Severe neurological decompression sickness associated with right ventricular dilatation and a persistent foramen ovale

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Abstract

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We present the case of a 28-year-old female diver who performed a scuba air dive with significant omitted decompression obligation. She developed constitutional and neurological symptoms. Brain magnetic resonance imaging post treatment demonstrated multifocal embolic infarcts and transthoracic echocardiogram with bubble contrast on day three revealed a persistent foramen ovale (PFO) and severe right ventricular (RV) dilatation. We postulate that the high venous bubble load from the provocative decompression caused an increase in pulmonary artery pressure, leading to RV dilatation and increased right to left shunting of bubbles across her PFO, resulting in significant neurological deficits. This mechanism is analogous to that seen in acute thromboembolic pulmonary embolism.

Introduction

Right-to-left shunt is promoted by increased pulmonary artery pressures in patients with submassive or massive pulmonary thromboembolism (PE) and a persistent (patent) foramen ovale (PFO).[1] We report a patient with severe right ventricular (RV) dilatation and PFO-associated neurological and constitutional decompression sickness (DCS).

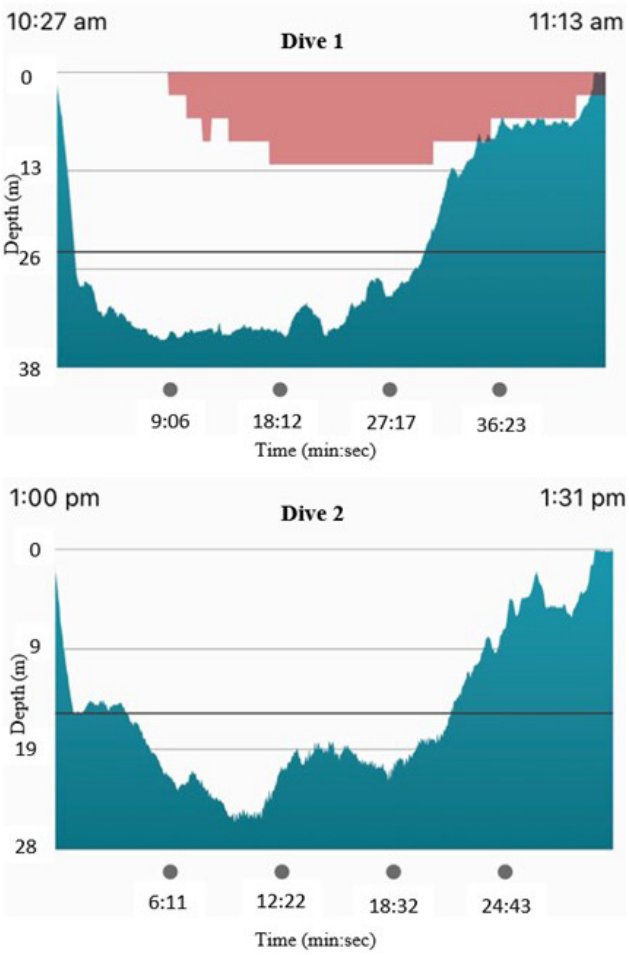
Case report

Written consent for publication of the history and images was obtained from the patient, and approval was obtained for data review and extraction by Governance, Evidence, Knowledge and Outcomes (GEKO) at Fiona Stanley Hospital (Approval Number 46996).

A 28-year-old woman, who had performed 44 previous dives and had no significant past medical history or medications except combined oral contraceptive, performed a scuba air dive to 35 metres of seawater (msw) for 44 minutes (min). She had performed a single dive to 12 msw for 89 min 24 hours (h) earlier. Therefore, according to The Defence and Civil Institute of Environmental Medicine (DCIEM) air diving tables, the deeper dive was a non-repetetive dive.[2] There was no rapid ascent but she omitted a significant decompression obligation, surfacing with a nitrogen loading of 122% according to her dive computer as calculated by the Bühlmann ZHL-16C algorithm. During the surface interval of 1 h 48 min, the patient developed progressive nausea and bilateral lower limb paraesthesia. Despite this, she completed a second dive to 26 msw for 30 min with her symptoms improving slightly at depth. The dive profiles are shown in Figure 1. The conservatism level on the dive computer was set to high for dive 1 and medium for dive 2.

