PATENT FORAMEN OVALE ASSOCIATED WITH TYPE II DECOMPRESSION SICKNESS IN EXPERIMENTAL NO-DECOMPRESSION DIVES

Gary W. Latson, M.D., Alfred Bove, M.D., Kenneth Hopper, Robert Perkins, M.D.
Navy Experimental Diving Unit (NEDU), Panama City, Florida, U.S.A.

INTRODUCTION: In recent years right-to-left shunting (RLS) via patent foramen ovale (PFO) or other cardiac defects has been increasingly investigated as a possible source of arterial bubbles in decompression sickness (DCS) and arterial gas embolism (AGE). Several studies suggest that divers who have experienced DCS have a prevalence of PFO higher than that of the control population.[1–6] Figures 1 and 2 demonstrate the potential mechanism. In the "normal" individual, venous bubbles are formed during decompression in a dive, but the lung acts to filter the bubbles from the blood. In the diver with a PFO, however, bubbles in the right atrium may pass through the PFO directly to the left side of the heart and may then be pumped directly to the systemic circulation, including the brain, where they may precipitate symptoms of CNS DCS.

The U.S. Navy does not routinely evaluate or exclude diving candidates for PFO/RLS, but some experimental divers and divers who have experienced DCS or AGE are evaluated. A series of studies at Navy Experimental Diving Unit (NEDU) resulted in observations of a higher than expected prevalence of PFO/RLS in divers with DCS resulting from experimental trials of extended bottom times in dive profiles at depths of 130 to 190 fsw with no decompression stops. This protocol aimed to further investigate this observation by determining the prevalence of PFO/RLS in a sample of divers who were from the same study and did not develop DCS.

BACKGROUND: NEDU conducted a series of experimental dives to test extended bottom times in dives to depths of 130, 150, and 190 fsw with no-decompression.[7] A series of 444 precisely controlled exposures in 88 divers resulted in 6 cases of serious Type II decompression sickness (DCS). Dive Profiles and DCS Cases are summarized in Table 1.

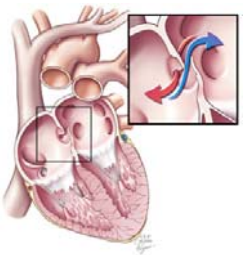


Figure 1. Foramen Ovale

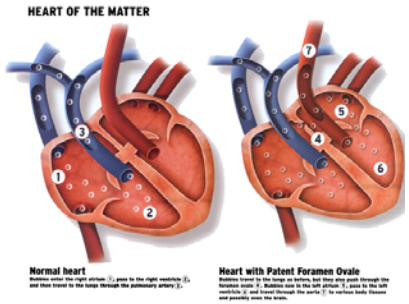
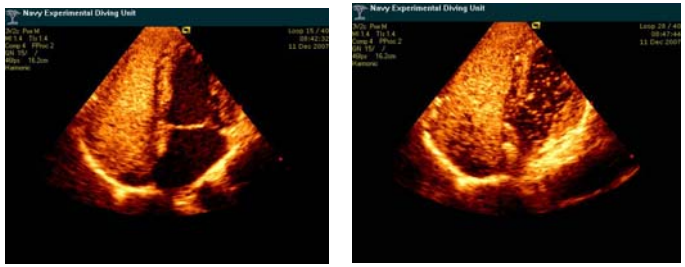


Figure 2.

Case	Age	Dive Profile fsw:min	Presenting Symptoms	Onset Time (min)	PFO, RLS Grade	Comments
1	41	190:11	"Heavy" legs, abdominal pain and numbness, hand numbness	40	Pos, II	Atrial Septal Aneurysm and Atrial Septal Defect
2	43	150:15	Visual field deficit (left eye, lower half)	25	Neg, 0	
3	43	130:20	Right LE weakness and numbness	20	Pos, II	Persistent residual sensory deficit
4	49	130:20	Dizziness, weakness/paralysis of arms and legs	18	Pos, II	Persistent residual sensory deficit
5	42	150:12	Hip/flank pain, LE weakness, altered mental status, visual field defect	10	Pos, I	Prior hx DCS, prior observation RLS at rest
6	37	190:9	Dizziness, gait disturbance, altered mental status, blindness	17	Pos, III	Elected PFO closure

Table 1. DCS Cases. Notes: Standard No-D limits 130/10, 150/5, 190/5



Figures 3, 4. The grading scale for TTE gas emboli is as follows:

GRADE 0 no bubbles detected in left heart on TTE.
GRADE I occasional bubble signal discernable, less than 20 bubbles in single static image.
GRADE II over 20 bubbles in static image, but still discreet, countable.
GRADE III Too many bubbles to count during all cardiac periods but not enough to interfere with cardiac motion image.
GRADE IV many bubbles throughout all cardiac cycles with bubble signal overriding the normal cardiac motion image.

