MODES OF PRESENTATION OF PATENT FORAMEN OVALE IN TEN DIVERS

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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.

References

1 Hagen PT, Scholz DG and Edwards WD. Incidence and size of patent foramen ovale during the first ten decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984; 59: 17-20

2 Folstein MF, Folstein SE and McHugh PR. Mini Mental State. A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975; 12: 189-198

3 Windecker S and Meier B. Percutaneous patent foramen ovale (PFO) closure: It can be done but should it? *Catheter Cardiovasc Interv* 1999; 47(3): 377-380

4 Wilmshurst PT, Byrne JC and Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. *Lancet* 1989; 2: 1302-1306

5 Wilmshurst PT and De Belder MA. Patent foramen ovale in adult life. *Br Heart J* 1994; 71: 209-212

6 Lechat P, Mas JL, Lascault G, Loron P, Theard M, Klimczac M et al. Prevalence of patent foramen ovale in patients with stroke. *N Engl J Med* 1988; 318 (18): 1148-1152

7 Fisher DC, Fisher EA, Budd JH, Rosen SE and Goldman ME. The incidence of patent foramen ovale in 1,000 consecutive patients. A contrast transesophageal echocardiography study. *Chest* 1995; 107 (6): 1504-1509

8 Balestra C, Germonpre P and Marroni A. Intrathoracic pressure changes after Valsalva strain and other manoeuvres: implications for divers with patent foramen ovale. *Undersea Hyperb Med* 1998; 25 (3): 171-174

9 Moon RE, Camporesi EM and Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989; 1: 513-514

10 Germonpré P, Dendale P, Unger P and Balestra C. Patent foramen ovale and decompression sickness in sports divers. *J Appl Physiol* 1998; 84 (5): 1622-1626

11 Bove AA. Risk of decompression sickness with patent foramen ovale. *Undersea Hyperb Med* 1998; 25 (3): 175-178

12 Knauth M, Ries S, Pohimann S, Kerby T, Forsting M, Daffertshofer M et al. Cohort study of multiple brain lesions in sports divers: role of a patent foramen ovale. *Brit Med J* 1997; 314: 701-705

13 Siostrzonek P, Zangeneh M, Gossinger H, Land W, Rosenmayr G, Heinz G et al. Comparison of transesophageal and transthoracic contrast echocardiography for detection of a patent foramen ovale. *Am J Cardiol* 1991; 68: 1247-1249

14 Droste DW, Silling K, Stypmann J, Grude M, Kemeny V, Wichter T et al. Contrast transcranial doppler ultrasound in the detection of right-to-left shunts. *Stroke* 2000; 31: 1640-1645

15 Bonmer W, Shah PM, Allen H, Meltzer R and Kisslo J. The safety of ultrasonic contrast. Report of the Contrast Committee, American Society of Echocardiography. *J Am Coll Cardiol* 1984; 3: 6-13

16 Lee F and Gintzon LA. Central nervous system complication of contrast echocardiography. *J Clin Ultrasound* 1983; 11: 292-294

17 Wong RM. Breath-hold diving can cause decompression illness. *SPUMS J* 2000; 30 (1): 2-6

18 Desola J, Lundgren CEG, Battle JM, Lopez B, Alos R, Vilas F et al. 30 neurological accidents in Spanish breath-hold divers: Taravana revisited? *Undersea Hyperb Med* 2000; 27 (Suppl): addition to session T, not printed in abstract

19 Kohshi K, Katoh T, Abe H and Okudera T. Neurological accidents caused by repetitive breath-hold dives: two case reports. *J Neurol Sci* 2000;

178: 66-69
20 Dunford RG, Vann RD, Gerth WA, Pieper CF, Huggins K, Wachholz C et al. The incidence of venous gas emboli in recreational diving. *Undersea Hyperb Med* 2000; 27 (Suppl): 65
21 Wong RM. How safe is pearl diving? *SPUMS J* 1996; 26 (Suppl): 49-60
22 Johnston RP, Broome JR, Hunt PD and Benton PJ. Patent foramen ovale and decompression illness in divers. *Lancet* 1996; 348: 1515
23 Eckenhoff RG, Olstad CS and Carrod G. Human dose-response relationship for decompression and endogenous bubble formation. *J Appl Physiol* 1990; 69: 914-918
24 Wilmshurst P, Walsh K and Morrison L. Transcatheter occlusion of foramen ovale with a button device after neurological decompression illness in professional divers. *Lancet* 1996; 348: 752-753

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SUBCLINICAL DECOMPRESSION ILLNESS IN RECREATIONAL SCUBA DIVERS

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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