at the beginning and at the end of his/her holiday. A neurological test (Sharpened Romberg) and an otological exam were also performed on those two occasions. The analysis of the results showed no difference in the prevalence of symptoms before and after the dives in either of the 2 groups. This suggests that there is no incidence of subclinical DCI among the population tested. It is important to emphasise that this study was conducted on a limited number of cases and that all the divers tested were usually diving in warm, shallow waters, well within the limits of the PADI decompression tables and that therefore they did not expose themselves to significant risk of DCI**.** It would be interesting to carry this study on further on a group of divers who expose themselves more risk of DCI by diving closer to the PADI no-decompression limits. Therefore the author is planning to continue this study in collaboration with dive centres diving on wrecks.

Introduction

The objective of this study is to search for subclinical forms of DCI in recreational divers diving within the limits of the commonly accepted decompression tables/computers.

Definition of decompression illness

The mechanisms of DCI are complex and will not be described fully in this text. The basic principle is supersaturation of tissues by a gas with the appearance of gas bubbles in the tissues. This can cause severe symptoms, such as joint pain, paraesthesiae, paralysis and coma. However it may only cause common and unspecific symptoms such as: fatigue, headache, weakness, dizziness, cognition impairment, itching etc.

Is decompression illness under diagnosed?

The diagnosis of DCI is usually made when a patient presents to a Hyperbaric unit.[1] As many divers who present for treatment put up with their symptoms for many hours,[2] and often for days, there must be a pool of people who recover spontaneously before they realise that they have DCI.[3,4] Mild cases of DCI probably remain undiagnosed most of the time because the diver hardly notices anything wrong. The subtle non-specific symptoms are not disturbing enough to seek medical attention.

Therefore we must ask ourselves "Is DCI widely under diagnosed?"

Ultra-sound studies show that many decompressions are accompanied by detectable bubbles in the circulation without symptoms.[5] When should we start to use the term

DCI? When bubbles are present? When MRI or other imaging techniques show changes? When the patient presents vague, unexplained symptoms such as headache or fatigue? Or simply when the patient presents with severe symptoms? As DCI incidence appears to have a normal distribution,[6] none of these suggestions is an acceptable end point. Instead of trying to establish a cut off point for the diagnosis of DCI we decided, in this study, to analyse the change in prevalence of symptoms before and after a series of dives.

The list of symptoms that were enquired about corresponds to those used to track improvements in the clinical condition of patients with DCI after treatment.[7]

Possible causes of under diagnosis of DCI

The main possible causes of under diagnosing of DCI among recreational divers include:

Most symptoms of DCI are non-specific to DCI and can easily be attributed to another cause. This applies particularly on holidays when divers participate in various activities they are not accustomed to (Table 1).

One of the more frequent symptoms of DCI is fatigue. A fatigued diver does not have the ability to assess his or her situation correctly and might disregard other neurological symptoms. He or she will first want to sleep it off.

There is a general belief among recreational divers that a diver cannot have a DCI as long as he or she dives within the Tables or within the limits of his or her computer.

There is a general belief that a diver suffering from DCI has made an error or dived unsafely. This alters the diver's image among friends, family, and diving partners.

Most divers know that treatment of DCI involves transfer to a recompression chamber. This involves money, time and can ruin the diver's and his or her family's holidays.

Some divers mistakenly believe that if they are diagnosed with DCI they will never be able to dive again.

Methods

The study was carried out as the "memoir" required for completion of a 3 year post-graduate sports medicine course at the University of Geneva, Switzerland. As the study was non-invasive and was not asking the volunteers to change their diving habits, it was not submitted to an Ethical Committee.

The study involved collecting data from volunteer guests at the Club Med resort on the island of Farukulofuschi, in the Republic of Maldives. The investigator worked there as a diving doctor performing fitness to dive assessments and treating possible DCI in the on-site recompression chamber.

Data collection started on 1997/4/15 and ended on 1997/6/15. All guests arriving at the resort were informed that a medical study was being carried out, that it was voluntary and that it only required two 15 minutes sessions of their time.

Those interested received an invitation to attend the first session the next morning. At this meeting they were informed about the purpose of the study and were given an "Information leaflet and informed consent form".

Volunteers were between the ages of 18 and 60, had not dived in the last 3 weeks (a check out dive to 7 m max was allowed), were able to read English or French fluently and signed the consent/disclaimer. They had to have no history of neurological disease, concussion, loss of consciousness, paralysis, loss of sensitivity, inner ear disease, or dizziness. Volunteers wishing to participate then signed the form and received an "Initial Questionnaire" (Figure 1).

After completing the initial questionnaire the volunteers had their ears examined by the investigator. Then the sharpened Romberg test (SRT) was explained and demonstrated once by the investigator before the volunteer attempted it up to four attempts.[8,9]

Two important factors, alcohol and seasickness, can effect the SRT. By doing the test in the morning the volunteers were probably sober and had not been on a boat

TABLE 1

COMMON DIFFERENTIAL DIAGNOSES FOR DCI SYMPTOMS AMONG RECREATIONAL DIVERS

Fatigue	Difficult dive, long boat trip, late partying, lack of sleep
Itchiness	Dry skin, salt water, sunburn, lotions, marine animals
Numbness	Cold water immersion, tight wetsuit, marine animals
Joint pain	Other unusual activities done during holidays (tennis, windsurfing, etc)
Dizziness and unsteady gait	Seasickness, dehydration, alcohol consumption

Do you have right now (or within the last half hour) any of the following symptoms:

If there is a word you do not understand, please ask the doctor to explain it to you.

Important: If you answer "Slight" or "Severe" to a symptom, you must give an answer in the column "Probable cause according to you". If there seems no obvious cause then tick "I don't know".

***Please consider carefully ***	No	Slight	Severe	Probable cause according to you	
				I don't know	*I am quite sure it is due to:*
Pains:					
Pain in a joint	❑	❑	❑	❑	.....
Pain in ear	❑	❑	❑	❑	.....
Headache	❑	❑	❑	❑	.....
Sore throat	❑	❑	❑	❑	.....
Pain when passing urine (in last 12 hours)	❑	❑	❑	❑	.....
Other pain (Where: .....)	❑	❑	❑	❑	.....
Fatigue, lethargic	❑	❑	❑	❑	.....
Muscular weakness	❑	❑	❑	❑	.....
Nasal congestion	❑	❑	❑	❑	.....
Tingling sensation in arm or leg	❑	❑	❑	❑	.....
Diarrhea (in the last 12 hours)	❑	❑	❑	❑	.....
Vomiting (in the last 12 hours)	❑	❑	❑	❑	.....
Numbness in arm or leg	❑	❑	❑	❑	.....
Dizzy or unsteady when walking	❑	❑	❑	❑	.....
Itchiness	❑	❑	❑	❑	.....
Feeling of having water in ear	❑	❑	❑	❑	.....
Hearing loss	❑	❑	❑	❑	.....
Other problem:		❑	❑	❑	.....

Figure 1. Part of the questionnaire used for the initial and final interviews.

in the last few hours. As all guests arrive by boat from the airport, and are usually tired from the trip, the initial assessment was not performed on the day of arrival.

Finally each volunteer was given an appointment on the morning of the day before his or her departure. If he or she stopped diving for more than 24 hours the final assessment was to be done not more than 24 hours after the last dive.

At the final appointment the volunteers filled in the "Final Questionnaire" which is similar to the initial one but also inquired about the dives done since the initial assessment: dates, depth, duration, non-respect of tables or computer (which would imply the exclusion of the study), etc. They again attempted the SRT and had their ears examined.

Bias

We assumed that there was no difference between the population who agreed to participate in the study and those who did not.

As this was a non-randomised and non-blinded study, the volunteer obviously knew whether he/she had dived or not. This might not affect the initial assessment; it might however influence the answers to the final questionnaire and perhaps even the outcome of the final SRT. A volunteer who has dived might be more inclined to give false positive answers to symptoms than the non-diver. In order to minimise this bias, non-DCI symptoms were included in the questionnaire. However this precaution is not totally foolproof, as many divers know what the symptoms of DCI are and therefore might be more likely to give false positives only on the DCI related symptoms. Only a blinded study would totally avoid this bias.

The investigator did not have the results of the initial SRT and otological exam available when he performed the final examination.

Results

Among the 56 volunteers participating in the initial evaluation (Table 2): 28 dived and returned for the final

TABLE 2

POPULATION DISTRIBUTION

	Study Group	Control Group	Unfinished Group
Number of cases	28	9	13
Males 17	3	6	
Females	11	6	7
Male/Female [% ratio]	61/39	33/67	46/54
Age [Average]	37	40	32
Have dived before	71%	33%	46%
Number of dives during study (average)	6.4	0	?
Total dives at end of study (average)	27	0.75	?

assessment (Study group), 9 did not dive and returned for the final assessment (Control group), 13 did not return for the final assessment (Unfinished group) and 6 volunteers, two female and four male, were excluded from the study because of previous medical history.

Initial data

The data gathered from the questionnaire was analyzed in the following way: First all the DCI compatible symptoms were considered. Table 3 gives the number of cases who present with 0,1,2,3,4, and 5 symptoms.

Comments

One can see that in Table 3 there were more DCI compatible symptoms on arrival (before the dives) than on departure. This was mainly due to the presence of a great number of "fatigued" volunteers. Even though only a few gave an explanation for their fatigue it seems obvious in that setting that many were fatigued on arrival due to their long trip they had the previous day. Due to those special circumstances we decided to exclude all "fatigue" symptoms from the analysis.

In the assessment forms the volunteers had the opportunity to attribute a cause to the various symptoms. Obviously the volunteer's judgment of the origin of a symptom may be wrong. However in this population of volunteers participating in all sorts of sports (besides diving) it was necessary to give them a chance to give a rational explanation (if there is one) for their symptoms. (Table 4). This led to the exclusion of a number of DCI compatible symptoms.

It must be noted that the same three volunteers in the study group who failed the SRT on the initial assessment also failed in the final assessment. No explanation was given by the volunteers (no relevant medical history) therefore those cases were not excluded.

Any evidence of middle ear barotraumas, either otological findings on examination or sensation of water in the ear expressed by the volunteer led to the exclusion of his answer for "Dizziness" and "Hearing loss" as well as the exclusion of the Romberg test result. The reason being that it is known that middle ear barotrauma can be associated with transient inner ear dysfunction responsible of dizziness, hearing loss and a failed sharpened Romberg test.

TABLE 3

NUMBER OF CASES WITH DCI "COMPATIBLE" SYMPTOMS
(a failed SRT is considered a symptom)

	Study Group		Control Group	
Total of	**On arrival**	**On Departure**	**On Arrival**	**On Departure**
0 DCI symptoms	10	11	1	4
1 DCI symptoms	12	10	3	3
2 DCI symptoms	4	5	2	0
3 DCI symptoms	1	1	2	1
4 DCI symptoms	1	1	0	1
5 DCI symptoms	0	0	1	0

TABLE 4

PREVALENCE OF DCI "COMPATIBLE" SYMPTOMS ON ARRIVAL AND DEPARTURE

Study Group (28 volunteers)

Symptom	Assessed on	Number of cases (prevalence)		Probable cause of symptom
Pain in a joint	Arrival	2	(7%)	1 Chronic post ski trauma (symptom not present at departure)
	Departure	3	(11%)	1 Chronic, 2 due to windsurfer trauma
Headache	Arrival	2	(7%)	1 "Flu"
	Departure:	0	(0)	
Fatigue	Arrival	11	(39%)	See comment in text
	Departure	6	(21%)	See comment in text
Muscular weakness	Arrival	1	(4%)	
	Departure	0	(0)	
Tingling sensation in limb	Arrival	0	(0)	
	Departure	1	(4%)	1 Known spinal problem
Numbness in arm or leg	Arrival	1	(4%)	1 Post skiing trauma
	Departure	1	(4%)	1 Post skiing trauma
Dizziness or unsteady gait	Arrival	5	(18%)	1 "Stress + personal problems"
	Departure	5	(18%)	1 Severe middle ear barotrauma
Itchiness	Arrival	2	(7%)	1 Insect bite
	Departure	3	(11%)	2 Insect bites
Hearing loss	Arrival:	0	(0)	
	Departure	2	(7%)	1 Middle ear barotrauma
Failed SRT	Arrival	3	(11%)	
	Departure	6	(21%)	3 Middle ear barotrauma with probable transient inner ear dysfunction (see comment further in text)

Corrected data

Considering the previous comments, the data was corrected to exclude DCI symptoms and signs *probably due to other causes*. (Table 5)

Discussion and conclusions

Recruiting volunteers among the guests on a holiday resort is a challenging task. When the study was presented to arriving guests, many did show an interest. Unfortunately a large number of them got involved in other activities and did not show up the next day for the initial assessment.

Among the volunteers the study stimulated a lot of interest, they asked many questions and some were eager to visit the decompression chamber.

The results of the data gathered show no change in the prevalence of DCI compatible symptoms before and after the dives, this both in the uncorrected and the corrected data. As mentioned previously, no attempt to set a cut off point (for example number of symptoms) to establish the diagnosis of DCI was made. We preferred to observe symptom prevalence. As the prevalence is the same before and after the dives we can suppose that the study also suggests that there was no incidence of subclinical DCI among the population studied.

TABLE 5

NUMBER OF CASES WITH DCI "COMPATIBLE" SYMPTOMS UNEXPLAINED BY OTHER EVENTS (an unexplained failed SRT is considered a symptom)

Total of	Study Group		Control Group	
	On arrival	On Departure	On Arrival	On Departure
0 DCI symptoms	20	21	3	4
1 DCI symptoms	6	4	2	3
2 DCI symptoms	1	2	3	1
3 DCI symptoms	1	0	0	0
4 DCI symptoms	0	0	1	1

Unfortunately we cannot generalise those results to all recreational diving for two reasons.

Firstly, the study was carried out on a limited number of cases. Secondly, the diving in the Maldives is usually shallow, the best coral being between 2 and 15 m, therefore most divers did not come close to the maximum bottom time permitted by the decompression tables or computers.

The incidence of subclinical DCI might be higher among recreational divers diving on wrecks, which are often deeper and divers usually spend most of their bottom time at the maximum depth of the dive.

In this study we assessed volunteers at the beginning and at the end of their holidays, thus every diver would accumulate dives during that period making the study more sensitive for showing evidence of sub-clinical DCI. We assumed that as long as the volunteer divesd at least once every 24 hours the risk would be cumulative. However had such an evidence have been found, the calculation of an incidence per dive would have been very difficult. Furthermore the validation of the data with such a small control group would have been difficult. The numbers of factors that intervene during an entire holiday are huge.

An easier but probably less sensitive approach would be to assess the volunteers before a dive and then again 6 hours after. For such a scenario, the best control group would be volunteers who went on the same boat and snorkeled while the study group dived (therefore also being exposed to the boat, sun, water, food, etc). This could be carried out on one-day dive/snorkel boats.

The author is considering continuing this study in collaboration with various dive centres diving closer to the table limits (such as diving on wrecks). Any organisation interested in this collaboration may contact the author.

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DIVING DOCTOR'S DIARY

PULMONARY OEDEMA, DYSPNOEA AND DIVING

Carl Edmonds

Key Words

Cardiovascular, case reports, drugs, equipment, immersion, medical conditions and problems.

Introduction

Over a decade ago I was contacted by the physician on St Thomas Island in the Caribbean and asked: Why was it that there were so many surface snorkellers developing pulmonary oedema, and who also had hypertension treated with beta-blockers?

I explained to him the significance of the beta-blockers, i.e. the production of asthma-like states with obstructive airways disease, the reduction in effort tolerance and the possible detrimental cardiac effects of bradycardia of immersion. I was not then very interested in snorkellers. We certainly had observed the cardiac death risk in divers who were also on these drugs.[1]

I was reminded of this association by the most recent *Alert Diver*, which described two scuba divers with pulmonary oedema, in two separate articles.[2,3] The first was in a hypertensive female tourist taking beta-blockers (with the implication that if she had been seen by an Australian or New Zealand diving doctor, this would have been avoided).[2] The second was in a middle aged male who went on to cardiac surgery once the cause was clarified.[3]

I was impressed with the coincidence of recently reviewing two similar cases of cardiac induced pulmonary oedema, but with some interesting variations. These were similarly dyspnoeic, but in swimmers, not divers, and both were doctors. Each was identified as pulmonary oedema by the treating institution.

These two physicians present a not uncommon problem, similar to those of St Thomas', but one not well recognised; the production of dyspnoea associated with immersion per se.

Case reports

CASE 1

Late last year I received an e-mail from a Swedish pulmonary physician. She wrote:

"I found your interesting article via the Internet, on snorkelling deaths. I am 61 years old and visited Australia in October for a conference. We went to Cairns and tried snorkelling on the reef. In the water I got pulmonary oedema and had to be transported to hospital by helicopter and stay there for 4 days. I have never had problems like that before although I have some hypertension. I did not aspirate seawater and was not diving. I am not a good swimmer and it has made me a little nervous although I have snorkelled before. That cannot explain what happened. My question: Could it have been caused by a tight wetsuit or the dead space in the snorkel? I do not have large lung volumes, but I am physically active for my age.

Well, I survived and am not going to snorkel any more, but it would be nice to hear your opinion".

I am not enthusiastic about answering questions over the Internet, especially when I know that I am not going to get the full history and am denied the luxury of a physical examination, let alone appropriate investigations. I replied as follows:

"Before I respond to your questions, I need to know:-

1 What medication were you on (especially beta-blockers)?
2 Have you had a stress ECG? If yes, give the full report.
3 Any history of heart or lung disease (e.g. asthma)?"

The reply came almost before I had finished sending my questions!

"Dear Colleague,

Thank you for reading my question (Internet is fantastic).

1 I am on a beta-blocker, metoprolol tartrate (Metoprolol) 100 mg, and an angiotensin 2 receptor antagonist, candesartan cilexetil (Atacand) 16 mg.
2 No stress test was done before the snorkelling but it was before I left the Hospital in Cairns and that was without any problem. It was made because of a raised troponin level.
3 No history of heart or lung disease before. I am physically rather active for my age and my ECG at rest has been normal."

A discharge summary from Cairns Hospital indicated that the diagnosis was a cardiac induced pulmonary oedema, with myocardial ischaemia as the cause. She was commenced on Simvastatin and aspirin. The exercise stress test, while on beta-blockers, produced a maximum blood pressure of 220/100 and a maximum workload of 10.4 mets. There were no ECG changes or chest pains during exercise, but a U-shaped ST depression infero-laterally became more

horizontal during the recovery phase and was considered to be probably significant.

As so often happens is these cases, the patient's description was not completely accurate.

I informed her that I considered further investigations should be carried out regarding her cardiac status and that full lung function tests be performed. The latter were undertaken and there was no sign of any bronchial obstruction, even when on the beta-blockers.

CASE 2

This was more informative, because of the subsequent developments.

A 55 year old male had mild hypertension, was taking a beta-blocker (metoprolol) and aspirin, had no problems associated with scuba diving. But with surfing he regularly developed mild dyspnoea, 5-10 minutes after entering the surf, irrespective of the sea state. It would be relieved as he continued his surfing activities. Over 5 years this observation was verified on hundreds of occasions, but the dyspnoea seemed to be increasing in intensity and was affecting his aquatic fitness.

By the time he had his angiogram, which demonstrated multiple coronary vessel obstruction, he was experiencing dyspnoea not only during surfing but also with other exertion. He did not suffer angina pectoris in the aquatic environment, but this was becoming pronounced with terrestrial activities, being provoked by cold temperature, mild to moderate exertion, emotion, excessive food intake, etc.

Following a successful coronary artery bypass graft, all clinical evidence of cardiac ischaemia disappeared and he had no evidence of ischaemia on either the stress ECG or thallium scan.

When he resumption surfing he experienced no further dyspnoea with immersion and no apparent impairment of physical fitness during this activity.

This case demonstrates the successful influence of coronary flow restoration in the prevention of episodes of dyspnoea and pulmonary oedema during immersion.

Discussion

The pathophysiology underlying these cases was relatively clear because of the absence of any diving activity. With scuba diving there is a greater complexity in the development of dyspnoea and pulmonary oedema, as described in a recent diving medical text.[4]

During the 19th century immersion in a bath was one treatment given for "dropsy", demonstrating the effects of immersion on the redistribution of peripheral fluid.

Linnarsson assessed subjects with cardiac disease during an increasing work load, both in the water and on land, until symptoms or ECG abnormalities developed. The aquatic environment consistently masked the anginal symptoms of myocardial ischaemia.[5]

Diving diseases

Pulmonary oedema has been described in a variety of diving diseases. In some of these, it is a consequence of other diving respiratory pathology, such as in;

- the drowning syndromes, including salt water aspiration,
- pulmonary barotrauma,
- decompression sickness,
- underwater blast,
- some gas contaminants,
- pulmonary oxygen toxicity,
- some marine animal envenomations (e.g. the Irukandji syndrome).

In other instances pulmonary oedema has been described as the primary manifestation from a diving exposure.[6] Sometimes dyspnoea occurs in otherwise healthy individuals during scuba diving. The disorders occur while swimming or diving in shallow water, sometimes in the head-out position. The symptoms include dyspnoea, cough, haemoptysis and expectoration of frothy sputum.

The pathophysiological explanations advanced are often not completely convincing. Nevertheless there are sufficient such cases to warrant concern. The various presumed aetiological types are as follows.

Pulmonary oedema of immersion.

