Underutilization of echocardiography for patent foramen ovale in divers with serious decompression sickness.

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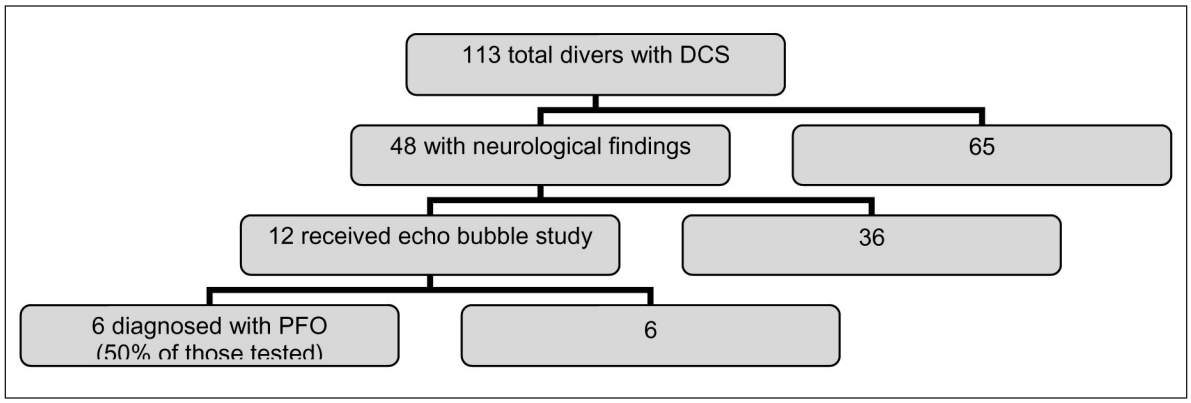
Harrah JD, O'Boyle PS, Piantadosi CA. Underutilization of echocardiography for patent foramen ovale in divers with serious decompression sickness. Undersea Hyperb Med 2008; 35(3):207-211. The presence of a patent foramen ovale (PFO) in compressed gas diving has been considered a risk factor for serious decompression illness (DCS) for more than 20 years. We conducted a ten year retrospective chart review aimed at determining if physicians treating DCS in a university medical center setting used echocardiography to assess PFO in patients with severe DCS, and if so whether PFO is over-represented in that population. Over the ten-year period, 113 divers underwent recompression therapy for decompression sickness. Of these patients, 48 had serious DCS defined by at least one objective neurological finding. We reviewed medical records for the presence of agitated saline contrast echocardiogram testing and whether or not PFO was present. Only 12 of 48 patients with serious DCS underwent transthoracic agitated saline contrast echocardiogram testing. Of these 12 patients, 6 (50%) had a resting PFO. Binomial proportion testing yielded 95% confidence limits of 21% and 79%. Given 27% PFO prevalence in the general population, PFO may be over-represented in our group of most seriously injured DCS patients yet 75% of patients with objective neurological signs did not undergo echocardiography.

INTRODUCTION

In 1986, Wilmhurst observed neurological decompression sickness in a recreational scuba diver and attributed its cause to venous gas embolism passing through a previously undocumented atrial septal defect (15). Since then, scuba divers with PFO have been considered at increased risk for severe DCS involving the CNS due to paradoxical embolization of nitrogen bubbles. In a sample of 66 professional divers, Cartoni found that PFO at rest was the only predictor for type II DCS.(3) Despite the fact that PFO has a prevalence of approximately 27% in the general population, Bove reported that the presence of a PFO resulted in only a 2.5-fold increase in the risk for developing serious DCS (1, 6). And Torti found that the presence of a PFO produced a 5-fold increase in the risk for developing serious DCS, but computed an absolute risk for severe DCS in divers with a PFO at only 5 per 10,000 dives (12). This retrospective chart review aimed to determine if physicians in a university medical center make use of this information by ordering bubble echocardiography in patients with severe DCS and if so whether PFO is over-represented in this cohort of patients.

Fig. 1. Breakdown of findings in the patients in this study.