After surfacing from the second dive her symptoms worsened, with vomiting, thoracic back pain and neurological symptoms including headache, unsteadiness, bilateral upper and lower limb paraesthesia and leg weakness. She denied chest pain, breathlessness or syncope. The patient was taken to shore by police boat with face mask oxygen, where she was met by an ambulance 2 h 45 min after surfacing from the second dive. Her observations on shore were respiratory rate 22 breaths·min^{-1}, oxyhaemoglobin saturation 98% with 15 L·min^{-1} oxygen via non-rebreather face mask, pulse 120 beats·min^{-1}, blood pressure 116/76 mmHg and temperature 36.5°C. She had clear lungs on auscultation with mildly

Figure 1
Dive profiles downloaded from patient's Garmin dive computer. X axis – time (minutes:seconds), Y axis – depth (metres); red shaded area – decompression ceiling as per Bühlmann ZHL-16C algorithm



increased work of breathing. She was transported by road to Fiona Stanley Hospital, the State Referral Centre for Diving and Hyperbaric Medicine in Western Australia.

Upon arrival to hospital, 3 h 40 min from surfacing, she reported ongoing nausea, thoracic back pain, headache and progressive paraesthesia to her legs and fingers. Examination revealed bidirectional end gaze nystagmus and reduced power (4/5) to the left leg, with equal and symmetrical limb reflexes. Tympanic membranes were normal and there was no rash. Blood tests revealed haemoconcentration consistent with dehydration and a leucocytosis (Table 1). Electrocardiogram (ECG) showed normal sinus rhythm without T-wave abnormalities or signs of right heart strain and a chest X-ray was normal. Bedside lung ultrasound excluded pneumothorax and pulmonary oedema. The patient was treated with intravenous (IV) antiemetics and 2 L of crystalloid fluid.

Table 1
Initial blood results upon arrival to hospital; CRP – c-reative protein; hs-TnI – high sensitivity troponin I levels

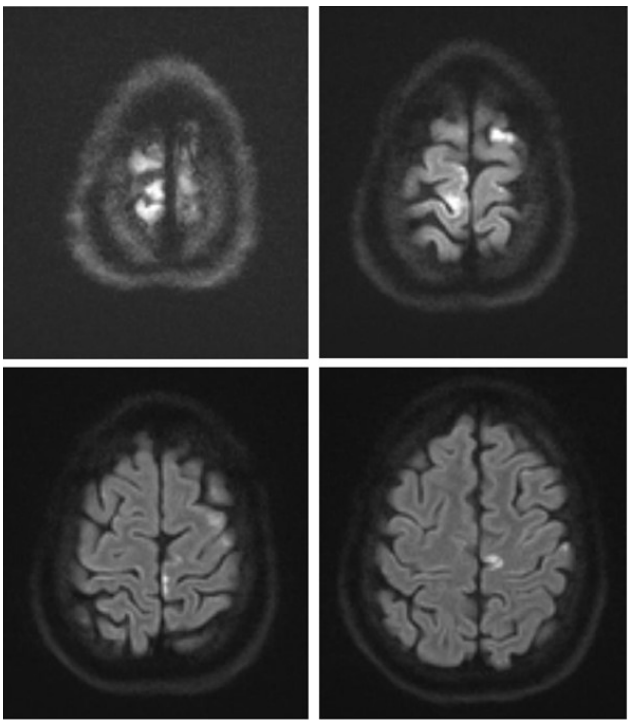
Test	Result	Normal range	Units
Haemoglobin	188	115–160	$g{\cdot}L^{-1}$
Haematocrit	0.57	0.37–0.47	$L{\cdot}L^{-1}$
White cell count	32.89	4–11	$x10^{9}{\cdot}L^{-1}$
Neutrophils	29.95	2.0–7.5	$x10^{9}{\cdot}L^{-1}$
CRP	6.7	< 5	$mg{\cdot}L^{-1}$
Sodium	138	135–145	$mmol{\cdot}L^{-1}$
Potassium	4.2	3.5–5.2	$mmol{\cdot}L^{-1}$
Bicarbonate	18	22–32	$mmol{\cdot}L^{-1}$
Urea	4.3	3.0–8.0	$Mmol{\cdot}L^{-1}$
Creatinine	96	45–90	$Umol{\cdot}L^{-1}$
Troponin I (Abbot hs-TnI)	6	< 16	$Ng{\cdot}L^{-1}$