Immersion of the body, with the head above water, will have significant pulmonary effects.[8] Because of the hydrostatic pressure exerted by the water there is a redistribution of the blood (over half a litre) into the large intrathoracic blood vessels and, to a lesser extent, into the pulmonary microcirculation. Immersion also greatly affects the structure and function of the thoracic cavity. The following changes occur;

- reduced vital capacity (approximately 5%),
- reduced static lung compliance,
- increasing closing volumes leading to,
  - increase in functional air trapping,
  - increase in diffusing capacity,
  - increased flow resistance in airways,
  - altered ventilation, increasing in the apical regions

and reduction in functional residual capacity.

Pulmonary oedema and dyspnoea associated with cardiac disease.

This may be related to pulmonary oedema of immersion. Wilmshurst recorded the observation of pulmonary oedema, inducing dyspnoea in divers, associated with hypertension and postulated a number of aetiologies.[9] The stress factors which combine to induce this disorder include; increased cardiac loading due to the effect of water immersion, the respiratory changes noted above and cold induced peripheral vasoconstriction increasing the workload on the heart.

The effect therefore included increased cardiac pre-load, increased breathing resistance and exaggerated cardiac after-load.[10]

Some older divers, especially males, will develop dyspnoea 5 to 10 minutes after immersion. In these cases there is probably also a precipitation of cardiac ischaemia or arrhythmias because of the many physiological effects of immersion.

The following cardiovascular effects of immersion have been observed;

increased central blood volume, with blood passing from the periphery to the lungs due to the hydrostatic gradient,
increased stroke volume with increased cardiac output,
increased peripheral perfusion,
extrasystoles during the early phase of immersion (due to cardiac distension),
the diving reflex, when facial immersion and trigeminal stimulation induce bradycardia, shunting of the blood to the heart/brain axis from the periphery and viscera, and peripheral vasoconstriction,
the cardiac effects of cold exposure
and hyperbaric bradycardia, usually by 10 beats per minute, which is possibly related to increased oxygen partial pressure, increased gas density, increased hydrostatic pressure and a narcotic effect of inert gas.

Each or all of these may play a part in inducing mild pulmonary oedema and the respiratory symptom of dyspnoea. Subsequent investigation will demonstrate the presence of coronary artery insufficiency or arrhythmia and the problem will disappear when this is corrected.

Troponin estimations may supplement the traditional ECG and cardiac enzymes (CK and CK-MB) in determining cardiac involvement.[10]

Asthma provocation.

The scuba situation is likely to induce asthma in those so predisposed. There are multiple provoking factors, including;

exercise, especially if swimming against a significant tidal current,
breathing against a resistance (the demand valve),
breathing cold dry air (decompressed air),
breathing dense air (related to depth),
salt water spray inhalation (leaky demand valve) and
psychological stress and hyperventilation.

Cold urticaria.

Cold urticaria is a specific disease due to histamine release after exposure to cold. It may affect the skin, gastrointestinal tract and the lungs.

In scuba diving there is a reduction in breathing air temperature as the air pressure drops during its passage from the high pressure cylinder to the diver (Charles Law) and by-passes the nasal cavity with its warming and humidification effects.

Deep diving dyspnoea.

Cold gas inhalation at depth makes all divers susceptible to dyspnoea at great depths, due to convective heat loss in the airways and the local response to this.

Other non-diving disorders

Other non-diving disorders may present as dyspnoea while diving, because of the occasional exceptional physical demands of this activity. Examples are lung disease, drug effects from beta-blockers or irritants such as cannabis.

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Mr Ron Nishi
Senior Scientist, Experimental Diving Unit, DClEM, Toronto, Canada. Developer of the DCIEM Diving Decompression Tables.

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SPUMS NOTICES

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

Requirements for candidates

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the society, the candidate must comply with the following conditions:

1 The candidate must be a financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of examined courses in both Basic and Advanced Course in Diving and Hyperbaric Medicine at an approved institution.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months full time training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written research proposal in a standard format for approval by the Education Officer before commencing their research project.
5 The candidate must produce, to the satisfaction of the Education Officer, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research proposed, and obtain instructions before submitting any written material or commencing a research project.

All research reports must clearly test a hypothesis. Preference will be given to reports of original basic or clinical research. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the "Joint NH&MRC/AVCC statement and guidelines on research practice" (available at http://www.health.gov.au/nhmrc/research/nhmrcavc.htm). All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. It is expected that research project and the written report will be primarily the work of the candidate.

The Education Officer reserves the right to modify any of these requirements from time to time.

The Education Officer's address is Dr David Doolette, Department of Anaesthesia and Intensive Care, The University of Adelaide, Adelaide, South Australia 5005. Telephone(0)8-8303-6382. Fax (0)8-8303-3909. E-mail <David.Doolette@adelaide.edu.au>

Key Words
Qualification.

REGISTRAR PLACES FOR THE DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

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Fax +61-(0)8-9431-2819
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Key Words
Qualifications.

EMBASE

Readers will be aware that the South Pacific Underwater Medicine Society Journal is now indexed in the Elsevier Science medical data base, EMBASE. The information below has been gathered from pamphlets provided by Elsevier Science Secondary Publishing Division.

EMBASE is a current and comprehensive pharmacological and biomedical bibliographic database. It has extensive coverage of the drug-related literature, the database gathers information from more than 5,000 journals published in 70 countries. EMBASE contains more than 13 million bibliographic records from 1974 to the present, and over 555,000 records will be added annually. There are 5 million unique MEDLINE records included in EMBASE which creates an unparalleled single-file bibliographic resource. More than 80% of recent records contain full author abstracts. All bibliographic record source material has been subjected to a stringent peer-review process, while all other content on the site is continuously screened and reviewed. The result is high-quality, validated information. Fully indexed citations and complete author abstracts appear on average within 10 working days after receipt of the journal.

Each record contains the full bibliographic citation as well as indexing terms and codes. EMBASE records are indexed using EMTREE, a highly-sophisticated, hierarchically-ordered thesaurus with over 42,000 drug and medical terms (preferred terms), approximately 10,000 codes and close to 180,000 synonyms, including alternative drug names and MeSH subject heading used by the USA National Library of Medicine. Unique fields, such as drug trade and manufacturer names and device trade and manufacturer names, provide searchers with powerful record retrieval tools.

EMBASE.com is designed with a highly intuitive interface so there is little or no learning curve. Which allows a novice or experienced searcher to pinpoint the information needed quickly and easily. A system of powerful links allows navigation with ease through multiple layers of rich content so searches are both cost-effective and time-efficient. Records are displayed in an easy-to-read format, and special characters (such as Greek letters and superscripts) are displayed as in the original document, enhancing comprehension.

EMBASE can be accessed at http://www.embase.com which offers a free demonstration to experience the full functionality of EMBASE using a limited data set (approximately 30,000 records).

Key Words
Research

LETTERS TO THE EDITOR

BLOOD SUGAR LEVELS AND HYPERBARIC OXYGEN

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01/4/10

Dear Editor

I was most interested to read the paper by Drs Ekanayake and Doolette in the March issue of the Journal, "Effects of Hyperbaric Oxygen Treatment on Blood Sugar Levels and Insulin Levels in Diabetics".[1] Although the numbers in the study are small, I commend the work done in the paper and hopefully an ongoing study at Duke University will bring more data.[2] It is of note, that Ekanayake and Doolette's paper is the first study of diabetics in which insulin levels have been measured in addition to plasma glucose.

A couple of areas in the paper are worthy of comment. The statement in the 3rd paragraph on Page 19 "one must be cautious about the accuracy of in chamber glucometer testing." should extend to use of blood glucose meters outside the chamber in all HBO_2 therapy patients. The accuracy of blood glucose meters (bedside and laboratory) in the setting of elevated PO_2 blood samples as those found under HBO_2 is variable.[3]

In comparing 2 blood glucose meters I found with blood samples at PO_2 1,200 mmHg the Precision PCx (PCx, Abbott Laboratories; Bedford, MA) to underestimate by up to 46% of the SureStepPro (SSP, LifeScan Inc; Milpitas, CA), which has been shown to be unaffected by high PO_2.[4] The Precision PCx glucose meter system is based on a glucose oxidase method, but instead of the photochemical detection used by the SSP, uses an electrochemical detection technique. This technique incorporates a mediator molecule or compound in the strip chemistry, which shuttles electrons from the oxidation of glucose to the electrode surface. This generates a current that is proportional to the amount of glucose present that is measured by the system. Because molecular oxygen can compete with the mediator molecule for electrons there is the possibility, under high PO_2 conditions, of reduced electron shuttle from the mediator molecule to the electrode surface. The test strips are factory-calibrated with a fixed level of blood PO_2 that is intended to be consistent with the oxygen level of most blood samples. If blood samples have relatively lower or higher PO_2, test inaccuracies could result.[5] For example, samples with hyperbaric PO_2 values are more likely to have less current generated via the mediator molecule. This could in turn be falsely interpreted as a lower glucose value.

With all that in mind the current study employed a hexokinase method which, in theory, should be less affected by elevated PO_2, however, in the past has also been shown to be inaccurate.[6] A possible reason being the increased PO_2 reacts allostericaly with one or more of the enzymes used in the reagent test strip so that an increased concentration of formazan (the brown coloured compound detected by the glucometer) is produced.

Other factors in the current study that would point to blood samples with a low PO_2 would be using an antecubital vein especially if a tourniquet was used (not stated) and the time to measurement (not stated) but presumedly at least 30 mins since measurements were performed at an adjacent Institute. However, the ultimate reassurance of accuracy was the use of control patients that showed no drop in measured glucose values with HBO_2.

Although the cause of drop in blood glucose is still unclear, the authors exclusion of HBO_2 stimulation of insulin secretion as the cause is implausible, since long standing type I insulin dependent diabetics do not secrete insulin. Secretion tails off over the first few months after diagnosis and can be confirmed by measuring the level of C-peptides that are cleaved during endogenous insulin secretion. Changes in insulin levels in such patients (4 out of the 5 were type I diabetics) would have to be accounted for by absorption from the injection site.

I commend the studies approach in doing the control measurements prior to commencement of HBO_2. At Duke we are looking at the cumulative effect of HBO_2 on diabetics during a course of therapy comparing glucose values at treatment number 1,10, 20 and 30. However, rather than an absolute drop in glucose value early data shows a reduction in range of glucose values during a course of treatment which may relate to improved monitoring, diabetic management or clinical status. As to the cause of this current studies drop in blood glucose during HBO_2 I would favour the hypothesised mechanism of hypermetabolic state with increased aerobic metabolism. Although I have no data to substantiate this hypothesis the measured blood glucose in the current Duke study showed a significant drop only after 45 minutes of HBO_2 and with time continued to fall. This would be consistent with an ongoing metabolic process.

Finally, to those involved in the care of diabetics undergoing daily HBO_2 treatment make sure you take steps to minimise the potential inaccuracies of blood glucose meter readings. These steps include

1 Glucometer measurement outside (not inside) the hyperbaric chamber

2 Venous blood sampling (not arterial or capillary) using a tourniquet to minimise PO_2 level.

3 Preferably a reliable glucometer using a measurement technique that is PO_2-independent, such as the SureStepPro (LifeScan Inc; Milpitas, CA) and the YSI 2300 STAT PLUS (Yellow Springs Instruments, Yellow Springs, OH). However, I cannot recommend the Precision PCx (PCx, Abbott Laboratories; Bedford, MA) and would be wary of any system that uses the glucose oxidase method with electrochemical detection techniques.

David Vote

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Key Words

Diabetes, hyperbaric oxygen, hyperbaric research, letter.

Dr Vote's letter has been shown to Drs Ekanayake and Doolette. Their reply is reproduced below.

Anaesthesia & Intensive Care
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01/4/26

Dear Editor

We thank Dr Vote for his comments about our study on the effects of hyperbaric oxygen treatment on blood sugar and insulin levels in diabetics.[1] The evidence from previous brief reports and from our small study suggests a fall in blood glucose in diabetics during hyperbaric oxygen therapy and we await with interest the results of the Duke University and Prince of Wales Hospital studies. Dr Vote's comments are a reminder that monitoring of blood glucose during hyperbaric oxygen therapy is problematic as clearly demonstrated in his and colleagues' careful study of two blood glucose meters.[2] It was an oversight on our part not to give more detail of the blood sampling and analysis methods, particularly since one of us was fortunate to see a pre-print of Dr Vote's manuscript before submitting our own.

In our study, blood was collected from the antecubital or a forearm vein as described, without a tourniquet. Samples were collected into fluoride-oxalate tubes and batch analysed by the hexokinase method under normobaric conditions following each study; the time between collection and analysis therefore differed for each sample but exceeded one hour. Samples from all four arms of our study were treated identically. Although PO_2 was not measured, we believe that the PO_2 of these samples would not have been elevated at the time of analysis and therefore not interfered with glucose measurement. As Dr Vote also points out, the use of control patients provides reassurance of accuracy. Edge's earlier study of the hexokinase method is an important illustration of the difficulties of glucose monitoring but is not directly comparable to the present study since it showed that a glucometer inside the chamber at 3.7 bar absolute air pressure produced falsely *elevated* glucose readings compared to instruments outside the chamber.[3]

We agree with Dr Vote that, in the case of insulin dependent diabetics, it is improbable that hyperbaric oxygen decreases blood glucose by stimulating endogenous insulin secretion, as has been previously hypothesised.[4] Indeed our study provides evidence against this proposed mechanism since serum insulin did not increase during hyperbaric oxygen therapy in any of the subjects in our study.

Lalith Ekanayake and David Doolette

References

1 Ekanayake L and Doolette DJ. Effects of hyperbaric oxygen treatment on blood sugar levels and insulin levels in diabetics. *SPUMS J* 2001; 31 (1): 16-20

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Key Words

Diabetes, hyperbaric oxygen, hyperbaric research, letter.

BOOK REVIEWS

ESSENTIALS OF DIVING SAFETY
Wesley Y Yapor, MD.
ISBN 0-941332-88-8. Published 2000.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 11.95. Postage and packing $US 7.00 Global Priority Mail. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

This is another of the Best Publishing Company Divers*ification* Series. It is a paperback with a glossy cover, 133 pages and a basic index. It is good value for those who want to learn more about diving safely.

The author takes a belt and braces (suspenders for speakers of American English) approach to diving safety. The book is divided into three chapters, equipment, technique and travel. Each chapter is full of commonsense advice that is often ignored by many divers. The opening paragraph of chapter 1 deserves to be quoted in full. "There are a few general recommendations that should be made concerning the care of diving equipment. It is not uncommon for a diver to leave a dive site missing a piece of equipment or with an extra glove or boot that belongs to another diver. For this reason, it is recommended you label all of your gear with a permanent marker or label. Writing the owner's initials or name on every item will simplify the identification of a diver's equipment. A careful inventory of one's gear should be done before departing for a dive and before anyone leaves the site." Be careful about everything, is advice to be expected from a neurosurgeon from a teaching hospital in Chicago, who holds a US Coast Guard licence as master of a charter boat of up to 50 gross tons.

Unfortunately this excellent book is a little spoilt by a mistake on page 4 where, under the heading Weight Storage and Spare Weight the following words appear "spare weights, enough to supply two divers (about 50 lb or 110 kg)" appear. Of course all our readers know that 110 kg is 242 lb and that 50 kg is 110 lb and that 50 lb is 22.7 kg. It is an understandable mistake that someone who thinks in pounds, as United States citizens do, could have multiplied by 2.2 when dividing was required. The proof reader should have picked this howler up. However much searching for other errors of fact failed to find any. As this is the only mention of metric measurements I suggest that it be deleted from the next edition.

An example of Dr Yapor's thoroughness is his advice about accessories. There are 31 items in his table of suggested diving accessories. It includes a First Aid kit with the notation (see section on First Aid Kit), spare tank, adhesive tape, neoprene glue and binoculars in case a search becomes necessary.

He offers sensible advice on diving from boats, including the duties of both customers and crew. He offers advice about equipment configuration, find out what suits you best, and the pros and cons of alternatives. This is one of the few books to offer a selection of actions to control one's ascent when the weight belt comes undone. He is a believer in the Sausage Buoy as an essential accessory, because it is easier to spot than a diver's head.

The travel chapter is short but covers altitude, air travel, equipment to take, currency, clothing and medications as well as other topics.

The appendix is the Emergency Procedures manual of the ship *Myles O'Joy*. This is suitable for US emergency services, but change the numbers to contact and it is good advice the world over.

I strongly recommend that all those who read this issue of the Journal get a copy.

John Knight

Key Words
Book review, diving operations, diving safety, emergency ascent, equipment, rescue, travel.

THE WHITE DIVERS OF BROOME
John Bailey
Pan Macmillan Australia, Sydney.
The publishers supplied the review copy,
RRP $Aust 31.00 inc GST

The Prologue to the book is an elegantly written word picture of a postmortem examination of a diver. This prologue is well justified as it sets the scene for the fascinating true-life tale that follows. The style is that of the historian novelist, with the facts, well researched and referenced, embellished with words and action that while being fiction ring with truth and greatly add to the book.

The book is set against the backdrop of Broome in 1912 and the pearl shell collecting industry that powered the thriving town. The Master Pearlers were a group of rich, opportunistic men who had little regard for the life of the pearling lugger crews. The "White Australia Policy", which was being imposed by the Federal Government, led to the Master Pearlers being in conflict with this policy, as they saw it as a direct threat to their profits.

The terms used in the book, I suggest, will jar any reasonable thinking persons sensibilities. They are cruel words that describe the racial mix of Broome at that time. The author was brave to include these racially prejudiced

terms, but without them the powerful message contained in this book would have been radically diluted by using more politically correct terminology.

In this era the population of Broome was as many Asians as Australians. Asian labour was cheap to hire and easy to replace. The White Australian Policy attempted to remove the Asian work force from the cane fields of Queensland and the pearling industry, which depended on these workers. The divers employed in 1912 were mainly Japanese. A Federal Government initiative, to import trained ex-Royal Navy divers and tenders, was intended to prove that the white man was superior to the coloured and end the Asian stranglehold on the pearling industry.

The experiment lasted only one season before about half of the divers were either in jail, dead from drowning or "The Bends". From the outset the white Master Pearlers wanted it to fail and, indeed, seemingly acted to ensure this outcome. The book offers details about the diving equipment and how it was used. As far as I can tell the diving content is in the main accurate, but not without error. But this is a minor point.

The story vividly conjures up Broome, with wonderful descriptions of the segregated white and Asian ends of town. The lanes and slums, the noodle stalls, opium dens and brothels were more in keeping with Asia than Australia. Side issues about Broome society, white, Australian aboriginal and the mix of Asians, in schooling, justice and social events bring to the reader the racist, brutal existence that was Broome. Pearl shell mattered more than life.

This book is much more than a tale of pearl diving. It is a sad recollection of what it was like to live in the very prejudiced, early days of Federation. A window on the past that echoes many of the same fears and hopes held in society today. White divers of Broome is a fascinating and utterly absorbing true-life historical drama, that should be read by everyone.

Bob Ramsay

Key Words

Accidents, book review, deaths, decompression illness, equipment, history, occupational diving.

A DEMONSTRATION OF THE DIVING ENGINE

Jacob Rowe. Edited by Michael Fardell and Nigel Philips. Historical Diving Society (HDS) in association with the National Maritime Museum UK.

Available from Historical Diving Society, 25 Gatton Road, Reigate, Surrey, RH2 OBH, UK. Telephone +44-(0)1737 249961.

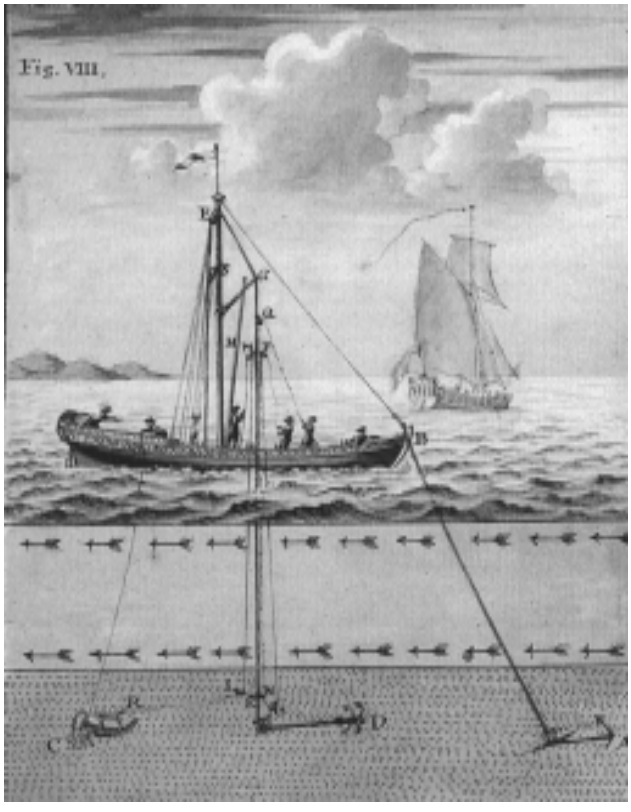
Price £18.00. Postage and packing about £6.00 for delivery to the Australasian region.

This book is a facsimile, a complete reproduction of the earliest monograph in English on diving. The original was written about 1730. In 1980 the manuscript was purchased at auction by The National Maritime Museum from Christies in London. But this book is more than just a facsimile of a hand written document. The original text has been greatly enhanced by the addition of an introduction, a transcript of the original manuscript and biographical notes on the author. Michael Fardell and Nigel Philips are to be congratulated on their work. Until now this unfinished short text has remained unpublished.