METHODS: After IRB approval, all cases were matched with 24 controls of similar age. Each underwent a uniform PFO screening procedure. Transthoracic echocardiography and transcranial doppler (TCD) monitoring of the middle cerebral artery were performed with agitated saline bubble contrast at rest and during a timed valsalva maneuver. Bubbles in the left atrium within four cardiac cycles of bubbles filling the right atrium was considered to be evidence of a PFO. A grade of 0 to IV was used to describe magnitude of RLS based on number of bubbles in the left heart (figures 3, 4). Detection of emboli by TCD was considered confirmatory evidence of RLS. An expert Cardiologist with no prior knowledge of the status of the diver evaluated the results and made the determination of PFO and RLS grade. Data was analyzed using Fisher's exact test and a logistic regression model of the form Logit (DCS)=RLS grade+number of dives.

RESULTS: 5 of the 6 case subjects (83%) were positive for PFO/RLS. The magnitude of RLS was grade III for 1 subject, and grade II for 3 subjects and grade I for 1 subject. Only 6 of 24 controls (25%) had a detectable PFO/RLS, and the magnitude of RLS was much less, with 2 subjects having grade II and 4 subjects with grade I. TCD results were consistent with echocardiographic observations. Fisher's exact test comparing DCS and PFO/RLS was significant (p=.015), as it was for DCS and shunt grade (p=.009). Logistic regression showed an odds ratio for each step in shunt grade of 5.57 (95% confidence limit 4.4 to 7.0). The number of dives each subject performed was not significant.

DISCUSSION: This is the first study involving a large number of controlled exposures, directly observed cases of Type II DCS, and consistent testing methodology for PFO/RLS. It contributes new evidence for the role of PFO/RLS in *some* types of diving exposures and *some* types of DCS. It should be noted that these diving exposures were beyond the normal range of no-decompression diving. It is also notable that these divers had years of uneventful normal diving experience, including several thousand working dives, and some prior dives in this series, without DCS. Readers should not generalize these results to normal diving activities where other evidence indicates a more modest effect of PFO on risk of DCS.[6] When a diver does suffer Type II DCS, it may be prudent to evaluate for underlying cardiac anomalies such as PFO, atrial septal defect, or ventricular septal defect since these may be associated with other conditions and are potentially correctable.

CONCLUSION: In these experimental no-decompression profiles, Type II DCS was associated with the presence of a PFO/RLS and the magnitude of shunt.

LITERATURE CITED

1. T. Gerriets et al., "Arteriovenous Bubbles Following Cold Water Sport Dives: Relation to Right-to-Left Shunting," *Neurology*, Vol. 55, No. 1 (2000), pp. 1741–1743.
2. S. Ries et al., "Arterial Gas Embolism after Decompression: Correlation with Right-to-Left Shunting," *Neurology*, Vol. 52, No. 1 (1999), pp. 401–404.
3. M. J. Saary and G. W. Gray, "A Review of the Relationship between Patent Foramen Ovale and Type II Decompression Sickness," *Aviat Space Environ Med*, Vol. 72, No. 12 (2001), pp. 1113–1120.
4. P. Germonpre et al., "Patent Foramen Ovale and Decompression Sickness in Sports Divers," *J Appl Physiol*, Vol. 84, No. 5 (1998), pp. 1622–1626.
5. P. Germonpre, et al., "Patent Foramen Ovale: A Risk Factor for Cerebral Decompression Sickness in Sports Divers," *Proc XXII Annual Congress of EUBS* (1996), pp. 509–512.
6. A. A. Bove, "Risk of Decompression Sickness with Patent Foramen Ovale", *Undersea Hyperb Med* Vol. 25 (1998), pp. 175-8.
7. D.J. Doolette, W.A. Gerth, K.A. Gault, "Empirical Evaluation of Extensions to No-Stop Limits for Deep Air Diving", *Undersea Hyperb Med*, Vol. 34, No 4 (2007), p. 232.