The patient received hyperbaric oxygen treatment (HBOT) with a modified United States Navy Treatment Table 6 (USN TT6) without extensions in a monoplace chamber. She vomited during the treatment, however her nausea settled as the treatment progressed. When reviewed after completing her initial recompression her symptoms had improved, but she had ongoing paraesthesia, nausea and ataxia. The patient developed a fever of 38.1°C after the treatment, with rapid antigen test for COVID negative and urinalysis showing a trace of leucocytes. She had no sick contacts nor focal infective symptoms and was treated with further IV fluids and commenced on IV antibiotics (piperacillin with tazobactam). The following morning she had persistent ataxia, nystagmus and lower limb paraesthesia and she received further HBOT with United States Navy Treatment Table 5 (USN TT5).

Brain magnetic resonance imaging (MRI) with angiogram on day one post-dive showed multifocal areas of cortical diffusion restriction in the supratentorial brain, including the right parafalcine motor strip, consistent with multiple embolic infarcts, without intracranial arterial stenosis (Figure 2). Spinal MRI was normal. The patient was commenced on aspirin and admitted by the neurology service. The patient completed twice daily HBOT for two days after her initial USN TT6, then daily until symptom plateau plus one for a total of 22 treatments (Table 2). Thrombophilia screening was normal. The oral contraceptive was stopped due to potential pro-coagulant effect.

A transthoracic echocardiogram (TTE) with bubble contrast was performed on day three post-dive. This showed normal left ventricular systolic function with ejection fraction of 59% by Simpson's biplane method. The RV was severely

Figure 2
Brain magnetic resonance imaging day one post-dive; Axial slices demonstrating multifocal cortical diffusion restriction (white areas) in keeping with multiple embolic infarcts



dilated (mid ventricle diameter 4.18 cm, normal < 3.5 cm) with flattening of the interventricular septum predominantly in diastole, consistent with increased RV filling pressures with normal systolic function (Figure 3). Agitated saline contrast revealed a large right-to-left interatrial shunt at rest. The following day, transoesophageal echocardiogram (TOE) with bubble contrast injection confirmed a dilated RV with preserved systolic function, a dilated right atrium (RA) with bowing of the interatrial septum indicating high RA pressure, and a PFO with moderate size right-to-left shunt at rest.

Cardiology were consulted and cardiac MRI (Figure 4) was performed on day five post-dive. The RV was moderately dilated with mildly impaired systolic function. There were no valvular lesions nor evidence of RV infarction. The RA was moderately dilated. The aetiology of the dilated right heart was thought to be increased pulmonary circulation resistance secondary to a high bubble load within the pulmonary capillary bed from DCS. Arrhythmogenic right ventricular cardiomyopathy was thought to be unlikely.

Repeat TTE on day 14 post-dive showed a reduction in RV size compared to previous (mid-ventricle diameter 3.95 cm) with preserved systolic function. The patient was discharged from hospital on day 14 post-dive, with her remaining HBOT performed as an outpatient. She received occupational therapy and physiotherapy. After completing a total of 22 sessions of HBOT, she still had residual deficits including

Table 2
Hyperbaric oxygen treatment table and chamber type used; 14:90:08 – 243 kilopascals / 2.4 atmospheres absolute for 90 minutes with 8 minute decompression; 14:90:24 – 243 kilopascals / 2.4 atmospheres absolute for 90 minutes with 24 minute decompression; Mono – Monoplace; Multi – Multiplace; USN TT5 – United States Navy Treatment Table 5; USN TT6 – United States Navy Treatment Table 6

Days post-incident dive	Treatment table	Chamber
0	USN TT6	Mono
1	USN TT5 x 2	Multi
2	USN TT5 x 2	Mono
3	USN TT5	Mono
4	14:90:08	Mono
5	14:90:08	Mono
6	USN TT5	Multi
7	14:90:08	Mono
8	14:90:08	Mono
9	14:90:08	Mono
10	14:90:08	Mono
11	14:90:24	Multi
12	14:90:24	Multi
13	14:90:08	Mono
14	14:90:08	Mono
15	14:90:08	Mono
16	14:90:08	Mono
17	14:90:08	Mono
18	14:90:08	Mono
19	14:90:08	Mono

reduced voluntary control of her left arm, gait disturbance to her left leg, and changes to higher level executive function.