The introduction, like all sections of the book, is extensively and well referenced. It offers the reader insight to the times and practice of the developing sunken ship salvage business of those times and they seem to have been interesting times indeed. Many questions that researchers of diving history may have about the technicalities, and indeed the feasibility, of the Lethbridge style barrel for diving have been addressed in this introduction, which also includes contemporary reports of Rowe's exploits as a salvor.

The publishing of this monograph and its supporting material is a great step towards the proper documentation of the late 17th and early 18th century period when the proliferation of new diving equipment led to men entering the sea for prolonged periods for the first time.

The transcript of the original manuscripts allows researchers to concentrate on the words and not have to



This illustration accompanies the original description of "The manner of diving in rapid tides or currents" and is used on the jacket of Demonstration of the Diving Engine.

decipher the original hand written 1730s' script which is also reproduced in full.

The publishers have endeavoured to faithfully reproduce the script and the delightful detailed drawings on quality paper of a suitable texture and colour. They have achieved a great effect that has been well worth their efforts. Their work can be judged by the copy of the jacket picture which appears on page 84. The book of 39 pages of 264 mm x 189 mm (Crown quarto) is case bound with dust jacket. I suggest that the limited and numbered first edition (750 copies) will sell out quickly as is fitting for this fine piece of work.

The Historical Diving Society and the National Maritime Museum are to be congratulated for this joint effort. Diving history needs many more such important pieces of our diving history to be well researched and published for the greater enjoyment of all interested parties. This book is a fine example of how this can be done.

Bob Ramsay

Key Words

Book review, equipment, history, occupational diving.

SPUMS ANNUAL SCIENTIFIC MEETING 2000

IS SCREENING FOR PATENT FORAMEN OVALE FEASIBLE?

Jürg Wendling, C Balestra and P Germonpré

Key Words

Cardiovascular, decompression illness, equipment, medical conditions and problems, medicals, risk, safety.

Introduction

More than 50 % of decompression illness (DCI) are unexpected, which means after regular dives without incidents or rapid pressure changes. As bubbles arise from the veins after most of the dives, cerebral embolism from bubbles by-passing the lungs, for instance by shunts through a patent foramen ovale (PFO), has been discussed. There are however other shunts possible and cerebral arterial gas embolism (CAGE) arising from air trapping in divers with lung pathology has also been proposed.[1] In a recent study Wilmshurst demonstrated that only a negligible part of unexpected DCI cases could not be "explained" by either a PFO or a lung pathology.[2]

Rationale

Bove, in a retrospective study, and Germonpré, in a case control study, recently determined the increase in probability for PFO related DCI as, respectively, 2.5 and 3.7 times non-PFO probability.[3,4] As almost 30% of all divers could have a patent foramen ovale, the non-diving population prevalence, one can wonder why DCI is not much more frequent than it is. Balestra demonstrated that, even with significant shunts, there is a need for an increased right-left atrial pressure gradient for bubbles to pass through shunts.[5] These gradients can be caused by intra thoracic pressure (ITP) rises. The investigators measured ITP by inserting a balloon tube into the oesophagus, filling the system with water and measuring the pressure in cm of water above the pressure during normal respiration. One of the most effective methods of raising ITP is a prolonged, forced Valsalva manoeuvre, but larger rises in ITP are produced by knee bends while performing a Valsalva as well as coughing and pressing down as hard as possible while holding one's breath. Figure 1 (page 86) compares the intra thoracic pressures reached.

The test manoeuvres charted in Figure 1 were:

Control

Maximal isometric arm and chest muscles exercises: while sitting in a standard position (with knees and hips in 90° flexion) with the hands one above the other and the arms extended forward horizontally, the subject had to push down as hard as possible on a set of scales , placed on the ground, by means of a wooden stick while holding his or her breath. This test was performed three times; the mean push-down force was noted, and the mean ITP reached was used as the control ITP value for the other tested manoeuvres.

Gentle Valsalva

Valsalva manoeuvre (as usually performed by the diver to equalise middle ear pressure).

Forced Valsalva

Valsalva manoeuvre with the subject blowing as hard as possible.

Calibrated Valsalva

Valsalva manoeuvre gradually increasing ITP until the ITP reached the level of the first maximal isometric exercise.

Cough

Forceful coughing.

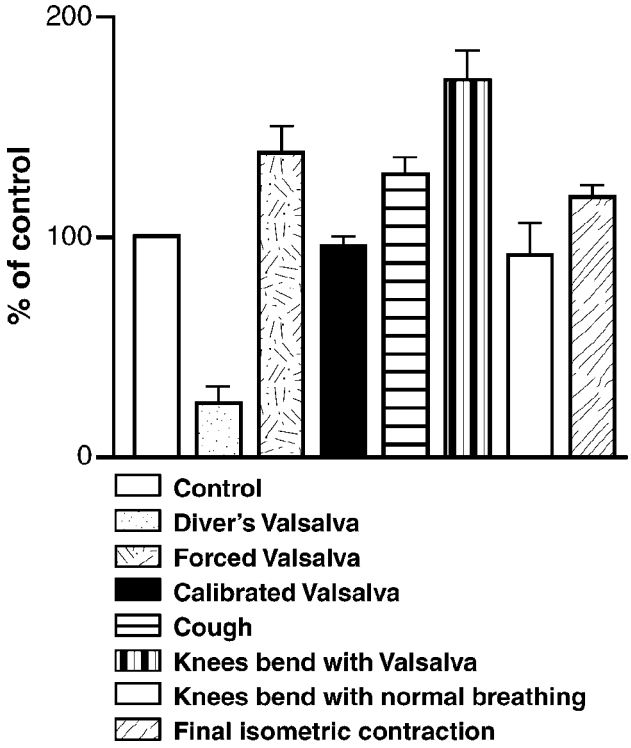


Figure 1. Intrathoracic pressure increase produced by different manoeuvres (from[5]).

Knees bend with Valsalva

Knees bend (squatting) with the subject performing a forced Valsalva.

Knees bend with normal breathing

Knees bend while the subject breathed normally

Final isometric contraction

A repeat of procedure 1 after the subject had completed tests 2-7. Care was taken to ensure that the same push-down force was reached.

Even with large PFOs (type II), the length of the ITP rise is of great importance in producing a considerable shunting volume in the pressure release phase, probably due to flow pattern characteristics in the right atrium.[6] Surprisingly, the normal diver's ear clearing manoeuvre produces almost no ITP increase. Figure 2 shows that a diver's ear clearing Valsalva results in an ITP rise of 5 mmHg while a forced Valsalva reaches about 105 mmHg! Small PFOs and an insufficient bubble load result in negligible shunting. Some pressure inversion between the atria is probably present in all respiratory patterns as shown in Figure 3,[7] but may be insufficient to produce large bubble transmission because of their short duration. Besides the ITP peaks during eventual post-dive exertion, such as raising the anchor or lifting cylinders out of a boat, there is a gradual rise in mean pulmonary artery pressure (MPAP) after decompression dives, induced by bubble embolisation of the lung vasculature.[8] This retrograde increase of the right atrial pressure might open a PFO, allowing bubble shunting, if sufficient bubbles are present in the veins.

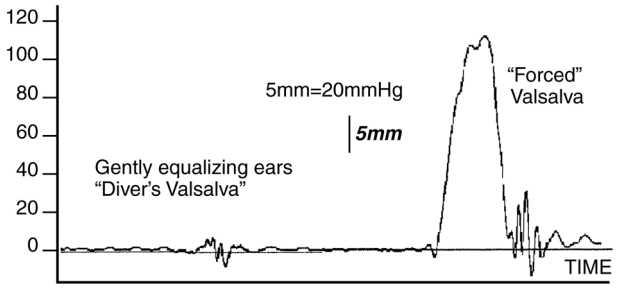


Figure 2. Intrathoracic pressure registration during normal pressure equilibration and forced valsalva manoeuvre (from[5])

The exact risk of diving with a PFO can only be determined by means of a prospective study. As DCI is a very rare event, a study with a large number of monitored divers is necessary to determine the risk increase. Such a study can only be conducted if an appropriate "screening" method for PFO could be designed. Then screening of divers could well be justified if the procedure was simple and there was no risk of unwanted consequences.

Feasibility

Beside the heavily invasive procedures like blood gas analysis and Swan Ganz catheter studies contrast-transoesophageal echo sonography (C-TEE) is recognised as the gold standard for PFO detection. Transthoracic echo (TTE) and transcranial doppler (TCD) examinations are not sensitive enough for screening purposes. Table 1 compares the sensitivity and specificity obtained with different techniques from,[6] after Di Tullio.[9]

As TEE is invasive, expensive and restricted to experienced cardiologists, we have proposed a screening technique using a much simpler Doppler bubble detector and monitoring the carotid artery.[10] As at the time no data were available, Germonpré and Balestra evaluated this technique in a prospective randomised study. They found that with their standardised procedure, described below, the sensitivity was as high as 100 % and specificity 88% when compared with TEE.[6] The intravenous injection of micro-bubbles as described here is a safe procedure and it is routinely used in TEE as a common cardiological practice.[11] However, for safety reasons, oxygen should be available and the diver should not have dived within the last 24 hours. The procedure is described below:

Standardised technique for carotid artery Doppler screening for PFO

1 Insertion of intravenous cannula to the right ante-cubital vein.

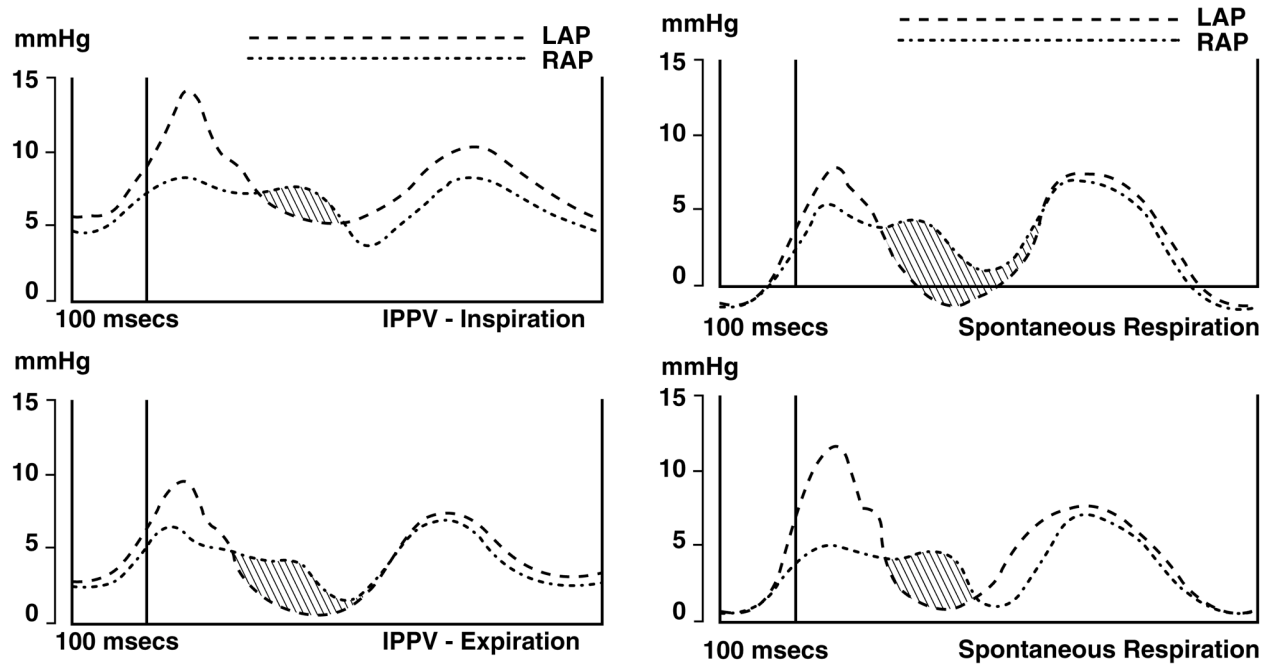


Figure 3. Left (LAP) and right (RAP) atrial pressure tracings after cardiac surgery during controlled ventilation (IPPV) (3a) and spontaneous respiration (3b); from Cambier.[7]

TABLE 1

CONTRAST ECHO-DOPPLER TECHNIQUES

Method	Abbreviation	Sensitivity	Specificicity
Contrast-transoesophageal echosonography	C-TEE	100%	100%
Contrast-transcranial Doppler	C-TCD	68%	100%
Contrast-transthoracic echosonography	C-TTE	47%	100%
Contrast Carotid artery Doppler	C-CD	100%	88%

2 Connect to a saline infusion and prepare 2 three-way taps and two syringes of 10 ml (Fig 4)

3 Fill syringe 1 with 9.5 ml normal saline + 0.5 ml air.

4 Mix by pushing the content from syringe 1 to syringe 2 and back 10 times (produces a micro-bubbles solution).

5 Inject immediately thereafter the whole content within 3 sec and continuously monitor the Doppler signal from the left carotid artery. If bubbles have been shunted through a PFO they will be detectable within 3 seconds after injection (bubbles appearing later than that will still have been shunted but through the lungs or elsewhere). During this injection, the patient should breathe normally.

6 Repeat steps 3-4 after 1 minute.

7 Perform a 10 second forced straining manoeuvre (take a deep breath, hold that breath and "push down" in the abdomen for approximately 10 sec). Inject another syringe of 10 ml with bubble mix, and instruct the patient to suddenly release the straining manoeuvre when the whole syringe is injected. Continuously monitor for bubble sounds at the carotid artery.

8 Repeat steps 6-7 twice.

The signal should be recorded on tape for later study.

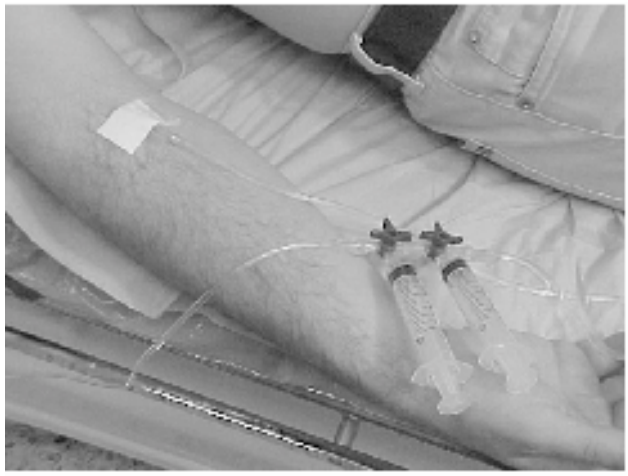


Figure 4. Injection device in situ, with two three-way taps and an intravenous catheter.

Consequences of a positive screening test

At present there is no generally approved consensus about the consequences of a positive PFO finding. Some diving medicine specialists would categorically declare anyone with a known PFO unfit, however the majority would not ban further diving. Professional divers are generally (HSE in England and others) declared unfit after DCI with neurological symptoms. Recreational divers rely on the notion of "acceptable risk". From a pathophysiological point of view, the risk of diving with a PFO must be higher than without PFO. However, since the actual cause of DCI is the gas bubbles in the blood, this risk can probably be reduced to within acceptable levels by any diving procedure that reduces the bubble load after the dive. A few possibilities are:

1 Use of dive tables calculated to reduce the supersaturation threshold to 1.4 (or 1.2 or even 1.0), which results in low-bubble-diving (almost zero bubbles). A similar effect can be obtained by setting the dive computer mode to altitude. The exact effect on threshold and DCI probability still remains to be determined.

2 Breathing nitrox while diving using air tables or a computer set for air diving, which will reduce decompression stress proportionally.

3. Avoid ITP peaks, that is no effort after exiting the water, use the Frenzel manoeuvre instead of the forced Valsalva for clearing the ears (Fig.5).

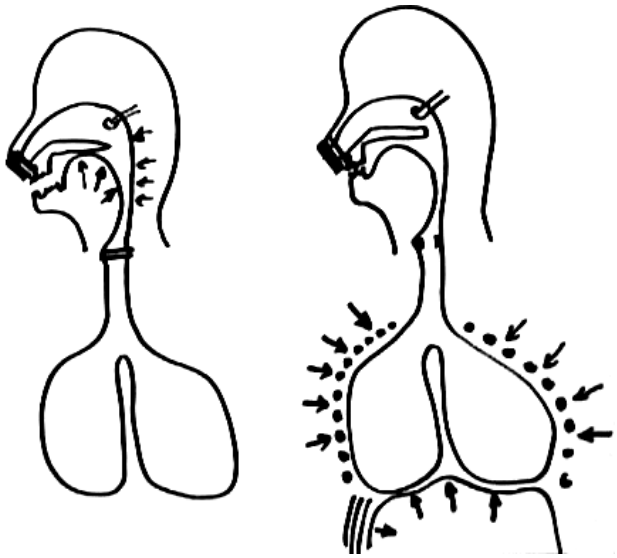


Figure 5. Two different methods for pressure equilibration in diving: the Frenzel manoeuvre (left) is performed by swallowing when the glottis is closed. The ITP remains normal. Performing a forced Valsalva (right) the thoracic and abdominal muscles produce a rise in ITP which is transferred to the ear through the open glottis and throat.

Conclusions

1 Carotid artery Doppler examination with saline bubbles injection and a standardised straining manoeuvre is a cheap, easily learned, minimally invasive and not painful screening method with very good sensitivity and specificity.

2 In victims of DCI, detection of PFO should be performed by a c-TEE, following a standardised technique.[5] For screening purposes, the Carotid artery Doppler examination provides good sensitivity and specificity.

3 PFO positive victims of DCI must be advised how to reduce the risk for decompression disorders by changing their diving practices (reduce DCI probability, special tables, altitude mode in computers) and avoiding elevation of ITP and changing their pressure equilibration practice (Frenzel manoeuvre). However, divers should stop diving if neurological DCI symptoms are due to other causes including lung disease.

4 Screening of divers should be done only in the context of a prospective longitudinal study. Data from such a study will contribute to the quality of scientific discussions about PFO. In the end we will have more evidence-based diving medicine and understanding of the pathogenesis of unexpected decompression illness.

5 A prospective study has been initiated by DAN Europe Research Division, aiming at screening 4,000 divers, with a follow up period of 5 years. Participation is encouraged (see <www.daneurope.org/research/carotid.htm>

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FITNESS TO RESUME DIVING AFTER A DECOMPRESSION INJURY

David Elliott

Key Words

Diving medicals, fitness to dive, medical conditions and problems, recreational diving, standards.

There are guidelines for the routine medical assessment of divers but, for post-accident assessment, the guidelines are less prescriptive. The circumstances of each decompression-related accident are so different that an even greater emphasis must be placed on the knowledge and experience of the examining doctor. The medical assessment of those who wish to return to diving after a decompression injury rests on the same basic principle as any other medical assessment of recreational divers, future in-water safety. Provided there has been a full recovery of function, a resumption of diving should not be associated with any greater in-water risk than before, particularly as decompression accidents rarely manifest before surfacing. The spotlight turns to longer-term considerations including the likelihood of another incident and the possibility of secondary long-term health effects.

But those people used to assessing recreational divers should be aware that, in any such assessment of a working diver, early consideration should also be given to motivation. If the diver really does want to get back into the water, this is not a problem but if, after apparent recovery, he/she might be looking for some medico-legal benefit from being unable to return to diving, then it will be difficult to declare them fit.

Immediate lay-off

Guidelines have been issued for naval and commercial diving that prescribe minimum lay-off periods after a full recovery, which usually occurs during recompression. From the time of full recovery a set number of days is recommended for each of the categories of decompression injury after which, unless the injury was only joint pain, the diver must be assessed by a doctor. In many treatments of working divers there was a chamber close to the site of diving operations and so, unlike recreational diving incidents, recovery has not been compromised by extended delay before recompression.

The recommendation of the US Navy Diving Manual is that a diver may return to diving 7 days after surfacing from a Table 6 recompression for a simple uncomplicated limb-pain only incident.[1] It recommends that a diver may resume normal diving 14 days after the treatment of patchy peripheral sensory-only neurological decompression illness that completely responded by the end of the second oxygen period at 60 ft (18 m). Those who have suffered any cardio-respiratory or any neurological manifestations (other than just peripheral and transient paraesthesia) should remain out of the water for four weeks and then should then be reviewed by a diving medical officer. Should any of these serious cases have warranted treatment on USN Table 4 or USN Table 7, the lay-off should be for at least three months. An assessment by an experienced diving doctor is recommended before the diver resumes diving.

The Diving Medical Advisory Committee (DMAC), which is recognised by the HSE in the UK and world-wide by the International Marine Contractors Organisation, recommends the following minimum periods before considering a return to diving after decompression illness.[2]

A *Limb pain, cutaneous (skin rash with severe itching), lymphatic (swelling of tissues) or non-specific (persistent headache, excessive fatigue, loss of appetite, nausea) manifestations only*

i With uncomplicated recovery, 24 hour lay-off.
ii Where there has been a recurrence or relapse requiring further recompression. 7 day lay-off.

B *Neurological or pulmonary manifestations*:

i Altered sensation involving the limbs only: 7 day layoff. Return to diving only after review by a diving medicine specialist
ii Other neurological (including audio-vestibular) or pulmonary manifestations: 28 day lay-off and return to diving only after review by a diving medicine specialist

C. *After an incident of pulmonary barotrauma resulting in a pneumothorax or mediastinal/ subcutaneous emphysema*.

The diver should be assessed by a diving medicine specialist. Return to diving may be permitted, but not normally until at least 28 days following complete recovery.

DMAC further says that in cases where there are significant residual neurological manifestations, even after repeated treatment, the diver should normally be considered unfit to dive. Return to diving should only be permitted if sanctioned by a diving medicine specialist.