METHODS

Using the Duke Center for Hyperbaric Medicine and Environmental Physiology standardized clinical database, we identified all consecutive, adult recreational divers with DCS treated between 1/1/97 and 10/6/07. A subset of this group with severe decompression sickness was defined by a provocative exposure and symptoms consistent with DCS and at least one objective neurological finding present on arrival. We excluded altitude-induced DCS patients and patients with isolated shallow-water signs and symptoms consistent with acute arterial gas embolism (AGE). Patients of both genders and all races were included. We reviewed the medical records of patients with severe DCS to determine if treating physicians had ordered agitated saline contrast echocardiogram testing. Patients found to have undergone echocardiogram testing were segregated according to the presence or absence of PFO and their presenting signs and symptoms were recorded. This protocol was approved by the Duke University Health System Institutional Review Board. Data was recorded without patient identifiers to ensure privacy of health information.

RESULTS

From 1997 to 2007, 113 scuba divers were treated for decompression sickness at Duke University Medical Center. Of these, 97% were Caucasian and the average age was 40 years. The youngest diver was 19 and the eldest 67 years old. Women represented 17% of the total population. Of the 113 decompression sickness patients, 48 (43%) had severe DCS evidenced by at least one objective neurological sign. Of 48 divers with severe DCS, only 12 (25%), received transthoracic agitated saline contrast echocardiography and all echocardiograms were performed at the same large, academic medical center. As these echocardiograms were performed over a ten year span, they were read by several cardiologists. Neither transcranial Doppler ultrasound nor transesophageal echocardiogram studies were performed. Six of 12 (50%) severe DCS patients tested by transthoracic agitated saline contrast echocardiography had a detectable PFO. Although Valsalva was used as a provocative measure at time of contrast injection, all demonstrated PFO were present at rest. These data are summarized in Figure 1. The 95% confidence limits on our observed proportion of 50% are from 21% to 79%. One of 12 echo-tested, serious DCS patients is

female, and she had a PFO. However, gender was not significantly associated with detection of PFO in this population (p=.99). For most of our patients, subsequent diving practice was not documented.

Table 1. PFO Positive Divers	
Signs / Symptoms	Dive profile
cutis marmorata / vertigo / ataxia	135 ft for 30 min, rebreather, multiple deco stops, cold water, drysuit, prior history of DCS
cutis marmorata / ataxia / altered mental status	117 ft for 59 min on EAN 32 with 20 min 75% O2 deco; then 110 ft for 39 min on EAN 32 with 10 min deco stop at 15 ft
Vertigo / altered mental status / ataxia	175 ft for 51 min, 2:20 SI then 106 ft for 40 minutes; dove the previous day
left leg sensation loss / unstable gait	115 ft for unknown time then 100 ft for unknown time; dove the previous day
right leg weakness / right arm weakness ataxia	122 ft for 29 min 32% EAN with safety stop and 2 hr SI; then 117 ft for 26 min without stop
Left arm weakness, unstable gait	114 ft for 32 min on 31% EAN with 120 min SI; then 113 ft for 33 min with 5 min deco; dove the previous day

Table 2. PFO Negative Divers	
Signs / Symptoms	Dive Profile
paraparesis/ sensory loss/ unstable gait	111ft for 20 min, 4 min safety stop
paraparesis/ sensory loss/ unstable gait	65 ft for 25 min with fast ascent then 65 ft for 25 min, several dives in prior week
paraparesis/ urinary retention/ sensory loss/ unstable gait	111 ft for 20 min with 3:10 SI; then 113 ft for 28 min; safety stops for both; dove the prior day
paraparesis/ bilateral leg sensory loss unstable gait	119 ft for 10 min with safety stop
Right arm & leg weakness with sensory loss	83 feet for 40 min on 34% EAN; extensive prior day diving
right arm and right leg sensation loss; vertigo, unstable gait	111 ft for 30 min on 30% EAN; rough seas with exertion at the surface

Table 1 lists the presenting signs and symptoms for each of the PFO positive, echo-tested divers with severe DCI. It also includes dive profile information for each of these divers. Table 2 lists the same information for each of the PFO negative, echo-tested divers with severe DCS. We note that none of the patients with a PFO had paraparesis, while 4/6 (67%) without PFO had suffered paraparesis (p=.06).