A TTE was repeated three months post-dive which showed interval reduction in RV size, with mild RV dilatation (mid ventricle diameter 3.5 cm) and normal RA size. Cardiac MRI was repeated five months post-dive which showed ongoing mild RV dilatation with an ejection fraction of 48%. She was reviewed at the hyperbaric unit six months post-dive and reported feeling well overall, however she reported persistent paraesthesia to both legs with ongoing balance issues and a best sharpened Romberg test of 25 seconds on her third attempt.

A PFO closure was performed seven months post-dive using a Gore Cardioform Septal Occluder® where she was found to have a large PFO, opening to six mm. She was treated with antiplatelet agents (aspirin and clopidogrel) for six months.

Figure 3
Transthoracic echocardiogram apical four chamber view day three post-diving showing end-diastolic right ventricular (RV) dilatation; medial - lateral diameter given in centimeters at base (normal < 4.1 cm) and mid ventricle (normal < 3.5 cm); bpm – beats per minute

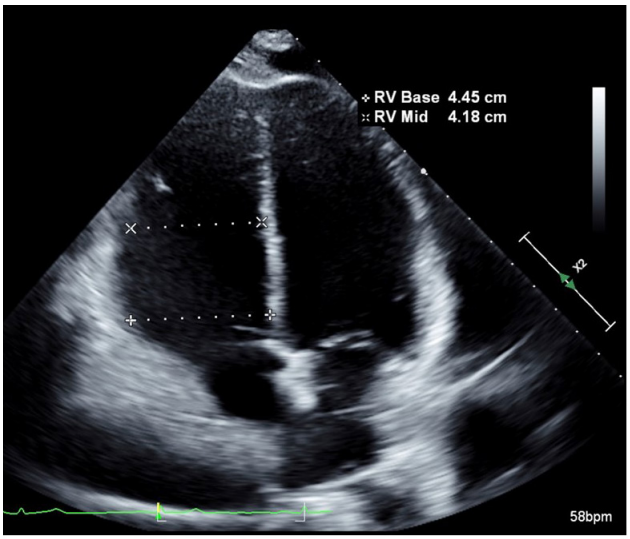


Figure 4
Cardiac magnetic resonance imaging day five post-dive; upper panel – short axis view; lower panel – four chamber view; demonstrates right ventricular dilatation in diastole. RV – right ventricle; LV – left ventricle