For the recreational diver, no such prescription is appropriate but some time on the beach is advisable before returning to the water, and only then if a full clinical recovery has been made. During that period of diving idleness, no doubt the diver will contemplate the risk of another incident against the relative safety of retiring from this particular recreational activity. The factors to be considered are much the same as those confronting a working diver though the weighting applied to each factor may be different.

No residua ?

Conventional wisdom suggests that after treatment for an episode of decompression illness, the diver may return to the water provided there are no objective residua, but this statement is an over-simplification. How are these residua defined? Is it just the presence of some muscular weakness or a patch of skin with sensory impairment, or are the results of special investigations needed?

The reason for concern is based upon the knowledge that in decompression illness, any neurological manifestation has probably left a permanent scar. So, except perhaps in the most rapidly treated cases, there is probably a diminished reserve capacity in the brain and cord for any future episode from which functional recovery may be required.

Possible investigations have included MRI, SPECT scans, electrophysiological and neuropsychometric investigations. The use of such investigations has been confounded by the findings of alleged "abnormalities" in perfectly healthy divers who have never had an incident. So, if there is a shadow on a scan, a delay on an evoked potential, or some diminution of short-term memory, what does that mean? It may be that the patient is better served by a review of other factors related to his/her bend.

The nature of the dive

Was the bend "deserved"? Did the decompression incident arise from a dive which was within the accepted envelope of safe decompression tables but which, for whatever reason, had not followed accepted decompression procedures? For instance, out-of-air leading to omitted stops. Repetition of such an incident is avoidable by better dive management.

Was the bend "unexpected"? Did it follow a safe dive using accepted tables? If so, the individual may be "bends susceptible" and its repetition cannot be so easily avoided. If the incident was neurological, and particularly if it was the second such incident, consideration should be given to echocardiography to detect a PFO. However because some 25-30% of the population has a PFO, its association with the causation of neurological decompression illness is far from absolute and other factors must be reviewed.

The number of decompression incidents

Depending on circumstances the number of episodes is not necessarily a critical factor but bends-susceptibility does appear to be a real phenomenon. It also appears to be transient, associated with some dive groups for a while, but not with other groups on similar schedules. One wonders if there might be some non-diving factor here, such as the subjective reporting threshold for symptoms, but this cannot be proved.

There have been some working divers, with *no* PFO and *no* neurological residua, who in the past have been medically disqualified after two mild neurological incidents, each treated immediately with complete relief. For a breadwinner this is a tough decision that may not seem justified and one hopes that similar cases in the future are reviewed with a more comprehensive assessment.

The nature of the decompression illness

Latency is a useful indicator and the more rapidly the onset arises after a dive, the more severe is likely to be the injury. The various manifestations have been categorised traditionally into mild (such as cutaneous and limb-pain) and serious (such as chokes, staggers and paresis). This can be misleading during the management of acute illness because the patient may be progressively deteriorating. When used retrospectively however this provides a simple guide to severity.

Within the neurological manifestations, Pitkin et al.[3] have tested an established scoring system[4] that provides a broad prediction for the success of treatment or severity of outcome. The variables used to assess each case are

Repetitive dive, yes/no.
Improvement, stability or deterioration of neurological symptoms before recompression.
A sensory deficit detectable to the examiner.
The extent of any motor impairment.
Urinary disturbance, yes/no.

Each answer is given a weighting (0 - 6) and the total score provides a guide to outcome. A low score was associated with a favourable outcome whereas a high score, though including all the severe outcomes, was less predictive.

The nature of the treatment

Some other factors, such as delay between onset and recompression, may need to be considered for prediction in these neurological cases. However Ross[5] has shown that, when all DCS presentations are considered together, outcome is not related to the delay. The tendency is for the minor cases (which numerically are in the majority) to improve with time thus obscuring deterioration in the neurological minority. This delay is a factor in maybe 15% of all DCS cases in his series. The relationship between delay and outcome is further confounded because the seriously ill tend to get accelerated attention. In two studies, outcome was related to clinical condition at the time of recompression.[5,6]

Was the patient recompressed and, if so, what was the delay before recompression? More importantly, was the response to treatment rapid and complete? Or was it slow? Was there any deterioration at pressure and, during or after the treatment and relapse or recurrence. Were one or more repetitive treatments with HBO required after the first recompression? One is tempted to specify what table was used but, whatever the treatment, all one needs to know is how effective it was.

The absence of detectable residua has already been mentioned whereas the advice to those with some residua will be discussed later. On the grounds of the potential consequences of another decompression incident upon an already damaged cord, a sports diver who has had one severe neurological incident, should be recommended to give up diving.

Nevertheless, a restriction to shallow diving may be appropriate for a few. Consider the ex-commercial diver who had a bad spinal bend with some minor residua, but who has no functional deficit. Consider the sport diver who had an "undeserved" but very mild neurological decompression illness after a "safe" decompression dive and who is then found to have a PFO. A shallow diving limit may be appropriate but there are other restrictions that might be more practical. One example is simply to restrict no-stop times, either by going "one down" on the tables or by using a decompression computer that at sea-level is switched to the altitude mode. Of course this can make the diver very unpopular with others because he/she always needs to surface early. Regular diving can be considered by someone who is prepared to go to the trouble and expense of nitrox-scuba training. This is not used to increase no-stop times (on the usual grounds that there is less nitrogen uptake) but instead the normal tables or computers for air diving are used when in fact breathing nitrox. No guarantees, but this should provide a good safety margin.

The assessment

Whatever the reason, if an examination is needed, it needs to be done by someone who is competent, a doctor who has a full understanding of the diving environment. A medical practitioner who is merely trying to follow printed guidance may reach an inappropriate conclusion and will be unable to give appropriate advice to a disabled diver or to a diver after some illness, surgery or accident. Any consideration of a restricted certificate of fitness must be based on a thorough understanding of the environment's possible demands upon the impaired diver.

In following up persons after decompression illness, long term surveillance for dysbaric osteonecrosis should be considered in order to detect any juxta-articular lesion before the articular surface is damaged.

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WHY DIVERS DIE
Two more cases

Rees Jones

Key Words
Cerebral arterial gas embolism, case reports, deaths, decompression illness, incidents.

Introduction

The Poor Knights Islands lie 25 km offshore from the Northland coast of New Zealand. The surrounding waters are bathed by the East Auckland current which brings warm clear water from subtropical areas of the eastern Australian region, and with it eggs and larvae of species of invertebrates and fish seldom seen on the mainland coast. The islands are nature reserves and the surrounding waters are marine reserves. As a consequence this island group is a Mecca for divers, both from New Zealand and overseas tourists.

During the summer tourist season, commercial operators together take up to a total of 100 divers per day to these islands with maybe another 50 sport divers going out in private boats. Popular dive sites range in depth from one or two metres (Lost World) to 40 m (Northern Arch). In past years fatal dive accidents were not unknown at the Poor Knights, but since the mid 1980s there have been only two recent deaths that I am aware of. These are presented here as case histories and show features which may indicate that the victims should perhaps not have been scuba diving.

Case 1

On 26th December 1997 diver A, a Caucasian male aged 55, was on a trip on a commercial dive boat with four companions, including two sons who had given the trip as a birthday gift to their father, a certificated diver with some years' experience. The dive site was at Northern Arch and the dive commenced at 1100. The group descended slowly as diver A had been feeling unwell before the dive. The maximum depth reached was stated to be 36 m. The total dive time was stated to be 25 minutes. During the ascent, at a depth of about 20 m it was noted that he had separated from the group. He was subsequently found floating on the surface apparently unconscious. He was rescued onto the dive boat and resuscitation was commenced. The Emergency Services helicopter was called and a paramedic was lowered who pronounced him dead.

EXTERNAL EXAMINATION

There was an old upper mid-line laparotomy scar. Otherwise there was no significant abnormality. A computerised axial tomography (CAT) scan was performed before the formal post-mortem examination 24 hours after death. This showed gas, (presumably air, but no analysis was undertaken) within intracranial arteries (Figure 1) and within the ventricles of the heart and aorta (Figure 2) confirming arterial gas embolism (AGE). The lungs showed shadowing consistent with haemorrhage or aspiration of gastric contents.

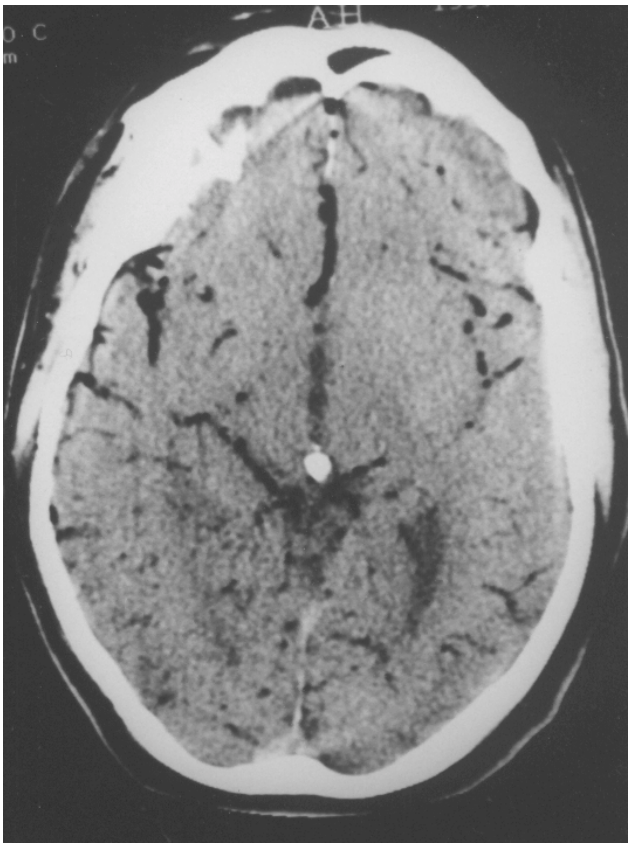

Figure 1. CAT scan of diver A's head before autopsy. The front of the head is to the top. Gas, black areas, can be seen in the intra-carnial arteries.

INTERNAL EXAMINATION

Cerebral artery branches on the surface of the brain showed small bubbles consistent with the CAT scan findings. There was no other abnormality of brain either externally or when sectioned.

The heart was slightly enlarged, weighing 450 g. All three of the major coronary arteries showed atherosclerosis with considerable narrowing of the right and of the anterior descending branch of the left coronary arteries. The left ventricular myocardium showed a healed infarct posterior-inferiorly, adjacent to the septum. Microscopy showed this to be at least three months old. The valves and chambers were otherwise unremarkable; in particular a foramen ovale was not demonstrable.

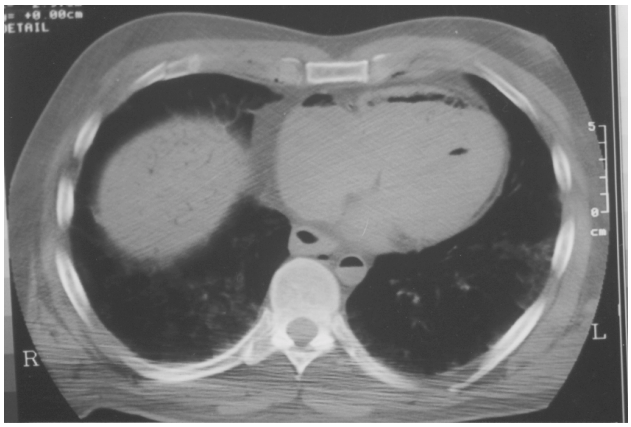

Figure 2. CAT scan through diver A's chest. The left side of the body is on the right of the illustration. The grey area in the right lung is liver. The spotty grey areas in the lung fields are haemorrhages or aspirated fluid.

The trachea and bronchi contained slightly bloodstained fluid and the lung substance showed widespread areas of haemorrhage confirmed by microscopy. The appearances were consistent with pulmonary barotrauma.

With the exception of adhesions around the stomach from previous surgery, there was no other significant abnormality.

SUMMARY OF FINDINGS

Evidence of pulmonary barotrauma leading to cerebral arterial gas embolism. Coronary artery atherosclerosis. Old myocardial infarction.

COMMENT

It was subsequently established that diver A had experienced severe angina about ten years before his death, resulting in his having an angioplasty which completely abolished his symptoms. As he was a keen diver he asked his cardiologists at the time if it would be safe for him to continue diving and being assured that it would be so, he dived regularly, usually during the summer vacations until his death. His wife and sons said that he had not attended any cardiologist for follow up from the time of his angioplasty until his death. I assume that because of the state of his coronary arteries and the demonstrated old myocardial infarct, that he may have experienced some cardiac related symptoms leading to his unnoticed ascent.

I would welcome comments from cardiologists as to management of post-angioplasty divers especially as to advice relating to increased risks from scuba diving, and what follow up is desirable for such patients, e.g. should a stress ECG be performed before allowing their return to diving, and at what intervals, taking into account that he had had trouble free diving for ten years.

Case 2

On 26th March 2000 diver B, a Caucasian female aged 58, was diving from a charter boat at the Poor Knights Islands at a site known as Anne's Reef. She and her buddy were approximately 16 m deep when she was seen to stop swimming and began waving her outstretched arms through a clump of seaweed; she then clutched the seaweed in both hands and her dive buddy noticed that there were no bubbles coming from her second stage regulator although there was said to be a small stream of bubbles exiting her mouth past it.

Her buddy surfaced with her after dropping her weight belt. She was retrieved to a nearby boat where she was given oxygen and CPR for approximately 75 minutes until an Emergency Services helicopter arrived and lowered a paramedic who administered adrenaline and atropine. There was no response and after confirming asystole by ECG the paramedic pronounced her to be dead. At the time of rescue and during CPR it was noted that fine creamy-white foam was coming from her mouth indicating probable inhalation of water, with drowning, and/or pulmonary oedema.

EXTERNAL EXAMINATION:

There was no external abnormality.

A CAT scan of head and thorax was performed before the formal post-mortem examination eight hours after her death. This showed small amounts of air in the basilar and anterior cerebral arteries (Figure 3), in the ventricles of the brain (figure 4) and in the left and right ventricles of the heart; as well as portal venous system and the azygous and hemiazygous veins (Figure 5), all indicating that some air embolisation had occurred.

INTERNAL EXAMINATION

The convolutions of the surface of the brain were flattened and the sub-arachnoid vessels were congested, both appearances consistent with anoxia with oedema. There was no free blood in the sub-dural or sub-arachnoid spaces, nor in the lateral ventricles. The sectioned brain showed no significant macroscopic abnormality.

The ribs from the second to the eighth were fractured in the mid clavicular line on each side; most likely this had occurred during CPR as there was little associated bleeding or damage to underlying lung. Both lungs were markedly oedematous, the left weighing 790 g and the right 890 g. The right lung substance showed an area of intra-parenchymal bleeding into the lower lobe. There was no evidence of previous lung disease. The appearances are consistent with aspiration of water with drowning and probable pulmonary barotrauma. In the absence of any macroscopic heart disease, left ventricular failure causing pulmonary oedema can be excluded.

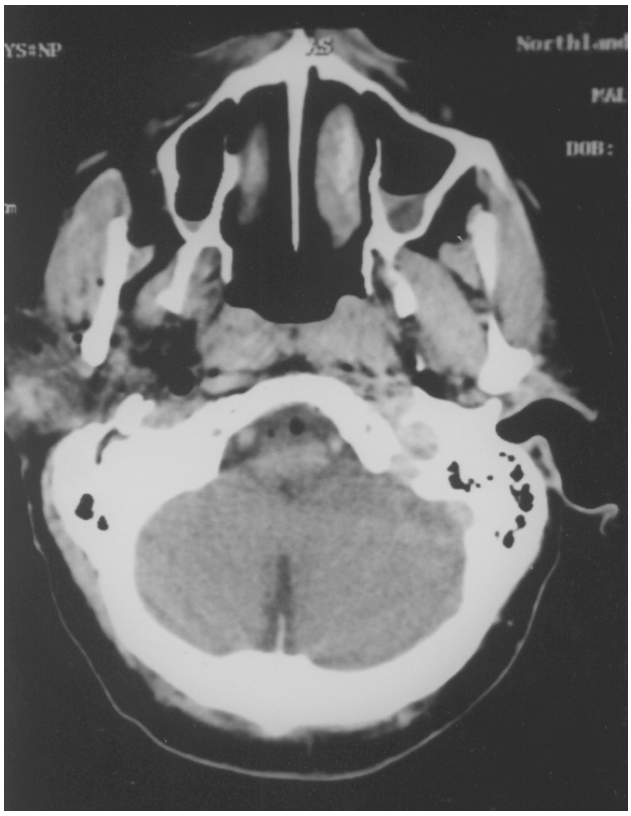

Figure 3. CAT scan through diver B's maxillary sinuses showing air, black dots, in the basilar artery and the anterior cerebral arteries .

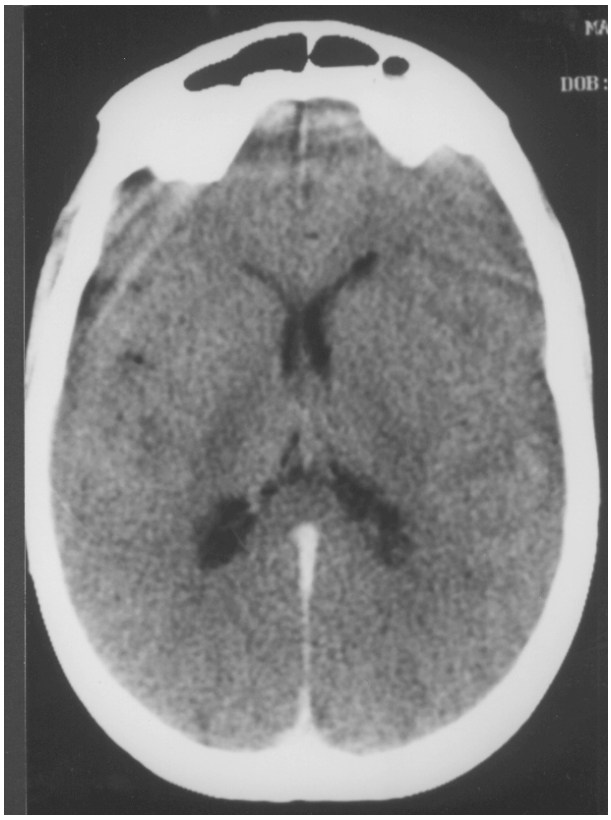

Figure 4. CAT scan through diver B's the upper part of the head showing gas, black areas, in the ventricles of the brain.

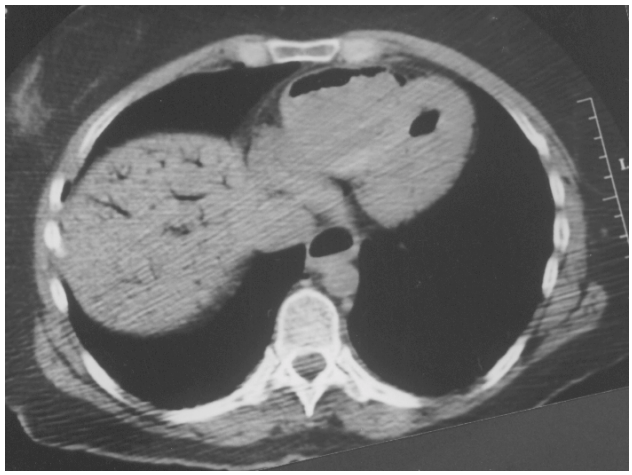

Figure 5. CAT scan through the lower chest of diver B. The left side of the body is on the right of the illustration. The grey area in the right lung is liver with the black streaks gas in the portal venous system. There is gas in both cardiac ventricles and in the azygos and hemi-azygos veins.

The heart was of normal size and appearance, weighing 300 g. All three major coronary arteries were free of atherosclerosis. The valves and chambers were unremarkable. A foramen ovale could not be demonstrated.

There was no other significant abnormality.

SUMMARY OF FINDINGS

Salt water aspiration leading to drowning; complicated by pulmonary barotrauma with intravascular air embolism. Multiple rib fractures from CPR.

COMMENT

The CPR was administered by a Master Dive Instructor and three nurses, one of whom worked in an Emergency Room. The multiple rib fractures due to the CPR are disturbing. Had she recovered she would have been in respiratory distress with a flail chest and in considerable pain.

She was making her ninth dive since completing her Open water Diver certificate. She was a competent swimmer and was said to be a confident diver. She swam laps every day in a large metropolitan public swimming pool with no apparent problems. However her husband recalled that approximately one year before her death she blacked out while doing her laps and was pulled from the pool unconscious. She was taken to the Emergency Department of a nearby hospital by ambulance and after a few hours observation, without formal admission, was allowed to go home. Her husband described her as "very groggy" for a day or two afterwards. Around this time her adult children told a general practitioner friend that she had experienced chest pain with shortness of breath. It appears that she has been well since the blackout incident until her death.

Unfortunately her Emergency Department notes are not available, nor are any details as to her pre-dive course medical examination, if she had one.

I think it likely that she, for whatever reason, experienced a change in consciousness during her final dive and aspirated water even though she apparently did not lose her second stage regulator. I think it likely that air embolisation occurred during ascent while unconscious. Air in the basilar artery would have involved her respiratory centre in the brain stem producing cessation of respiratory muscle activity.

Assuming that the history of blackout one year earlier was provided to her training agency or to her examining doctor, should she have been passed as fit to dive? I think not!

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This is the second paper in the Why Divers Die series by Dr Rees Jones. The first will be found in SPUMS J 1998; 28 (2): 113-117.

PEAK EXPIRATORY FLOW AT INCREASED PRESSURE

Paul S Thomas, Clifford Ng and Michael Bennett

Key Words

Equipment, hyperbaric research, medical conditions and problems.