DISCUSSION

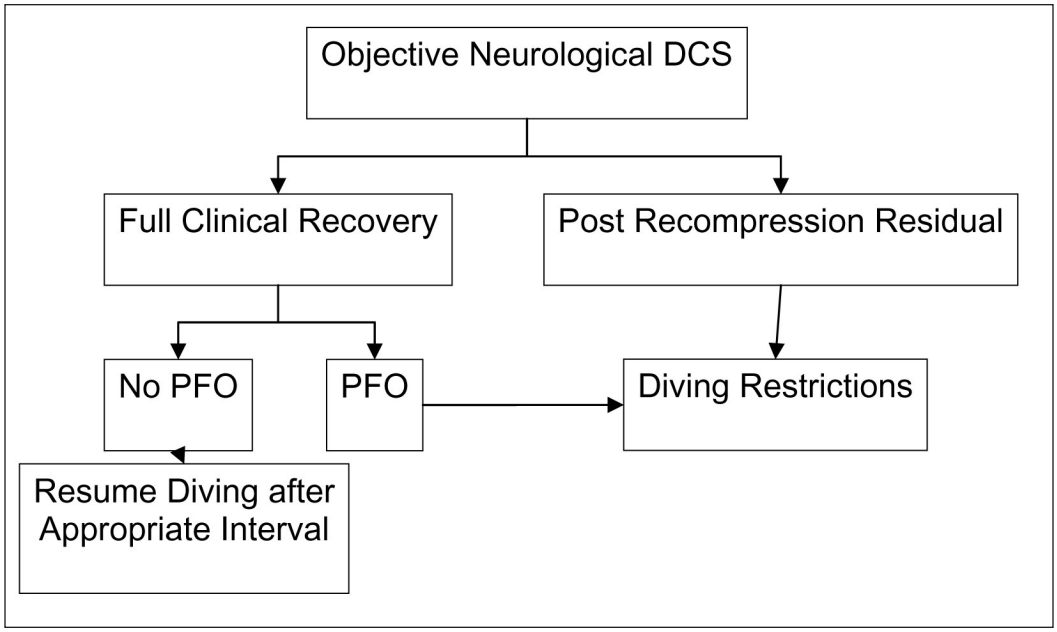
In this series of consecutive cases, 50% of echocardiogram-tested, severe decompression sickness patients were found to have a PFO. Given the confidence interval of 21% to 79% and prevalence in the general population of 27%, we cannot conclude that PFO is significantly overrepresented in our sample (p=0.1521) (6). However, the small sample size and low p value increases the possibility of a type I statistical error.

This data is consistent with the 1989 findings of Moon et al (10). In their sample of 18 severe DCS patents, 61% had echo-proven PFO. Our data is also consistent with the findings of Germonpre, who found that 60% of 37 severe DCS divers had a PFO (4). Furthermore, it is consistent with the findings of Honek, who found that 53% of 15 severe DCS patents had a PFO (7).

In our sample of agitated saline contrast echocardiogram-tested severe DCS patients, those with PFO appear to have had more cerebral, cochleovestibular and cutaneous signs. This finding is consistent with prior work showing that these signs are more likely in divers with PFO (4) (2) (8) (14). Moreover, we found that paraparesis was more common in patients with severe DCI and no PFO. This suggests the possibility that the most common mechanism of neurological injury is different in these two subsets of injured divers.

A significant limitation of our study is that it is prone to referral bias, and one of the reasons for collecting the data was to determine if physicians treating patients with serious DCI in an academic setting order the echocardiogram to check for PFO and then use it to advise patients about future diving practices. The data suggest that this is not the case despite the confirmation that PFO is overrepresented in divers with severe DCI. Our findings suggest that transthoracic agitated

Fig. 2. Flow diagram for the evaluation of patients with objective neurological findings of DCS on clinical presentation.



saline contrast echocardiography should be used to test for PFO in injured divers with objective neurological findings. A positive PFO bubble study might tip the scale away from continued diving. At a minimum, it should lead to more conservative diving recommendations.

A simple flow diagram for evaluating these patients is provided in Figure 2. We believe the evidence supports performance of transthoracic agitated saline contrast echocardiography on all severe DCS patients, as some patients who initially say they are finished with diving do change their minds. For those with permanent neurological injury or presence of a PFO after neurological DCS, diving restrictions should be considered. These restrictions could vary from "no diving" to a graded return to conservative diving, but this would depend on the severity of the deficit and patient-physician negotiation. Residual post-recompression neurological deficits, especially if they involve the spinal cord, are contraindications to further diving as they do represent an increased risk of further disability from another episode of decompression illness (5).

While percutaneous transcatheter PFO closure can be performed, this procedure is not without risk and its effectiveness in preventing DCS is uncertain (9, 11, 13). Routine screening for PFO in asymptomatic recreational divers is not recommended; however, it seems prudent to provide this information to all divers who have suffered a neurological injury from DCS.

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