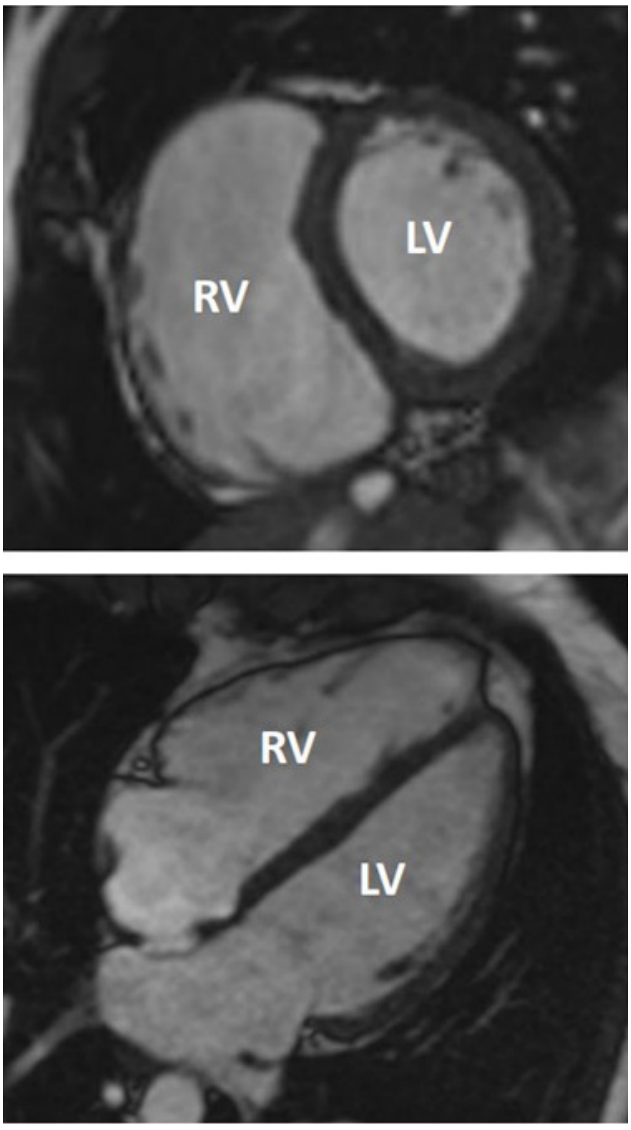
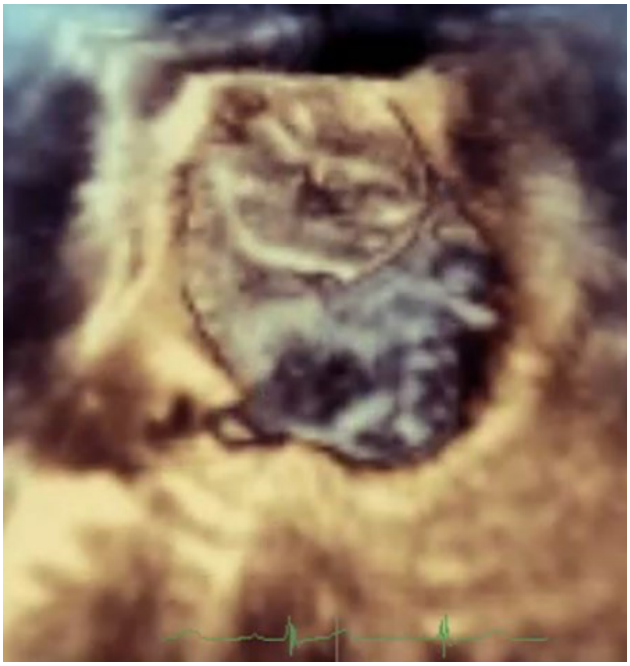


Figure 5
Transoesophageal echo 6 months post-PFO closure showing Gore Cardioform Septal Occluder® *in situ*



A TOE with bubble contrast performed six months post-PFO closure showed a well seated device without residual right-to-left interatrial shunt (Figure 5).

Discussion

Cardiopulmonary DCS typically presents early after surfacing from provocative dives, with a spectrum of symptoms including chest pain, dyspnoea and cough progressing to shock and death in severe cases.[3,4] Historically, severe cardiopulmonary DCS was associated with unsafe pressure-time profiles, particularly in caisson workers. For example, Ghawabi and colleagues reported a DCS rate of 0.97% after caisson workers were exposed to air pressures equivalent to 28 msw for up to six hours and 25 msw for up to eight hours.[5] The authors reported that 48 of 55 (87%) workers had at least one episode of DCS during the project, and 37 of the 55 (67%) of workers experienced cardiopulmonary DCS ('the chokes'). The high incidence rates are consistent with unsafe profiles and, because nearly every worker had DCS at least once, there is no need to postulate the role of physical predisposition to DCS, such as right-to-left shunts. Wilmshurst and colleagues compared the prevalence of atrial shunts in 97 divers who had DCS with 109 divers who had

not had DCS. Twelve divers had cardiorespiratory DCS and seven of those had an atrial shunt, which was a significantly greater prevalence than in normal divers, which was 26 of 109 (24%).[6]

In the patient we describe, cardiorespiratory symptoms were not prominent, despite the severe degree of right heart dilatation, but she was initially tachycardic and tachypnoeic. The lowest oxyhaemoglobin saturation recorded acutely post-dive was 98%, although she was on supplemental oxygen for the duration of her initial management.