Abstract

It has been estimated that asthma is as common amongst divers as in the general population. Thus, asthmatic individuals may require hyperbaric therapy and monitoring in this treatment. Asthma is usefully monitored by the Wright's standard and mini-peak flow meters. These devices are versatile and can be used under conditions where electrical supply is difficult or inadvisable. In a hyperbaric chamber electrical sources are restricted due to the risk of fire. We therefore compared the performance of Wright's mini- and standard peak flow meters with a rolling seal spirometer, especially adapted for use in the chamber. The hypothesis tested was whether the peak flow meters, which are density dependent, would over-read compared with the spirometer which is unaffected by changes in density because it is volume dependent.

Seven normal subjects performed volume-dependent spirometry to derive peak expiratory flow (PEF) and PEF manoeuvres using standard and mini-PEF meters at sea level and at 3, 2.5, 2, 1.5 and again at 1 atmosphere (ATA). There was a progressive and significant decline in PEF with increasing pressure as measured by the spirometer (69.5+/-0.8% baseline at 3 ATA), while the PEF meters showed a progressive increase in their readings (107.9+/-1.7% at 3 ATA). Using these data points we were able to derive a correction factor which allows the appropriate PEF values to be calculated from the Wright's PEF meters, if the pressure is known. Thus, the Wright's PEF meters can be used under conditions of hyperbaria, if a suitable correction factor is used.

Introduction

It is well recognised that flow is dependent upon density and the flow of gas from the lungs is no exception. In situations of increased gas density, flow is reduced and becomes increasingly turbulent, which can result in an increased time to exhale and less efficient gas exchange. If there is already a critical narrowing of the airway, then this reduced flow can impede exhalation, so that it is incomplete before there is a need for the next inspiratory cycle to begin. This can lead to gas trapping in the airspaces or alveoli. Asthma is the most common reversible disorder with airway narrowing. Airway obstruction in the older population is usually secondary to smoking and only partially reversible.

In situations where gas density is reduced airway obstruction can be measured under both field and experimental conditions, e.g. mountaineering expeditions and hypobaric chambers.[1] In hyperbaric situations where gas density is increased, e.g. diving, studies must be confined to hyperbaric chambers.

Previous work has indicated that the peak expiratory flow (PEF) and forced expiratory volume in the first second (FEV_1) are increased under conditions of reduced gas density. Therefore, as gas density increases the PEF and FEV_1 will fall. This has been demonstrated previously by breathing mixtures of gases of different density at sea-level and in hyperbaric chambers.[2,3]

The number of indications for hyperbaric oxygen therapy is increasing,[4] and therefore some individuals with airway obstruction may have indications for such treatment. In addition, since an unknown number of asthmatic subjects dive, it is evident that some may well develop problems relating to decompression and may require treatment in a hyperbaric unit. A subject with asthma or airway obstruction may require airway monitoring while in the chamber. To reduce the risk of fire, equipment within a hyperbaric

chamber is preferably non-electrical or low voltage, particularly where supplemental oxygen is being used. A number of incidents, many fatal, have been described where these precautions were not observed.[5]

Most respiratory monitoring equipment requires an electrical supply, with the exception of the peak flow meter. We hypothesised that it would be easy to use a handheld peak expiratory flow meter in the hyperbaric chamber, which could be used to monitor those who require hyperbaric therapy and who also have airway obstruction.

Peak flow meters, however, only measure one point of the expiratory phase, and also are dependent on gas density.[6,7] True peak flow increases as gas density falls, because gas flow under these conditions exhibits less turbulence and becomes more laminar. Previous work has demonstrated that under hypobaric conditions (either in a hypobaric chamber or at altitude), or breathing gas which is less dense than air, then the "true" peak flow as measured by a spirometer will increase.[1,2] The mini-PEF meter, however, demonstrates a fall in PEF under these conditions, as it is dependent upon the density of the ambient gas. Previously we have demonstrated that these values can be corrected to yield the true values by the use of an appropriate equation.[1]

We therefore hypothesised that, under hyperbaric conditions, the mini-PEF meter would over-read, while true PEF would fall. The data derived from the mini-PEF meter should, however, enable a correction factor to be employed to derive true PEF. This presentation is based upon data which has already been published elsewhere.[8]

Methods

We compared the performance of Wright's mini- and standard peak flow meters with a rolling seal spirometer, especially adapted for use in the chamber. The hypothesis tested was whether the peak flow meters, which are density dependent, would over-read compared with the spirometer which is unaffected by changes in density because it is volume dependent, and whether a correction factor could be derived to yield "true" PEF.

Seven normal subjects performed volume-dependent spirometry to derive peak expiratory flow (PEF), and also PEF manoeuvres using standard and mini-PEF meters at sea level (1 atmosphere absolute, 1 ATA) and at 3, 2.5, 2, 1.5 and again at 1 ATA. The chamber was pressurised from sea-level to 3 ATA and then held at 15 minutes at each stage thereafter.

Results

There was a progressive and significant decline in PEF with increasing pressure as measured by the spirometer (69.5+/-0.8% baseline at 3 ATA), while the PEF meters showed a progressive increase in their readings (107.9+/-1.7% at 3 ATA) (Figure 1). Figure 2 indicates the difference between the spirometer flow-volume loops for normal subjects at 1 and 3 ATA. It can be seen that flow is significantly reduced at 3 ATA under these conditions ($p<0.001$ ANOVA).

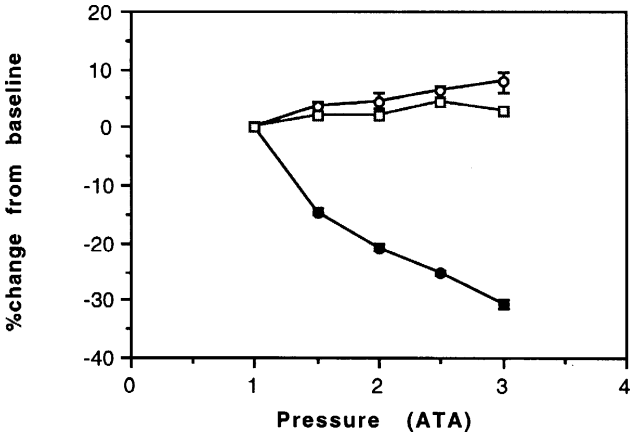

Figure 1. Change in PEF with increasing pressure, as measured with a Wright's standard peak flow meter (open squares), a mini-peak flow meter (open circles) or a spirometer (closed circles). Change in mean PEF is expressed as percentage of sea-level value. (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

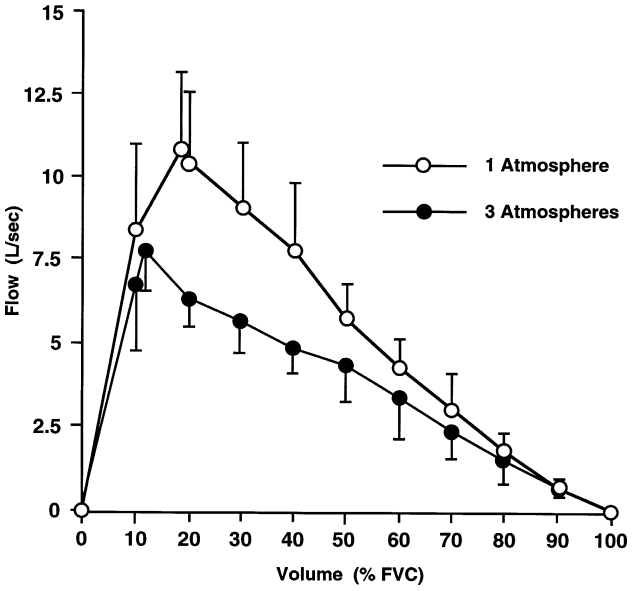

Figure 2. Mean flow volume loops for seven subjects at sea level (open circles) and at 3 ATA (closed circles). (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

Using the these spirometric PEF data points, and the spirometric PEF data from the previous study under hypobaric conditions,[1] plus data from the experiments of Schilder et al.,[2] we were able to demonstrate a relationship which indicates that PEF readings depend mainly on gas density (Figure 3).

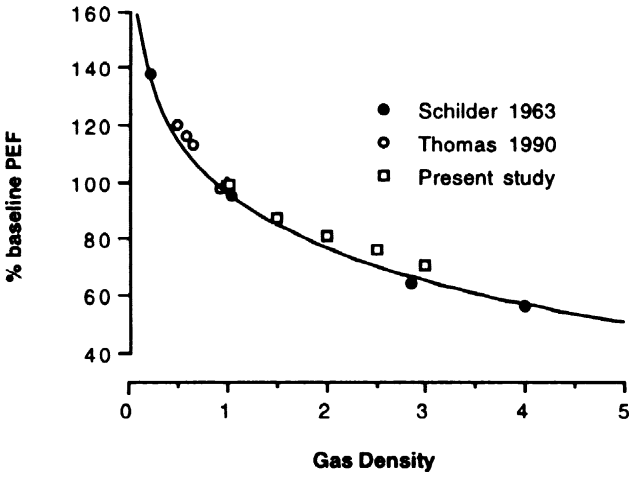

Figure 3. Effect of gas density on peak expiratory flow. The plot was obtained by using data from the present study, (open squares), from Thomas et al.[1] (open circles) and from Schilder at al.[2] (closed circles). (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

From these data, it was possible to derive a correction factor which allows the appropriate PEF values to be calculated from the Wright's PEF meters, if the pressure is known (Figure 4).

The change required in mini-PEF reading to give "true" PEF(%) is
= [77.92 x log(pressure in ATA)]-1.61

Using this correction factor, PEF meters can be relied upon under conditions of hyperbaria.

Conclusion

As predicated, Wright's mini-, and standard PEF meters over-read under conditions of increased pressure. These values can be corrected to the "true" PEF values by the use of a correction factor. It is therefore feasible to use the Wright mini-PEF meter in the hyperbaric chamber, should PEF monitoring be appropriate.

The study has some limitations in that it assumes that dynamic resistance remains unchanged, which may not be the case in smokers who have small airway obstruction. These latter subjects also have lung function abnormalities

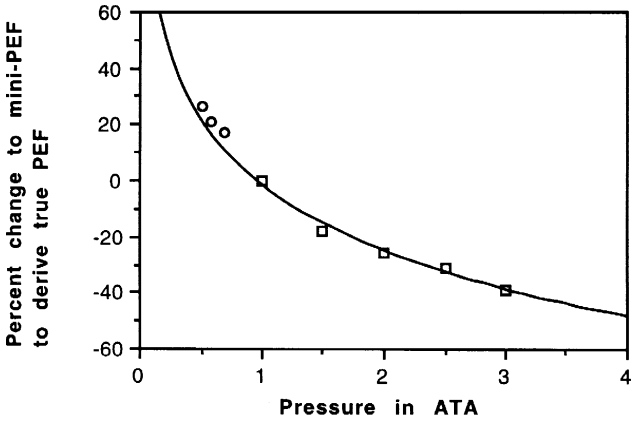

Figure 4. Plot of pressure against percentage change required to derive "true" peak expiratory flow, as measured by the spirometer, from the Wright's mini-PEF meter values. Data were derived from the present study (open squares) and from Thomas et al.[1] (open circles), using identical devices. (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

which are particularly seen in the latter part of expiration, after the point of peak expiratory flow. Therefore, it should be appreciated that PEF does not reflect the entire flow volume assessment of expiratory flow.

Asthma is a condition of reversible airway obstruction, most manifest in the expiratory phase of respiration. It affects up to 10% of the population and, in some studies, this prevalence is the same in the recreational diving community.[9] Most recommendations suggest that those who have poorly-controlled asthma should not dive,[10] and some more stringent guidelines suggest that those with any history or evidence of asthma should not dive. If there is doubt about the history of asthma, the subject may be referred for a bronchial provocation test. These use one of a variety of different substances (e.g. methacholine, histamine, hypertonic saline) in increasing concentration to induce constriction of the airway. Criteria for these tests have been established and define those who are likely to have asthma bronchoconstrict (usually a 20% fall in the forced expiratory volume in the first second (FEV_1)) at or below a certain concentration or dose of the substance inhaled. Those who do not react at this concentration, are considered non-asthmatic. On this basis, potential divers can be advised not to dive. As many asthmatic subjects nonetheless dive, it is likely that some will require monitoring in the hyperbaric unit either for decompression illness or for hyperbaric therapy.

Hyperbaric treatment is indicated for a variety of conditions and, inevitably, the benefits of the treatment will have to be considered in the light of any relative contra-indications, such as airflow obstruction. A simple PEF monitor may help to monitor such patients should such therapy be strongly indicated.

Acknowledgments

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VISION, DARWIN AND THE DEEP BLUE SEA
The visual sense and adaptations for terrestrial and aquatic sight.

Malcolm Le May

Key Words

Physiology.

Introduction

When we learn to dive we are told that the ocean is a "hostile environment" and that we are wholly adapted for life on the land. This is only partly true. Man has shown a liking and a fascination for immersing himself in water which is unlike that of many of our fellow apes. Our closest relative, the chimpanzee, has such an aversion for water that a narrow moat is sufficient to confine captive apes. With the exception of a Japanese relative with a penchant for volcanic springs, other monkeys tend to avoid immersion.

In spite of our seeming adaptation to the land, we continue to carry with us reminders of our life in the sea. Our internal environment is isolated from the hostile dry outside by a space suit of waterproof skin, and when that is damaged, we leak. If we damage enough of our skin, for example by burning, we die. Our internal osmolarity, equal to 0.9% saline, is a reminder of the salinity of the primordial sea. The process of reproduction remains a function conducted in a moist environment and we spend the first nine months of our life immersed. During the process of birth we mimic the change from an ocean dweller to a land mammal, repeating the invasion of the land in microcosm over and over again as each child is born.

In spite of a long history of living on the land, our eyes remain an aquatic based sense. In changing from vertebrate life underwater to life above, gills and swim bladders have become lungs, fins have become limbs, but eyes still remain essentially the organ that evolved in the sea. The continued function of our vision requires an adequate supply of tears and our eyelids ensure that our eyes are kept moist, returning briefly to the aquatic environment twenty times every minute as we blink to maintain the pre-corneal tear film.

We do not see well underwater without the aid of a mask.

Land animals rely on the interface between the air and the cornea for most of their refractive correction and in water man is hyperopic by some +43 Dioptres. Vision evolved in the sea and an interest in how the eye was adapted to different environments led me to a search for the origins of the eye as we know it.

Evolution

It is said that embryology mimics evolution. We begin our gestation as a unicellular creature and build our organs and our systems in the early part of our development, looking like any other embryo in the early stages and passing through stages that resemble our simple vertebrate ancestors. In our early embryonic development we resemble all other *Chordata*, equipped with gills and tails, and developing eyes like all others. Later we develop eyelids, lose our gills and develop other features necessary for life on land.

Charles Darwin wrote, "To suppose that the eye with all its inimitable contrivances for adjusting the focus to different distances, for admitting different amounts of light, and for the correction of spherical and chromatic aberration, could have been formed by natural selection, seems, I freely confess, absurd in the highest degree." This statement has been used by the opponents of evolution to support their beliefs, but Darwin followed this statement with one of the best supportive arguments of his monograph in favour of evolutionary theory. The idea of progressive development of one creature from an earlier predecessor was not new when Darwin published *The Origin of Species* in 1859.[1] The ancient Greek philosophers, Empedocles and Aristotle, hinted of a belief of one species developing from another. In the years preceding Darwin's famous publication, Jean Baptiste de Lamarck, and Darwin's own grandfather, Erasmus Darwin, had written on the same subject. Darwin returned from the voyage of HMS BEAGLE in 1836, and worked on the eventual contents of *The Origin* for some years. He read *Malthus on Population (1798)* in 1838, which reportedly stimulated his own ideas. Publication of *The Origin of Species* was precipitated some twenty-two years after the *Beagle* when the biologist Alfred Russell Wallace foreshadowed publication of his own independent conclusions on "survival of the fittest" in 1858. It is difficult for us now to imagine the revolutionary nature of Darwin's publication which was to become the bestseller of 1859. At a time when the Bible was taken literally by many, the age of the Earth was by them believed to be less than 6,000 years, and even scientists like Lord Kelvin believed the age of the Earth to be only 100 million years, too short for Darwinian evolution.

Life on Earth is some four billion years old.[2] Four billion is a very large number and most of us find it difficult to comprehend. Australians know that Sydney and Perth are some four thousand kilometres apart. There are four billion millimetres in 4,000 km. Continuing this analogy, for someone living in one of these cities, the Cambrian era began 570 km from home, the dinosaurs ruled from 220 to 65 km away and hominids first appeared about four kilometres from the front door. The biblical teaching in Darwin's time put the creation at the year 4,004 BC, a few metres away, rather than the entire width of the country.

For the first three billion years life was unicellular and for the first billion anaerobic organisms dominated. It is estimated that 99% of all species that ever lived have become extinct, either as part of numerous sporadic mass extinctions or as part of a continuing "background extinction".[3] For some three billion years, aerobic stromatolite producing cyanobacteria dominated, eventually declining due to predation by more complex oxygen breathing animals. Stromatolites still exist at sites in Western Australia and in The Bahamas. Animal life as we know it began at the start of the Cambrian period, around 570 million years ago. During an explosion of life in the Cambrian sea nearly all known phyla appeared, including the first chordates.[4]

The early unicellular organisms were equipped with a light sense. Today, light sensitive organelles are present in some unicellular organisms and can include a directional or focussing mechanism or primitive "lens". Multicellular eyes are found in segmented worms, including lenses to concentrate the light and to increase directional accuracy. The compound eye of the *arthropoda* is an alternate design not seen in other phyla. The compound eye offers good directional sense, an ability to perceive the direction of polarised light, and colour vision, but with much lower resolution than the optical based systems. Other invertebrates have a variety of designs of eyes, ranging from a simple pit to the complex eye of the octopus. The eye of the octopus, a *cephalopod*, is structurally similar to the vertebrate eye, with a retina on the rear of a spherical eye and a similar lens, iris, cornea and accommodative mechanism. This in spite of the separate evolution of vertebrates since the Cambrian era and confirms that good designs will appear repeatedly due to convergent evolution. Another *cephalopod*, the nautilus, retains a more rudimentary eye like a pinhole camera, with a 2 mm pupil and the cavity of the eye filled with seawater. The vertebrate eye differs only from the eyes of some invertebrates in its origin.[5]

Vertebrate eyes are derived from neuroectoderm. The invertebrate eye forms wholly from the surface ectodermal layers and, although the vertebrate neuroectoderm also originates from surface ectoderm, the slightly different origin of the vertebrate eye has the advantage of closer integration of vision with the eventual nervous system. An early Cambrian chordate, *Pikaia*, had the basic design of the evolved vertebrate eye, not dissimilar to the eyes of modern *Agnatha* like the lamprey. The fossil records of the *Agnatha* are sparse. The lamprey, like its predecessors, has a sub-epithelial eye which was originally possible due to the small size and transparency of the organism. As size and opacity increased, the eye migrated to the surface, but has remained essentially similar in all vertebrates up to the present day. The lens and cornea form from surface ectoderm, their differentiation stimulated by the presence of the underlying embryonic optic vesicle.

Vision above and below water

The aquatic eye does not use the cornea as a refractive surface. The eyes of fish therefore have a large spherical lens and a relatively flat cornea. Fish are myopic in the unaccommodated state. A constant relationship exists between the radius of the lens and the radius of the eye in all fish and is described as Matthiessen's ratio. A spherical lens resists deformation and the accommodative mechanism found in most fish consists of a displacement of the lens within the eye by a small internal muscle, rather than deformation of the lens itself. In the *Agnatha*, the muscle moves and flattens the cornea as well. Some fish need good vision both in and out of the water and achieve this either with an increased range of accommodation or by means of a dual optical system found in the sloping retina of rays, or the twin pupils of the curious South American fish, *Anableps*. Colour vision is relatively rare in nature, being present in some arthropods (insects and crustaceans), fish, birds and in higher apes, including man. Sharks do not have colour vision, and uniquely amongst fish have a lower eyelid which protects the eye during close quarter encounters. Benthic fish have relatively huge lenses, an adaptation to make use of sparse available light. Only in the extreme depths and in permanent cave dwellers do eyes become rudimentary.

The *Sauropsida*, reptiles and birds, have structurally similar eyes. The lens, although spherical, is equipped with an equatorial annular pad which enables the lens to be squeezed strongly by a powerful ciliary muscle. The eyes of the *Sauropsida* are not spherical and various structural modifications are present, which enable the eye to retain its non-spherical shape in spite of the positive intraocular pressure and accommodative forces. Typically, a ring of bony plates is present anteriorly, forming a firm base for the action of the ciliary muscle. This ring of scleral ossicles can also be seen in the fossils of many Ichthyosaurs (Figure 1) and exists in present day *Chelonians*, the tortoises and turtles. Scleral ossicles are also found in the Coelacanth, *Latimera*. The *Chelonians* are considered more primitive than the *Sauropsida* and have additionally a powerful iris sphincter which compresses the anterior lens, assisting underwater vision. The kingfisher, *Alcedo*, has two maculae, an egg-shaped lens with both a short and a longer diameter and has adopted the strategy of the dual optical system to enable vision both above and below the water. Diving ducks and loons are primarily focussed in air but use the third eyelid, the nictitating membrane, underwater. The nictitating membrane is transparent and contains an area of high refractive index, providing a powerful accessory lens.

Mammals evolved from common ancestors present throughout the long rule of the dinosaurs. It was not until after the great Cretaceous/Tertiary extinction of the dinosaurs that modern species evolved. The history of the mammalian common ancestor means that the mammalian eye has evolved from an ancestor that was not only nocturnal, but had lost colour vision as well. The basic mammalian eye is poorly equipped for accommodation and has a thin sclera.

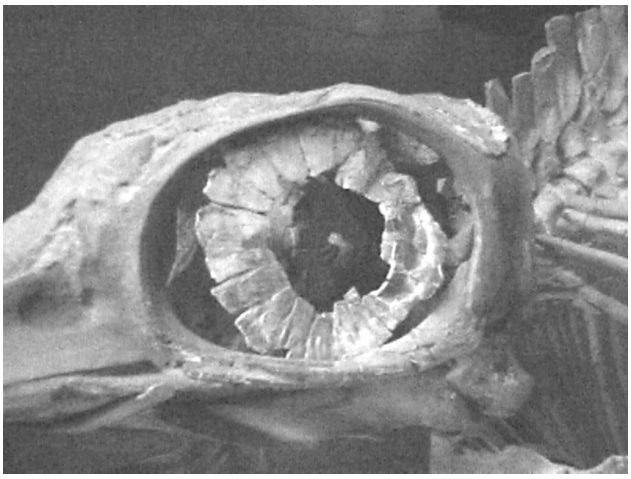

Figure 1. The scleral ossicles preserved in a fossil Icthiosaur. Photo: Malcolm Le May.

Nocturnal mammals have a *Tapetum*, a reflective layer in the upper retina, which increases the light sensitivity in the lower field of vision by reflecting light a second time through the retina. The mammalian eye has subsequently evolved to suit all habitats, including a return to the water by several different groups. The *Cetaceans*, dolphins and whales, have a spherical lens similar to fish. Whales have extremely small eyes relative to their great size and the eyes of the larger whales have an enormously thick sclera posteriorly, a feature shared with large land mammals like the elephant. These eyes are relatively immobile. The otter has a mechanism not unlike the turtle for clear underwater vision. It needs to have good vision both above and below the water and has a deformable lens assisted by a powerful iris sphincter. *Pinnipeds* such as seals are myopic on land and have clear vision underwater. Their terrestrial vision is assisted by a very small vertical slit pupil when on land, producing a pinhole or stenopeic effect, a mechanism also found in the sea-snake.

Conclusion

The same design of eye exists with minor differences in all vertebrates. The early fossil record is incomplete, but the organ appears to have emerged in its present form following the initial evolution of the *Chordata* during the Cambrian era.

The eye evolved for vision in a shallow sea, but has adapted to suit all environments including the land, the air and a return to the sea by several groups.

Some animals require vision both above and below water and a number of different strategies have developed to ensure clarity of sight in media with markedly different refractive indices.

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LONG TERM HEALTH EFFECTS OF DIVING

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Key Words

Diving safety, investigations, medical conditions and problems, treatment sequelae.

Introduction

With the improvement in prevention and treatment of diving accidents over the last few decades, attention has now been focussed on the possible undesirable long-term health effects of diving. Much of the investigative efforts in this field have centred on the professional diver, however, for large numbers of recreational divers these concerns are very real. If we, as medical practitioners, are going to assess our patients' fitness to dive, we must also be able to provide advice as to how diving may affect their health.

Long term effects

A long term effect of diving can be defined as an effect outside the range of normal for an appropriately matched population. This effect must be causally related to diving and must persist beyond the acute and rehabilitation phase of a diving accident. There must be no non-diving pathology to explain the effect and it must produce a reduction in the performance or the quality of life of the diver.

There is no dispute that diving accidents can result in permanent sequelae such as dysbaric osteonecrosis, decompression illness (DCI) and pulmonary barotrauma with cerebral arterial gas embolism. A diver who suffers hypoxia from any cause with resultant brain injury may have permanent damage. Other diving related injuries, such as barotraumas, can result in hearing loss and vestibular damage. Gas toxicities and marine animal injuries may also result in permanent sequelae.

A more difficult question to address is, do long-term health effects occur in divers who have not suffered an overt injury or a specific diving accident?

A variety of physiological and pathological changes have been postulated to produce a great variety of long-term health effects, not related to a specific diving accident. This non-exclusive list includes increased environmental pressure, increased gas partial pressure, oxygen toxicity, gas induced osmosis, asymptomatic bubble development with local tissue effects, blood bubble interaction and blood brain barrier disruption, barotrauma damage to surrounding tissues, asymptomatic lipid emboli and adaptive effects of diving. I will specifically discuss dysbaric osteonecrosis (DON), barotrauma, decompression illness (DCI), ^{99}Tc-HMPAO-SPECT scanning (which is an investigation that has been in and out of favour), ophthalmological effects, ear nose and throat problems, pulmonary effects, subclincal pathological deficits (which from their very nature are hard to detect and quantify), neuropsychology, behavioural factors, and finally mention some miscellaneous findings.

Dysbaric osteonecrosis

Dysbaric osteonecrosis is usually assumed to be the direct or indirect result of gas bubbles that form during decompression interfering with the blood supply to vulnerable areas of bone. Juxta-articular, or A, lesions are found near articular joint surfaces and may eventually result in the collapse of the joint. Medullary, or B, lesions are found away from the articular surface, are usually asymptomatic but occasionally result in the development of sarcomata.

DON is often thought of as a consequence of deep diving, although it was first observed in caisson and tunnel workers. It became a problem in the commercial diving world as exploration went deeper.

DON is rare in recreational and military divers who have not been involved in experimental diving. It has been generally assumed that there was no risk of DON in divers who did not descend below 30 m. However, tunnel workers, who work long shifts under pressure, have had a significant incidence of DON, although they were typically working at pressures less than 12 m.

DON has been known to occur after a single pressure exposure.[1] In 1931 a Royal Navy submarine became disabled in the China Sea and three out of the five survivors who escaped subsequently developed DON. They were exposed to a maximum pressure of 4.8 bar (ATA) for 2-3 hours during their escape, their only exposure to increased pressure. This refutes the perception that DON only occurs in people who have had extensive diving careers. A more realistic view is that DON is the consequence of imperfect decompression procedures.

It is known that conservative diving practices offer some protection. By the mid 1980s the British Decompression Sickness Registry, which was responsible for X-raying occupational divers, was closed because the number of new cases were so few, largely due to government regulation of decompression table usage in the North Sea.

While it is presumed that DON is a long term consequence of inadequate decompression and bubble formation, the exact mechanism by which those bubbles cause injury is unknown. Proposed aexplanations include direct bubble injury to bone, infarction secondary to arterial gas embolism, vascular obstruction secondary to platelet aggregation and activation of inflammatory pathways, raised intra-osseous pressure due to bubble formation within bone and raised intra-osseous pressure as a result of gas osmosis during decompression.[2]

There does appear to be an association between DCI and DON although not all divers with DON have a history of DCI. Neither do all divers who suffer DCI develop DON, nor do all divers with a history of high risk exposures develop DON.

Barotrauma

Barotrauma may produce, albeit infrequently, permanent damage to the facial, infra-orbital, maxillary, cochlear and vestibular nerves.

Decompression illness

There is no doubt that DCI causes residual health sequelae. Spinal DCI may result in paraplegia with urinary and bowel disturbance despite recompression therapy. DCI can result in permanent sensory disturbance with paraesthesia or disturbance along the distribution of peripheral nerves. DCI produces psychometric abnormalities with some studies reporting up to 50% of individuals with residual neuropyschological abnormality after experiencing an episode of DCI.[3]

It has also been suggested that when one is diving sub-clinical and cumulative lesions may develop within the brain and spinal cord. Todnem et al. compared 156 divers with 100 non-diving controls and reported that divers had a higher incidence of specific neurological symptoms and episodes of cerebral dysfunction, in non-diving situations.[4] The latter predominantly involved the distal spinal cord and peripheral nerves. There was an independent correlation between these abnormalities and diving exposure, DCI and age.

Whether or not subclinical and cumulative lesions can occur in divers who have never experienced clinical DCI or develop after complete recovery is uncertain. If they do occur, can they be avoided by conservative diving practices and how important are they clinically?

Central neurological damage may be transient, or may be permanent and cumulative. Electro-encephalographic (EEG) changes after diving accidents or incidents may indicate damage, but the alteration in EEG behaviour is sometimes difficult to interpret. CT and MRI scans, as well as evoked cortical potentials may reveal only gross abnormalities and may miss small areas of multi-focal damage. Newer radiological and scanning techniques have been used in the investigation of divers, yet many of these techniques, in their own right, have yet to be adequately standardised.

^{99}Tc-HMPAO-SPECT scanning

Technetium-99 hexamethylpropyleneamine oxime single photon emission computerised tomography (^{99}Tc-HMPAO-SPECT) is a technique used to image regional blood flow. The ^{99}Tc-HMPOA is injected intravenously and diffuses across the blood-brain barrier and remains bound in the tissues for up to 8 hours so therefore it effectively produces a frozen image of the regional brain blood flow at the time of injection.

In 1989 Adkisson et al. first reported the use of this technique in people who had suffered an episode of DCS.[5] They studied 28 patients within one month of presentation. Twenty-three of those were said to have neurological DCS, 4 had cerebral arterial gas embolism (CAGE) and 1 had a limb bend. They reported cerebral perfusion deficits in all cases of neurological DCS and CAGE and a high degree of correlation between the clinical picture and the site of the perfusion deficit. The possibility of occult neurological damage was raised by the appearance of cerebral perfusion deficits in divers who showed clinical signs only of spinal cord involvement. The patient with the limb bend had a normal scan.

However, subsequent investigators have challenged some of these findings. Hodgson et al. compared 10 divers with acute DCS to 10 divers who had been treated some 3-5 years earlier for DCS, 10 divers who had never had DCS and 10 population controls.[6] Although there was a trend towards a larger number of deficits in the individuals who

had had DCS, there was no statistical difference between the groups and there was no apparent correlation between the sites of the perfusion deficits and the clinical presentation. There was also a higher than predicted number of positive scans in both the divers who had never been treated for decompression sickness and the non-diver controls. This study does not therefore support the assumption that asymptomatic divers sustain neurological injury, as the same deficits were seen in individuals who had never dived. The significance of these deficits and the incidence in control populations is yet to be determined.

Following this, Evans et al. studied 54 patients who presented to the Aberdeen Hyperbaric unit with DCS.[7] Of these 62% were said to have abnormal ^{99}Tc-HMPOA-SPECT scans but there was no correlation between the site of the deficit and the clinical presentation. They then extended the study to include divers who had no history of DCS and non-divers.[8] Again they concluded that although the divers with abnormal scans in the group that had DCS tended towards a greater diving exposure, this was not repeated in the diver group. Therefore they could not conclude that a greater diving exposure increases the likelihood of an abnormal ^{99}Tc-HMPOA-SPECT scan.

Ophthalmological effects

Because the eye develops as an extension of the forebrain it has long been recognised that the fundus may reflect changes within the central nervous system (CNS). Polkinghorne et al. studied 84 divers and 23 non-divers using retinal fluorescein angiography to determine whether blood vessel changes are common in the ocular fundi of divers.[9] It was proposed that these changes might indicate the presence of vascular obstruction elsewhere, particularly in the CNS. Twelve of the divers had previously been diagnosed with DCS (9 neurological) although none had visual symptoms. Three presented with joint pain only. The authors reported that the retinal capillary density at the fovea was low in divers and microaneurysms and small areas of capillary non-perfusion were evident. The divers had significantly more abnormalities of the retinal pigment epithelium than the comparison group of non-divers. They also found a positive correlation between the presence of a fundus abnormality and the length of diving history. It was concluded that all changes were consistent with obstruction of the retinal and choroidal circulation and the obstruction was likely to be due to intravascular bubble formation during decompression or to the altered behaviour of blood constituents and blood vessels under hyperbaric conditions. However, no subject had any recorded visual loss as a consequence of diving and other investigators have not been able to reproduce these findings.

Holden et al. performed angiography on 26 divers, who had used safe diving practices for at least 10 years and 7 controls.[10] No significant difference in the incidence of macular abnormalities was found between these groups. It was suggested that adherence to safe diving practices may protect against the effects reported by Polkinghorne.

Murrison et al. studied a cohort of Royal Navy divers with retinal fluoroscopy and angiograms and no difference was found between the divers and the non-divers and the prevalence of abnormalities did not correlate with diving experience.[11]

Ear nose and throat problems

There is no doubt that hearing loss can occur as a result of diving, whether it is noise induced from exposure to noise in compression chambers or as a result of inner ear barotrauma. Exostoses, which are a long-term effect of cold water exposure, are common in divers, but rarely become symptomatic.

Sinus disease is common in divers. It is often a cumulative result of inappropriate diving activities, especially when divers persevere with descent despite the evidence of barotrauma and infections. Chronic inflammation and gradual scarring results in reduction of the lumen of the sinus ostia or ducts.

Pulmonary effects

It has been proposed that long-term effects damage the lung as a consequence of diving. Some divers tolerate high levels of carbon dioxide without increasing their respiratory rate.[12] It is uncertain as to whether this reflects an inborn physiological abnormality or is an adaptation from diving.

It is generally accepted that divers have larger vital capacities than non-divers, with one study suggesting an initial adaptive increase in lung volumes in divers, followed by a progressive decline, presumably age related.[13] It has also been demonstrated that some divers develop a degree of airflow obstruction due to airway narrowing.

It has been suggested that some of these effects may be related to a cumulative pulmonary oxygen toxicity or to repetitive pulmonary gaseous micro-embolisation.[14]

It has also been shown that there is a slight decrease in pulmonary transfer capacity after deep dives which tends to improve in the subsequent weeks, but which may not be totally reversible.[18]

Proposed mechanisms for these changes include lung over-distension, hyperoxia, bubbles, pressure per se and gas and particulate contaminants.[13]

Subclincal pathological deficits

Palmer et al. reported the autopsy of a male sports diver who had recovered almost completely from an episode of spinal DCS 4 years before his death from an unexpected cause.[15] He was clinically asymptomatic, yet at autopsy was found to have extensive changes in the posterior and lateral columns of his spinal cord.

Palmer, Calder and Hughes examined the spinal cords from 8 professional divers and 3 amateur divers who died accidentally.[16] All but one had had a diving medical examination somewhere between 2 and 38 weeks before their death. None had a history of DCS and none had any documented neurological abnormality at the time of their medical examination. Marchi positive tract degeneration was found in the cords of 3 professional divers, variously affecting the posterior, lateral and to a lesser extent the anterior columns. These changes were difficult to see on haematoxylin and eosin sections, but were clearly shown by the Marchi staining technique. Marchi positive material does not appear in degenerating myelin fibres until 7-10 days after the initiating lesion and does not appear intracellularly until some 10 weeks after the insult.

Palmer, Calder and Yates also studied the brains from 12 amateur and 13 professional divers who died accidentally.[17] Only 3 of the 25 had reported a previous episode of DCS. In 15 of the 22 divers who died from diving accidents there were grossly distended, empty vessels in brain tissue presumably due to post mortem decompression artifact. The most striking change observed was perivascular lacuna formation found in the cerebellar white matter of 8 brains (3 amateur, 5 professional). In addition hyalinisation of vessel walls was observed, along with necrotic foci in the grey matter and perivascular vacuolation of white matter in 7 cases.

The authors propose these changes probably arose from intravascular gas bubble formation producing a sudden distension and occlusion of small arterial vessels. With passage of the bubble the vessel returns to its normal size leaving a surrounding area of degenerated tissue within the lacunae.

Hyalinisation of the vessel wall is also believed to occur as a consequence of this rise in luminal pressure. In one previously asymptomatic professional diver, there was also unilateral necrosis of the head of the caudate nucleus.

This study provides evidence of chronic changes (lacuna formation and hyalinisation of vessel walls) in the brains of asymptomatic divers who did not have a history of DCS when compared with control subjects. This is important as it is now generally accepted that bubbles are produced with all but the most innocuous dive profiles.

Neuropsychology

There have been many anecdotal reports in the literature supporting the hypothesis that diving per se produces brain damage and dementia. Reputable studies to support this hypothesis are lacking. Carl Edmonds studied the Australian abalone divers.[18] These divers had dived on average for greater than 16 years, for greater than 5 hours per day, 105 days per year, to depths greater than 50 feet and had suffered DCS on average 4 times. There was no evidence to support a conclusion of chronic mental impairment or diving dementia in this population.

The US Navy studied 421 divers over a 10-year period and found no evidence, in the absence of a specific injury, to support a correlation between altered neuropsychometric tests or function with diving exposure.[19]

Behavioural factors

In discussing behavioural changes as a consequence of diving it is most important to establish what divers are really like. It may come as a surprise to some but most of the literature suggests that divers differ from the normal population![18] Divers tend to be more adventurous; they take risks and are more physically active. They perform quite differently on psychological testing. They appear to have a love of adventure and have reduced levels of anxiety. Divers have a preponderance of traumatic causes of death. They have higher suicide rates and more motor vehicle accidents.

Therefore it is important to consider whether diving causes them to act that way or are they simply a subgroup of risk-taking, adventure loving people. The verdict remains open on this question.

Miscellaneous

It has been claimed that divers are sub-fertile or have a preponderance of female offspring. There is no evidence to support either of these statements.

There are many haematological and vascular changes that have been reported in animal and experimental models but few persist for any length of time post-ascent or in the human population. An increased incidence of chromosomal aberrations in cultured T lymphocytes in divers has been demonstrated but the health effects related to these changes have not been fully evaluated.[20]

It has been postulated that arthritic disorders have a higher incidence in divers than non-divers and a study of over 11,000 US navy divers showed an increased hospitalisation rate for joint disorders in the 23-28 age group without any obvious explanation.[21]

Conclusions

In 1993 an international consensus conference on the long term health effects of diving summarised current knowledge and produced the following statement:[22]

"There is evidence that changes in bone, the CNS and the lung can be demonstrated in some divers who have not experienced a diving accident or other established environmental hazard.

The changes are in most cases minor and do not influence the diver's quality of life. However, the changes are of a nature that may influence the diver's future health.

The scientific evidence is limited, and future research is required to obtain adequate answers to the questions of long term health effects of diving."

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This paper, and its subsequent presentation, formed the basis of a chapter in the forthcoming text Diving and Subaquatic Medicine, 4th Edition, *to be published by Arnolds later in this year.*

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

TASMANIA'S AQUACULTURE INDUSTRY: A TEN-YEAR REVIEW OF IMPROVED DIVING SAFETY

David Smart, Sean Rubidge, Peter McCartney and Corry Van Den Broek

Key Words

Decompression illness, diving industry, diving operations, occupational diving, risk, safety.

Abstract

Tasmania's marine finfish aquaculture industry has developed from humble beginnings in 1986 to become a leading export earner for the state. Marine aquaculture is diving intensive, and divers have made a significant contribution to product quality. The early years of the industry were hampered by significant levels of diver morbidity due to risky diving activities. This ten-year review outlines the major improvements in safety which have been achieved by broad-based changes to diving training, operations and procedures. The number of divers treated annually for decompression illness has fallen from 5.5 per 2,100 dives in 1988-90 to 0.5 per 8,768 dives in 1996-98. The industry now has a decompression illness incidence of 0.57 cases per 10,000 dives and is in line with world's best practice.

Introduction

Tasmania possesses a rich maritime heritage. Since European settlement, the Tasmanian economy has been reliant on its close links with the sea for trade. The unpolluted waters off its coastline support a substantial wild fishing industry. Tasmanians have always enjoyed access to quality seafood. Until the 1970s, wild fisheries were the only significant source of revenue from fishing in the State. A natural progression of the Tasmanian's close relationship with the sea has been the development of marine aquaculture. After initial success with oyster and mussel farming in the 1970s, Atlantic salmon farming commenced in 1986. More recently, marine farming ventures have explored scallop, abalone and striped trumpeter aquaculture. The aquaculture industry is a major contributor to Tasmania's economy, now producing 35% of total fisheries value of $213.9 million.[1] From small origins, the marine finfish aquaculture industry grew to employ over 500 people directly in 1997, producing exports worth $ 64 million.[1,2]

Figure 1. Diver entering a typical aquaculture pen.