Animal models have demonstrated that when the pulmonary circulation is overwhelmed by increasing numbers of venous gas emboli, the pulmonary artery pressure can increase such that right heart failure and haemodynamic instability can ensue.[7,8] In this patient, the postulated mechanism of RV dilatation, with bubbles causing physical obstruction of the pulmonary vasculature and hence increasing pulmonary artery pressure, is analogous to that found when pulmonary thromboembolism (PE) causes increased pulmonary vascular resistance and pulmonary artery pressure.[9] The clinical effect of PE in patients with a PFO is well described. Submassive or massive pulmonary emboli can promote right-to-left shunting of deoxygenated blood across a PFO due to elevated RA and RV pressures, causing worsened hypoxaemia.[10] In patients with haemodynamically significant PE, those patients with a PFO were found to have a higher incidence of peripheral or visceral arterial occlusions and of cerebral infarction.[1] Of 139 patients with major PE (echocardiographic evidence of RV pressure overload or presence of pulmonary artery hypertension), the incidence of ischaemic stroke was increased in those with a PFO (13% vs 2.2%) as well as peripheral arterial embolism (15% vs 0%).[11] In our patient, elevated right heart pressure could similarly have increased the shunting of venous gas emboli across the PFO, causing the profound embolic manifestations seen on brain MRI.

Coronary artery gas embolism is another potential mechanism for bubble mediated right heart dysfunction in this case. In the supine position, in which the patient was initially managed, gas emboli can preferentially enter the right coronary artery as the right coronary sinus is the most superior and anterior in this position.[12] Coronary air embolism could potentially stun the myocardial territory supplied i.e., the RV. This is seen rarely after iatrogenic air embolism during cardiac catheterisation, but can cause complications including haemodynamic instability, myocardial infarction, ventricular arrhythmias and death.[13] This is another possible cause of right heart dilatation, but is unlikely in this case given the absence of ECG or troponin abnormality.

Consideration was given to the alternative hypothesis that the patient might have had pre-existing pulmonary hypertension with subsequent right heart dilatation, however this was deemed unlikely. The PFO was considered too small to cause significant right heart dilatation through a left-to-right shunt. Furthermore, if this were the case, a gradual reduction in RV diameter over time would not have been anticipated.

Another hypothesis was of chronic thromboembolic pulmonary hypertension resulting from asymptomatic pulmonary emboli, with the use of oestrogen containing oral contraceptive being a risk factor for thromboembolism. However, this was also considered improbable, as the patient was asymptomatic and had good pre-morbid exercise tolerance, although imaging for pulmonary embolism was not performed. The patient was treated with antiplatelet agents but was not anticoagulated, therefore we would not expect any chronic thromboembolism to resolve, with subsequent reduction in pulmonary hypertension in the absence of anticoagulation.

If an acute submassive pulmonary embolism had occurred around the time of the dive causing right heart dilatation, this would have caused symptoms such as chest pain and breathlessness, which the patient did not report. Instead, the patient displayed other signs of DCS, including nausea, vomiting and paraeshesias. These symptoms, coupled with a provocative dive profile with omission of decompression obligation and improvement with recompression therapy, make DCS a more plausible explanation.

Typically, RV dilatation due to acute pulmonary thromboembolism can take a significant time to resolve with right ventricular dysfunction being persistently present a year later in around 34% of cases.[14] In this case, the RV dilatation decreased from severe (mid-ventricle diameter 4.18 cm) on day three to the upper limit of normal (mid-ventricle diameter 3.5 cm) within three months. This further reduces the likelihood that thromboembolic disease was the underlying cause.

Conclusions

We report a case of DCS-related right heart dilatation, likely caused by bubble-mediated increase in pulmonary artery pressure. This is analogous to PE increasing pulmonary vascular resistance and right heart pressures. The increased right atrial pressure can increase right-to-left shunting across a PFO and facilitate paradoxical embolism of venous bubbles. This is the first report to the authors knowledge of transient RV dilatation from DCS associated with a PFO.

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