Atlantic salmon farming and ocean trout farming are the main sectors of the aquaculture industry which employ divers. Divers contribute substantially to the quality of these fish, which obtain premium prices on world markets. The fish are farmed in floating pens up to 120 m in circumference, which enclose the fish in a cylindrical net suspended from the surface (Figure 1). Pens vary in size, depending on the type of fish farmed and the size of the farm's operations. An aerial photo of a typical lease is shown in Figure 2. Divers in the marine aquaculture industry undertake many roles including maintenance of mooring lines and farm perimeter nets, supervising the setting of fish pens, and undertaking checking and repairs of individual fish pens with removal of dead fish from the pens. In addition, they perform many "land-based" activities. In 1990, two of the authors provided an overview of the industry and described how salmon were farmed from smolt to the finished product bound for interstate and international markets.[3] The industry is further described elsewhere.[2]

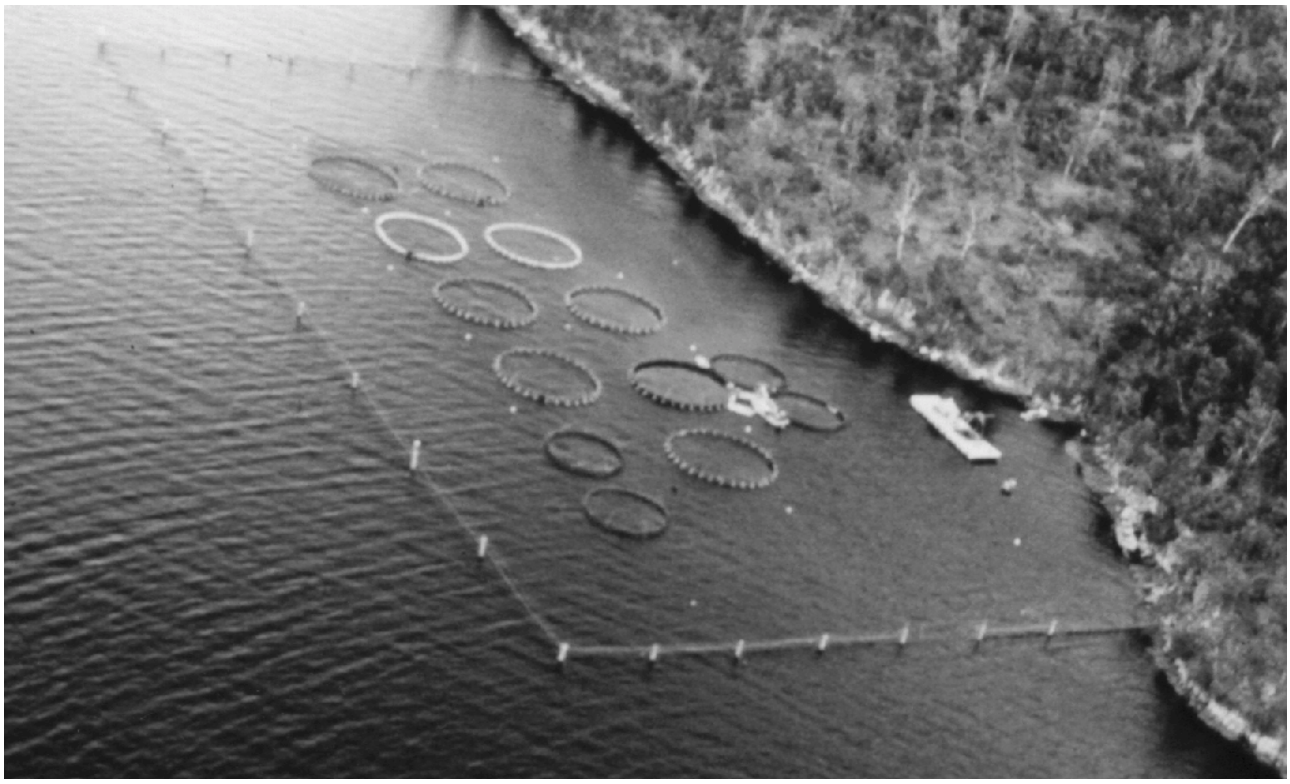

Figure 2. Aerial photo of an aquaculture lease showing perimeter net around circular fish pens.

The 1990 study by Smart and McCartney found significant levels of diver morbidity occurring in Tasmania's fledgling aquaculture industry, due to high risk diving practices (Table 1).[3] Land-based activities also had some impact on levels of post-diving morbidity.

In 1990, episodes of decompression illness (DCI) from the aquaculture industry were unacceptably high. Despite employing only 6.2% of the professional divers in Tasmania, the industry produced 47.8% of the treated cases of DCI at the State's hyperbaric facility at the Royal Hobart Hospital.[4] In 1990, limited recording of diving activities created uncertainty about the number of dives undertaken in the industry each year. An estimate of 2,100 dives per year, based on verbal reports of diving activity occurring at the time, was available to calculate the incidence of DCI.

A major concern identified by the 1990 study was that very few of the divers had training specific to the industry, and 44% of the injured divers had no diving experience, training or qualifications prior to commencing work as divers.[3] This had two potentially serious effects: not only were the diving practices high risk, but the individuals employed as divers lacked the skills and knowledge to recognise safety hazards as they undertook their daily work. The type of diving required by the industry created a further difficulty. Most divers were undertaking multiple descents and ascents in and out of the shallow fish pens (termed "bounce diving" or "yo-yo diving"), and there were no known diving tables to cover this type of diving.

Bounce diving was considered to be higher risk for DCI than standard "square profile" diving, involving a single descent then ascent.[5] This higher level of risk was supported by cases of DCI occurring in aquaculture divers who had undertaken all of their dives in less than 9 m.[5] During 1990 and 1991, there was a strong push from the Tasmanian industry, union representatives and diving medicine specialists to improve diving operations within the industry and to protect and maintain the health of its divers. Cooperation between industry participants resulted in significant changes to many areas including diver training, operations, equipment and emergency procedures.

Aim

The aim of this paper was to review changes in the diving operations of the aquaculture industry, over a ten-year period from 1/7/1988 to 30/6/1998. We also aimed to assess the safety outcomes of changes to diving practices.

Methods

A longitudinal review of ten years of diving activity within the aquaculture industry was conducted, assessing a number of factors:

1 diver participation;
2 evolution of diving practices;
3 major episodes of morbidity due to DCI.

This review used information collected from the field, surveys of participants in the diving operations, and the treatment database of the Hyperbaric Medicine Unit at the Royal Hobart Hospital (RHH). We stratified the study into five periods, each of two years. The ten-year period was chosen because it spanned the time during which most of the diving activity has occurred in the industry. It also covered the period during which substantial changes in training and work practices were made from within the industry. The selected ten-year study period overlapped with the original work of Smart and McCartney,[3] with the exclusion of the months of April to June 1988.

We selected the number of decompression incidents requiring treatment at the RHH as our principal outcome measurement. Since the RHH receives all decompression emergencies for the State of Tasmania, this allowed us to capture all *treated* cases of DCI. Information relating to decompression episodes was readily accessed from the Hyperbaric Medicine database; divers who presented symptoms after diving but were not referred for treatment at the hyperbaric facility were excluded. Recording of these incidents was unreliable at the aquaculture farms in the years 1988-91. We did not include minor diving incidents such as ear and sinus barotrauma or other non-diving trauma as outcome measures.

Data were sourced by the following methods:

1 A telephone and/or written survey of all finfish aquaculture industry participants was undertaken. A structured questionnaire was administered, covering the number of divers employed, the use of log books, the number of dives from logged data, the depths of the dives, the type of diving and its frequency, limits placed upon bounce diving and specific tables used, diving equipment used, safety procedures, diver training and measures to reduce reliance on diving. Data were collected, tabulated and added to data from a previous survey in 1994.

2 Data on diving activity from the 1988-90 period had been sourced during the preparation of the 1990 paper by Smart and McCartney.[3] Telephone surveys or visits to industry participants had been used to obtain diver numbers. Because of direct contact made with industry members by two of the authors, the number of divers for the 1988-90 period was considered reliable. However, due to a lack of systematised recording, only estimates could be provided of the diving activity occurring in the industry, and we recognised from the outset that the initial data on the number of dives were unreliable.

3 Field surveys were undertaken to allow direct inspection and assessment of diving practices and procedures, and of equipment.

4 Information from the industry and also from the Tasmanian Department of Primary Industry and Fisheries was used to calculate the total number of divers in the State and the productivity of the industry.

TABLE 1

HIGH RISK DIVE PRACTICES AMONG TASMANIAN MARINE FISH FARM DIVERS IN 1990*

1 Diver training
- (a) inadequate or inappropriate;
- (b) almost 50% with no training;
- (c) lack of specific training for the industry.

2 Equipment
- (a) hookah used at excessive depths (>30 m);
- (b) no safety reserve cylinders;
- (c) inadequate thermal protection (5 mm thick wetsuits in 10 °C water);
- (d) limited use of specialised underwater tools.

3 Equipment Maintenance
- (a) no maintenance schedules;
- (b) tampering by untrained personnel;
- (c) incorrect oil and filters in compressors;
- (d) salt water in regulators;
- (e) no air purity standard maintained.

4 Safety procedures
- (a) no protocols for emergencies;
- (b) no backup diver;
- (c) no training for accidents;
- (d) no oxygen or first aid equipment or protocols;
- (e) no diver to surface communication.

5 Dive schedules and profiles
- (a) no logs of time/depth;
- (b) no depth gauges;
- (c) minimal consultation with tables;
- (d) multiple (20-50) bounce dives at depths up to 8 m;
- (e) deepest dives performed last;
- (f) missed safety stops.

6 Other practices
- (a) “Vaulting” the salmon pen;
- (b) Heavy physical work performed after the dive;
- (c) Diving with respiratory tract infections.

* Identified by Smart & McCartney (1990).[3]

5 During the survey process, the authors attempted to identify other issues relevant to diver safety in the industry.

Figures were then tabulated over a ten-year period to allow comparison of the data trends over the period of the study.

Because of the difficulties in obtaining accurate statistics of the total number of dives undertaken in the

industry in its early years, a number of criteria were used to assess diver safety over this ten-year period.

A numerator was chosen: the number of divers with proven decompression illness requiring treatment at the Royal Hobart Hospital.

Denominators were also chosen as follows:
1 number of divers employed in the industry;
2 number of dives undertaken.

During the survey, it became necessary to define what was meant by a "dive" in order to maintain consistency between reports from each of the industry participants. For the purposes of this study, a dive was defined as "the period of time during which the diver was undertaking continuous diving activity with surface intervals of less than 15 minutes". Hence, where a diver entered and exited through multiple pens during the course of work, it would be counted as a single dive (multiple bounces) if the surface intervals between pens were less than 15 minutes.

Data were tabulated onto spreadsheets and statistical analysis was undertaken using Graphpad™ Prism software (San Diego, California, USA). Chi-squared analysis was undertaken for rates and proportions, with p values of <0.05 being regarded as significant. Confidence intervals at the 95% level were provided, where relative risks and odds ratios were calculated.

TABLE 2

EXAMPLES OF CURRENT PRACTICE IN TASMANIA'S AQUACULTURE INDUSTRY

Limits on bounce diving and bottom times

	Depth (m)	Bottom time	Maximum bounces
Example 1	10		8
	>10		6
Example 2*	6	150	8
	12	120	8
	15	60	4
	18	40	2
	21	30	1
	30	15	1
Example 3	9-12	150	8
	12-15	75	6
	15-18	50	4
Example 4†	0-6	240	10
	7-9	180	8
	10-12	I10	8
	13-15	75	4
	16-18	50	4
	19-21	35	2

* Maximum depth.
† Safety advice provided to divers in this dive log:
1 Do the deepest dive first;
2 Always use maximum depth when finding no-decompression times;
3 Add 10 minutes to bottom time for every bounce;
4 Ascend slowly (hand over hand last 3 m);
5 After diving >10 m or bounce diving, take a safety stop at 3 m for 5 minutes;
6 Reduce dive times when working hard underwater.

Results

Study information was obtained from all seven marine fish farm operations in Tasmania. Two of the farms had most or all of their diving operations subcontracted and, in these cases, further surveys were undertaken to ascertain the diving practices of the contractors and obtain a complete picture of the diving being undertaken in the industry. It was of particular interest that the fundamental purpose of diving in the aquaculture industry had not changed over the decade of the study. Divers still undertook the same tasks (outlined in the introduction), which were originally described in 1990.[3] However, there had been significant changes to the way in which diving was undertaken since the original study. The results of the present study have been grouped under the same broad headings as outlined in table 1.

DIVER TRAINING

At the commencement of the 1990s, all marine farms required their divers to be trained to a minimum level of open water recreational certification. This was further upgraded, in 1995-96, to Australian Standard 2815.2 (Restricted). Training for this was significantly more advanced than for recreational diving and equipped aquaculture industry divers with the skills necessary for diving on air to 30 m. It covered dive theory and physics, equipment maintenance, surface-supply diving, full-face masks and communications, search techniques, gas handling and testing, safety training, rescue and emergency procedures, oxygen-provider training, legislation and standards, and detailed coverage of the risks of bounce diving. In addition to training dives, a significant amount of practical work was undertaken at the marine farms; a specified duration of diving experience was included. At the time of this survey (September-December 1998), the industry goal of higher level training had almost been achieved. All farms except one (three divers) were working towards AS 2815.2(R) certification for all of their divers. Five farms already had their divers trained to this standard (52 divers); one had ten divers trained and three divers with open water certification. All of the contract divers (13) were

certified to a minimum of AS 2815.2(R). Hence, with 75/81 divers operating in the industry trained in accordance with AS 2815.2(R), there had been a significant move towards appropriate diver training. This compares with the 1990 study, where only two of nine divers treated for decompression illness had training appropriate to the industry.[3]

EQUIPMENT AND EQUIPMENT MAINTENANCE

Two out of seven marine farms used "Hookah" (surface lowpressure pump) apparatus to supply air to their divers, three used high-pressure ("pods" of four cylinders) surface-supply breathing apparatus, and two farms used both (contractors using both types of apparatus). All farms had maintenance schedules for their equipment. An advantage noted for the high-pressure cylinder surface-supply "pods" was that the air purity could be independently tested, and the filling stations were subject to Australian Standard 2299 air purity requirements. This eliminated some of the vagaries of potential malfunctions of the Hookah apparatus and their adverse effects on diver.[3] Divers had their own personal second-stage breathing equipment (masks and regulators) at three of the marine farm operations and were responsible for its maintenance; most used safety reserve cylinders. Two of the larger operations used diver-tosurface communications, as did the contractors, and four farms used scuba as a backup to their surface-supply apparatus. All operations undertook regular maintenance of their equipment in accordance with preset schedules; cylinder pods and surface air pumps received professional maintenance and repair. Dry suits were used by many of the divers for thermal protection during colder months. Ladders were used by five farms and all contractors for divers to exit the fish pens (Figure 1). This prevented the hazardous activity of "vaulting the pen", which was described in the 1990 paper.[3]

SAFETY PROCEDURES, DIVER SCHEDULES AND PROFILES

At the time of survey in 1998, all of the marine farms had protocols for emergencies and carried resuscitation equipment and oxygen for use in emergencies. Individuals were appointed as safety officers and trained in first aid. Backup divers were available at four operations and were used by the contractors.

All marine farm operators and contract firms kept logs of all dives undertaken by their divers. These logs documented entry and exit times and depths for the divers, as well as documenting pre- and post-dive checks, air consumption and other dive notes. Comparisons with recognised tables also took place, in order to assign repetitive groups to the diver for residual nitrogen calculations. All farms had bounce diving limits and maximum allowable bottom times for the various depths. Table 2 summarises examples of bottom time limits provided to the authors during the survey. There were significant variations in the limits recommended by each farm. This was, however, an improvement compared with the 1988-90 period, when there was no correlation of dive times with accepted tables.

TABLE 3

NUMBER OF DIVERS AND ANNUAL NUMBER OF DIVES IN TASMANIA'S AQUACULTURE INDUSTRY

Marine farm identifier	Number of divers	Number of dives
A	6	150
B	9	150
C	13	468
D	0*	0
E	3	100
F	3	100
G	34	5,950
Contract pooled	13	1,850
Totals	**81**	**8,768**

* Contract only.

Divers were advised to undertake safety decompression stops during ascents from dives >10 m depth and to rest immediately post-diving. Recommended ascent rates at the end of each "bounce" were 5 m/min. Divers were also advised to ascend "hand over hand" in the last 3 m (aiming for 3 m/min) after their safety stop. This method has been used by the pearling industry to undertake slow ascents. Three farms included safety advice in the dive log (Table 2, see example 4). With better documentation, a more comprehensive industry picture was constructed, compared with the 1990 study. The total number of dives performed annually for the period 1997-98 and the number of divers is shown in table 3. Available data allowed a calculation of the average number of ascents for each "dive". The average from all farms was 3.8 (range 3-7.3). Two farms employed contract divers to undertake most or all of their diving. None of the marine farm divers were proceeding deeper than 30 m; instead relying on contract firms to undertake this work. Contract divers undertaking deeper diving all had certification to higher level than AS 2815.2.

OTHER PRACTICES

Creative thinking from within the industry has led to a number of other improvements in diver safety. In order to reduce bounce diving and improve productivity, larger deeper pens were introduced in the early to mid 1990s. This

permitted more fish to be held in individual pens (15,000 versus 5,000 previously), with reductions in maintenance and other procedures created by the economy of scale. For the divers, this meant deeper diving but less ascents and descents in their daily work, and overall less diving. In the early phase of development of the industry, a significant proportion of diving was undertaken to remove dead or diseased fish each day from the pens. The development of "mort cones" reduced the need to dive for these fish on some leases. The mort cone was a wide conical net which was lowered into the deepest part of the centre of the pen, to catch fish as they died and sank to the bottom. Perimeter nets have been traditionally used to keep seals out of the aquaculture leases. These required maintenance and were frequently in very deep water (greater than 40 m) adding to the risk for divers undertaking maintenance. Changes in practices reduced the need for these perimeter nets in some leases. Seals were trapped using baits, then transported many kilometres away from the lease. Stronger, tensioned nets, set from the surface, and "double nets" have reduced the ability of seals to injure fish and reduced seal-bite holes in the net, which previously allowed fish to escape. The ability to set from the surface has also reduced the number of dives required. Some farms were also investigating the use of video to assist inspection processes.

SAFETY OUTCOMES MEASURED BY KEY PERFORMANCE CRITERIA

Figure 3 demonstrates the number of cases of DCI treated at the Royal Hobart Hospital in two-year periods over the ten years, 1988 to 1998. There has been an increase in the number of cases treated, particularly over the last four years. Conversely, there has been a progressive reduction in numbers of aquaculture divers with DCI, which was statistically significant using the Chi squared test for trend, $P < 0.0001$. Table 4 shows the number of divers employed in the aquaculture industry for three of the two-year periods 1988-90, 1992-94 and 1996-98, and the number of cases of DCI treated at Royal Hobart Hospital. The table also documents the annual number of dives undertaken by the divers, although the 1988-90 figure is an estimate because no formal records were kept. Data were not available on diver activity for the years 1990-92 and 1994-96. Using available data, it was possible to calculate incidence of DCI per 100 divers per year and per 10,000 dives. When comparing the first two years of the survey, to the last two years, relative risk reductions for decompression illness have been significant across both parameters:

DCI per 100 divers

relative risk reduction = 16.15

(95% CI = 2.11 to 123.4)

DCI per 10,000 dives

relative risk reduction = 45.81

(95% CI = 5.91 to 354.9)

Hence divers were less likely to suffer major morbidity in the form of decompression illness in 1996-98, compared with 1988-90.

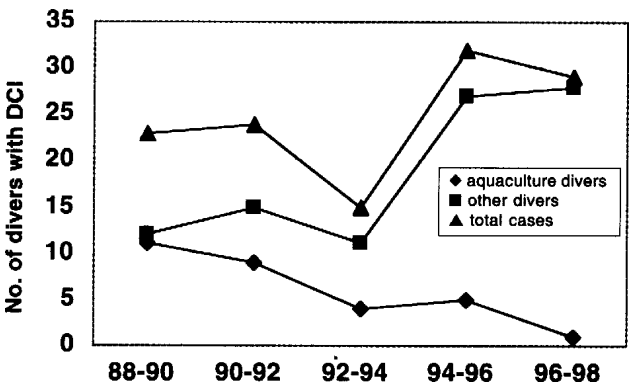

Figure 3. Cases of DCI treated at the Royal Hobart Hospital Hyperbaric Unit, 1/7/1988 to 30/6/1998. Test for trend, $c^2 = 17.34$, $p < 0.0001$.

DISCUSSION

There are parallels to be drawn between the early development of the Tasmanian aquaculture industry and the South Australian tuna farming industry. A report by Kluger and co-workers in 1994 outlined similar roles for divers in the tuna farming industry and similar challenges relating to level of training, multi-ascent dives, lack of tables and high levels of diver morbidity.[6] Kluger's paper did not provide data on the incidence of DCI from the tuna industry; however, the problems faced by the South Australian industry were identical to those faced by the Tasmanian aquaculture industry at the end of the 1980s.

Information obtained in this survey of Tasmanian marine farms has demonstrated significant improvements in the industry's diving operations, when compared with the 1990 study by Smart and McCartney.[3] The changes have addressed many problem areas, and it is pleasing to note that the process has been initiated and driven from within the industry.

Prior to the 1990s, training of divers in the aquaculture industry was ad hoc and, in some cases, non-existent. During the early 1990s, all divers were trained to a minimum level of open water certificate. This has been followed by further training by most operations in accordance with AS 2815.2(R). Compressed-air diving is unforgiving of anyone ignoring established practices and safety procedures. By equipping divers with appropriate knowledge and skills to perform their activities, the industry has invested in a safe working environment and improved productivity. With appropriate training, divers have been empowered to dive safely within established guidelines and limits, also to recognise hazardous procedures and correct them. Diving activity is now carefully recorded, depth, time and bounce limits observed, and safety measures such as decompression stops implemented. Ascent rates (3-5 m/

TABLE 4

CASES OF DECOMPRESSION ILLNESS* FROM THE AQUACULTURE INDUSTRY

2 year period	No. of divers	No. of dives/year	2 year cases DCI†	DCI rate per 100 divers/year	DCI rate per 10,000 dives
1988/90	50	2,100	11	11	26.19
1992/94	86	5,600	4	2.32	3.57
1996/98	81	8,768	1	0.62	0.57
Chi squared value (2 DF)				$\chi^2 = 18.03$	$\chi^2 = 46.26$
P value				P = 0.0001	P <0.0001

* Treated at the Royal Hobart Hospital. †For three separate periods where diving activity was known.

minute) are slower than conservative recommendations of dive tables (6 m/minute in the last 6 m, summarised by Wong).[7] Equipment is now appropriate for the tasks and maintained in accordance with established schedules. The industry has accepted that safety procedures and protocols for emergencies are an essential component of its operations. Creative thinking has reduced the amount of diving in certain activities (for example setting nets and mort diving). Use of ladders has prevented the "Polaris missile" ascent required by the diver to "vault the fish pens".

As a result of many improvements in the diving procedures of Tasmania's aquaculture industry, a significant improvement in diver safety has been demonstrated over the decade 1988-98. The incidence of decompression illness in 1998 was 0.57 per 10,000 dives (0.0057%). The reduction in risk to divers has been achieved with a background of steadily rising fish production (Figure 4). The risk of DCI in the aquaculture industry now compares favourably with Pearling Industry reports of 0.015%,[7] and reports from the Scottish fish farm industry of 0.03%.[8]

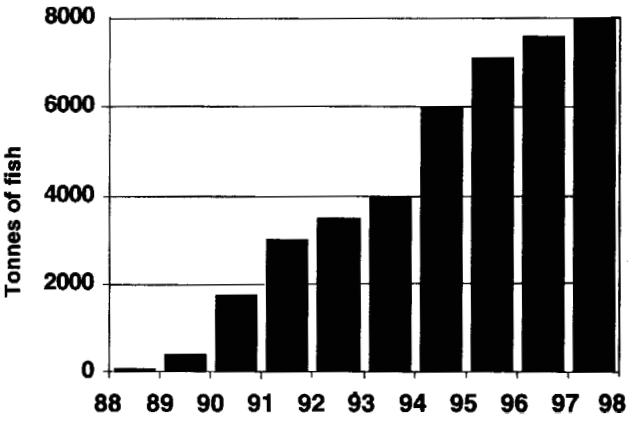

Figure 4. Tonnage of Atlantic salmon produced 1988-98. (Source: Tasmanian Salmonid Growers Association 1997; 1997 figures from ABARE 1998.)

When combined with slow ascent rates, limits on bounce diving for the Tasmanian aquaculture industry may have contributed to improvements in safety. The recommended limits on the number of ascents during bounce diving for Tasmanian aquaculture divers were empirically derived, because of lack of data in this area. The average number of ascents undertaken on all farms was 3.8 (range 3-7.3). This figure was lower than expected, but probably reflects recent moves to larger, deeper pens by many of the industry participants. It is known that divers undertaking similar work in Scotland had an acceptable incidence of DCI when the number of ascents was limited to ten.[9] Using the US Navy probabilistic decompression model, Parker and co-workers demonstrated increased risk of DCI for yo-yo diving when greater than ten descents were made, and a progressive increase in risk with deeper dives.[10]

Further research is required, undertaking carefully controlled experimental dives in sufficient numbers, before Tasmania's aquaculture industry dive schedules can be fully validated. This work should also include Doppler studies using similar methodology to the pearling industry.[11]

Bottom time and depth limits may have also contributed to the reduction in risk of DCI. The last three examples had bottom time limits which were consistent with, or more conservative than the DCIEM (1983 model) table limits.[12] These tables, developed in Canada by the Defence and Civil Institute of Environmental Medicine, are more conservative than the US Navy diving tables and have been rigorously tested during working dives in cold water.[13] Given the added (unquantifiable) risk of bounce diving, conservative limits on diver bottom time are essential. It may be possible in the future for the industry to agree on a standard set of bounce diving tables, after further more rigorous validation of existing procedures.

Dive logs also provided general advice on reducing risk of DCI. Some of the advice covered factors identified as risky practices from the 1990 study (table 1).[3] In 1990, Smart and McCartney described aquaculture diving activity

to depths in excess of 40 m.[3] Deeper dives have been identified as an independent risk factor for DCI.[14,15] Since 1992, marine farm diving has been limited to depths less than 30 m (in most cases less than 21 m). Depth restrictions may also have contributed to reduced risk of decompression illness.

Improvements in diver safety have occurred with a background of increasing output from 380 tonnes of fish in 1989-90 to 8,000 tonnes in 1997-98. The risk to divers of injury in 1990 was 45.8 times the risk in 1998. If the initial rates of diver injury had persisted, in 1996-98 the additional treatment costs for the industry would have been $551,000 (based on the average hyperbaric treatment cost of $3,000). This figure does not include costs of lost productivity or rehabilitation expenses. These cost savings would have significant positive impact for the industry as a whole, independent of the long-term health benefits to the divers.

CONCLUSIONS

This ten-year review of Tasmania's marine finfish aquaculture diving operations has demonstrated significant improvements in diver safety, on a background of rising output from the industry. A cooperative approach, driven from within the industry, has made substantial improvements in many areas, including training, equipment and equipment maintenance, diving procedures, dive schedules and profiles, and emergency procedures. Multifactorial improvements have led to significant reductions in the number of divers treated for decompression illness from the industry. Based on the improvements in diver safety demonstrated in this paper, marine farming operations in Australia and other countries may be able to profit from the experience and achievements of Tasmania's aquaculture industry.

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CAN ANYBODY SEE ME?

Alister Wallbank

Key Words

Diving operations, rescue, research.

A major report on surface marker aids for divers has been carried out for the Health & Safety Executive by Heriot-Watt University. Alister Wallbank explains how his team came up with its findings - and why yellow is the colour if you want to be conspicuous at sea.

What divers use now

A delayed Surface Marker Buoy (SMB) is far the most common location device used by recreational divers, with the most popular colour red or orange, largely because most SMBs are made in those colours. Divers prefer self-inflating or self-sealing SMBs as they do not expel air if allowed to fall over.

This was confirmed when we surveyed more than 80 UK sub-aqua clubs. The response was a little disappointing but a general picture was revealed.

Flags, torches and whistles appeared frequently, with almost half the divers questioned using or having used folding flags. Red and orange were again the most common colours.

More than half of respondents carried two or more torches, with a small unit frequently used as a backup to a larger, lantern style main torch. A similar proportion of divers regularly carried a strobe, usually attached to their buoyancy compensator (BC). Whistles used were generally of the moulded plastic, mouth variety and incorporated as a fixture on BCs. A few divers used air-powered sirens attached to their buoyancy devices. An equal number considered whistles to be next to useless in many circumstances.

When asked which colour stood out best, divers reckoned it was orange. Five percent said that they were colour blind.

The problem

Almost 50% of divers have gone "missing" in the water at some time or other, for an average of 30 minutes. That, at least, was the result of a survey carried out with diving club members.[1] That is worrying, but carrying a surface marker aid can reduce the risk of your cover boat missing you, and increase the chances of you being rescued if you do get lost.

The investigation

The Health and Safety Executive was concerned about incidents involving missing or lost divers on commercial diving projects and contracted Heriot-Watt University to undertake a study. We wanted to find out which location aids would get divers noticed on the surface, whether any device was significantly better than others and which shape, size and colour was most readily sighted by observers on a cover boat.

The range of primary devices tested included telescopic flags, surface marker buoys, torches, strobes, pyrotechnics, whistles and fluorescent dye. Some have been trialled by manufacturers and groups of divers have conducted independent assessments.[2] Much of this work has provided qualitative information, but there has been a need for more extensive and quantitative evaluations, particularly under different conditions.

What we studied

We looked at yellow, red, orange and black folding flags from Bowstone (£15) and an A-Flag; Polyform buoys (£5), Bowstone self-inflating decompression bags (£120), AP Valves (£30) and Bowstone (£15) delayed surface marker buoys; Underwater Kinetics MiniQ, SL4, UK 400, UK 1200 torches (£15-£150); Seeman Sub Signal Flash (£22) and Jotrun AQ-4 (£70) strobes: Pains-Wessex Miniflare 3 pyrotechnic rockets (£27) and Day/Night Distress Signal flares and smoke (£32): and Sea-Streak (£l5) and Presto Dyechem (£14) marker dyes.

We also tried out a PLB7 EPIRB (emergency position indicating recovery beacon) a model since upgraded by Sea Marshall Rescue Systems. We consider an EPIRB to be a secondary emergency location device to be used as a last resort. Our study focused mainly on devices that would help spot a diver as soon as he or she resurfaced. Our findings were not included in the original publication in Diver for legal reasons.

Searches were carried out from inflatables and hard boats, as well as Royal National Lifeboat Institution (RNLI) lifeboats and a search and rescue helicopter, in Scapa Flow between January and March. We certainly got the range of weather we needed. With one session carried out in winds gusting to 45 mph (72 kph).

To find the maximum distance at which the various devices could be located, we secured them to weighted shotlines and recorded their position with a GPS fix. When an observer on the search vessel sighted it another fix was taken to calculate the relocation distance.

A variety of strategies was used to simulate real search conditions and ensure, as far as possible, that direct comparisons could be made under comparable conditions. At or close to sea level, devices were tested under sea states from calm to marginal and a broad range of light intensities.

As these conditions deteriorated, we found location distances of devices at sea level that did not provide an artificial light source decreased correspondingly. The aspect of a device above sea level and its colour also had a bearing, and the observer's eye height had a pronounced effect.

A fully kitted diver without any additional location aid was located at between 250 m and 700 m under ideal conditions. The upper distance was recorded with the diver raising his arm. We also measured the distance at which the diver could see the boat by deploying a diver on a shotline as the boat moved away.

In daylight with a wave height of 0.5-0.75 m, the diver lost sight of the inflatable at 1,300 m and the hard boat at 4,400 m. So a diver without a location aid can see a recovery vessel some time before it is close enough to locate him or her.

Flags

The folding flags were by far the most reliable and, at about £15, cost-effective location device we tested, particularly the day-glo yellow pennant, which was consistently spotted at more than 2 km and up to 3 km. Yellow was the most conspicuous colour in all sea states, even with breaking wave crests, and could be located in deteriorating light when it was impossible to locate pennants of any other colour.

Red and orange flags were located at up to 1,600 m. Two of our observers suffered from degrees of red/green colour blindness and had difficulty spotting these colours, particularly in intermediate light conditions. The A-flag performed least well in all conditions. Not surprisingly, flags were most easily located when the search heading was abeam to the wind direction, so that the pennant presented the greatest visible surface area.

Surface marker buoys

Location distances of the SMBs were similar, up to 1,200 m. The Bowstone Self-Inflating Decompression Bag had a slight advantage, in that it came in day-gIo orange/red and was brighter than the AP Valves and red Bowstone decompression sausages. It was difficult to make a positive identification until the observer got much closer than the initial sighting, because at a distance this device resembled a single red creel buoy.

Several observers found that the AP Valves sausage was easier to locate than the red Bowstone version, being slightly wider and taller. Most found the Bowstone sausage, which was a dull yellow, difficult to locate, but the location distances were not significantly lower than the other SMBs.

An SMB is invaluable as a location aid on wreck, drift and decompression dives. If sausage-shaped, it should be wide and stand tall above sea level. Self-inflating deco sausages should retain their form at the surface for some time under adverse sea states. Decompression markers might provide increased location distances if they were made in the same day-glo yellow as the folding diver's flag.

Paired red Polyform buoys, the sort often used as permanent SMBs, provided location up to 1,100 m and were much easier to relocate than a single buoy, especially where single creel buoys of similar size and colour were found. The separation distance between them meant that they would yo-yo under increasing wave heights, so that at least one buoy usually remained visible.

We would recommend that such paired buoys be at least 40 cm in diameter with at least 2 m between them. Divers using any marker buoy from which they might become separated should always carry a secondary device, such as a flag.

Torches

The diving torches produced very bright beams and were clearly located during daylight at more than 4 km, increasing to beyond 9 km in darkness.

Their location was restricted when the diver thought that they were pointing directly towards the search vessel, but when moved slowly but steadily in a scanning motion (both horizontally and vertically), they could be detected over a wider sector. This also avoids temporarily blinding the skipper at close range.

Using a torch as a signalling device is strongly recommended, but we would recommend that one be kept in reserve for the purpose, because the unit used during a dive might not have the power needed.

Strobes

We used the strobes only by attaching them to the top of folding flags. We know that when attached to the shoulder of a diver's BC, the visible flash is extremely intermittent as waves lap over and around it.

The strobes were not observed until the light was very low, the Jotrun at distances up to 4 km but on average at around 2 km. It appeared to generate a more intense flash and to be more effective than the Seeman Sub strobe.

A high-intensity, good-quality strobe should always be carried when diving in low light levels, but it needs to be mounted as high as possible. We would recommend that divers carry two if they are to be primary location aids.

Any electrical device, such as torches or strobes, must be reliable and robust if it is to be relied on as a location device. Regular, thorough inspection and maintenance is needed, with special attention paid to sealing surfaces and O-rings.

Our staff frequently use torches and strobes in normal diving and have found supposedly high-quality devices as prone to leaking as lesser-quality models. The reliability of waterproof cases could be greatly improved if more manufacturers incorporated two independent seals.

Pyrotechnics

A limited number of trials were conducted with pyrotechnics, through prior arrangement with the Coastguard. The orange smoke from the PainsWessex Day/ Night Distress Signal had a burn time of 20 seconds. It produced a smoke cloud that appeared dense at 4 km and lingered for about 90 seconds. As the already considerable distance of the vessel was increased to 8.5 km, the smoke remained visible for a similar duration, but appeared more diffuse and had a much lower profile. Wind speed on this occasion was less than 10 mph (16 kph).

A smoke flare was also used during a helicopter search in Shetland, when wind speed was more than 20 mph (32 kph). The smoke cloud was rapidly diffused.

It is unlikely that any observer would locate smoke unless they happened to be looking in roughly the right direction. Smoke would be best deployed by a diver who could see that a vessel or helicopter was heading towards him.

The same applies to the night flare on the Day/Night Distress Signal, which produced a dense cloud of white smoke that was visible in daylight at 8.5 km, but was not located in darkness.

Miniflares had consistent burn times of 10 sec and were faintly observed up to 8.5 km away in daylight. In darkness they were easily located at more than 9 km. As they come in packs of 10, they would be best deployed intermittently, keeping several back for when a search vessel or aircraft is sighted.

Cold hands hamper activation of these pyrotechnics and we were not convinced that they would be practical for a diver wearing gloves. We would not recommend their use by divers under a wide range of typical diving operations.

They are not designed to be taken under water, are relatively expensive, and inconvenient to transport. Long-term reliability after repeated underwater exposure is unknown.

Dyes

A series of aerial location exercises by an SAR helicopter in Orkney and Shetland failed to locate several devices. But seen from an altitude of 300 m and then 75 m were the Presto Dyechem Sea Marker Dye (3,400 m, 760 m), the yellow flag (2,500 m, 1,450 m), the decompression bag (2,30O m, 1,200m), and the paired red Polyform buoys (900 m).

In a full-scale search exercise in the main body of Scapa Flow, co-ordinated by Pentland Coastguard at the end of our trial period, we tested a large number of devices. An area of 8 square nautical miles was searched by two lifeboats and an SAR helicopter. In just over two hours the lifeboats located all the devices within their designated sectors and some deployed in adjacent search areas.

The yellow flags provided the greatest location distances, similar to those we had found from our earlier observations.

The helicopter covered the search area in about an hour and located six out of 10 devices. The Sea-Streak Marker Dye was not located, its plume of dye being extremely diffuse. Recovery of the buoy to which it was attached required several minutes of searching at its GPS deployment position to locate it.

The Presto Dyechem dye had produced a very impressive slick from which the release remained consistent for around two hours, easily observed by helicopter. But dye generally is unsuitable as an aid for sea-level location and was never located by the search vessels.

Conclusions

It became clear during the trials that ability to relocate devices at or close to sea level varied considerably between

observers. For some divers, skippers and boat crews, visual impairments could have a profound effect on that ability.

We also found that the ability of many observers to spot different devices in varying conditions improved with practice, the conclusion being that, ideally, look-outs on diversupport vessels should be competent at relocating devices. They should also know what it is they are looking out for, so information on the devices in use might be invaluable if search and rescue (SAR) facilities are required.

Under adverse conditions, or where there is believed to be a higher risk of divers becoming separated from the cover boat, at least two people should remain on watch.

And if there is a high risk of divers becoming separated from the vessel, careful attention should be paid to how many divers are in the water at one time, and which location devices are carried.

The full report, *Diver Emergency Surface Location Devices*, can be viewed on www.jeanelaine.co.uk/diveraids/contents.htm. Reports in the OTO series can be obtained from Health & Safety Executive, Research Strategy Unit, Bootle, Merseyside, L20 3DL, United Kingdom.

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Since July 1998 (18 months) Alister Wallbank has been closely involved with hyperbaric therapies for divers with DCI as Heriot-Watt University provides its Recompression Chamber to the Local Health Board for treatment of divers with DCI. Between April and December 2000 (8 months) the chamber conducted 27 separate treatments for recreational divers with DCI. Early in 2001, four members of the Heriot-Watt University recompression chamber staff (two nurses, the Diving Officer and the author completed the Clinical Hyperbaric Medicine course. This course is UHMS approved and is most likely to become one of the recognised certifications in hyperbaric medicine.

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LOST OFF BOGNOR

John Bantin

Key Words

Environment, incident, rescue.

Imagine floating at the surface of a glass-calm sea on a lazy summer afternoon. Then imagine that you and your buddy are alone, with no sight of land, nor any surface cover! This happened to Tom Burton when he had to learn a few tough lessons as a novice in the early '70s. He went on to become a BSAC National Instructor and Regional Coach.

The sea is a big and lonely place at times. Dangerous, too. Tom and Dennis had dropped into the water from the club's boat full of enthusiasm. The surface was slightly choppy but otherwise it was a gorgeous English summer's day. They were about three miles offshore from Bognor.

Tom was relatively new to diving but Dennis, though younger, was quite experienced.

A strong current was running and the two buddies were seen hurtling along at about 15 m deep, enjoying the buzz of a fast drift dive. It was wonderful, the nearest man could get to flying like a bird. The contours of the seabed unrolled beneath them. Skates broke cover from the mud and took flight before them. Banks of dead men's fingers passed in a white blur. Without a care in the world they stayed down for as long as the air in their tanks would let them. Then it was time to surface.

The sea had taken on an oily calmness. They could see their cover boat and the tiny figures of its occupants way over on the horizon. They could make out the

movements of people looking in the water. They were obviously looking out for the divers, but in the wrong place.

Tom and Dennis shouted. They waved their arms. They whistled. They yelled. They screamed at the tops of their voices. All to no avail; no one looked in their direction. It was very frustrating. And then frustration turned to disbelief. Instead of motoring over to where the divers waited, the occupants of the boat seemed to give up and headed off in the direction of Bognor.

Tom looked at Dennis. Dennis looked extremely worried. They were all alone in the ocean. They could not see the low-lying coast, only water.

Tom told Dennis that a helicopter was bound to come to rescue them. He knew it would be all right, and he had never had a ride in a helicopter. Dennis was near to tears. Tom tried to sound calm and confident. "Maybe they'll send an RAF Shackleton to pick us up," he suggested naively.

They were wearing wetsuits. They decided to dispense with all that diving gear not required for their survival. They needed only their masks and fins. Dennis dropped everything but Tom decided to keep hold of his regulator. It was the only part of his diving gear that did not belong to the club. He had only just purchased it and it was his first regulator - a Silver Snark II.

Suddenly they became aware of an SAR helicopter. It was like a tiny yellow wasp up in the clear blue sky and was obviously searching for them. Again they waved as hard as they could. But the chopper turned and flew away.

Even Tom began to get depressed. He could not believe what was happening. The awful reality had dawned, they were divers lost at sea. Tom told Dennis he was sure that nothing bad was going to happen to them. He owed someone some money and he never got away with leaving a bad debt. Tom talked about anything he could think of. Otherwise they just dozed in the water.

About six hours had passed since they had surfaced. It was an awfully long time. The sun was beginning to get low on the horizon. Then they saw a lifeboat moving slowly in the distance. They waved furiously. Tom thought: "Beautiful!" And then it too went off the other way.

Doing nothing began to play on their minds, so they decided to try to swim for the shore. The problem was that because the coast was so low-lying near Bognor they could not see which way to go. The sun gave them a bit of a clue. They did not get very far, but it kept them busy.

Tom swam on his back. He says that he knew Dennis was spooked because he was making a definite effort to swim, whereas Tom was merely marking time. They had stopped talking altogether. Tom was just dozing on his back when Dennis saw the lifeboat again. This time it was coming near. Dennis swam frantically towards it. Tom waited for it to come to him. The lifeboat had a large net hung over the side. The two divers were grateful to be able to scramble up it.

They were soon on board and talking nineteen to the dozen. They each had a cup of tea. Tom smoked five cigarettes in succession, which was strange because he did not smoke, never had done and doesn't to this day!

They were asked many questions. An elderly lifeboatman had spotted them. He said everyone on board had thought they were two cormorants. He said he had never seen cormorants with what had looked like four wings, so they had come over to have a look.

Tom and Dennis were returned safely to shore, where the gentlemen of the press awaited them. Tom got home later than his wife expected. He got into bed and decided to eat the sandwiches which he had left in his car. His wife Sandra woke up and asked him if he'd had a nice day's diving, and why he had not eaten his sandwiches at lunch-time.

In fact she had heard Tom and Dennis had been lost on the radio news. "She's tricky like that," says Tom.

I asked Tom what lessons he had learned from that experience so early in his diving career. "It seems funny to look back on now, but it certainly wasn't funny at the time. It was summer and our wetsuits were adequate, but we were lucky to be alive. We never bothered with surface marker buoys in those days. We didn't really know what they were."

"How did the boat usually find you after a dive?" I asked.

"I don't really know, but they always seemed to manage it. Diving was not organised the way it is now. We really didn't know about a lot of the dangers. I was new to diving then and I was diving with people I thought were pretty experienced. I thought they knew what they were doing. I learned some tough lessons in those early days."

"What was the single most important thing you learned from that experience?" I asked.

"Always to use an SMB when there are tides and currents," said Tom.

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