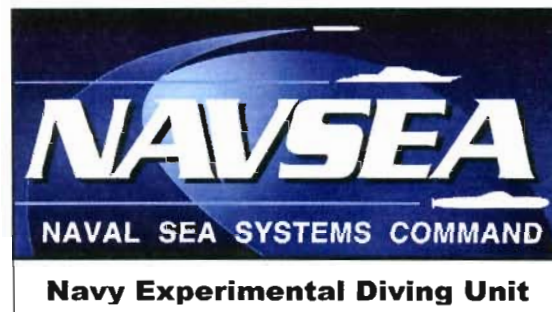


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**CASE CONTROL STUDY OF TYPE II DECOMPRESSION
SICKNESS ASSOCIATED WITH PATENT FORAMEN OVALE
IN EXPERIMENTAL NO-DECOMPRESSION DIVES**



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19. ABSTRACT (Continue on reverse if necessary and identify by block number) This is a retrospective case control study comparing decompression sickness (DCS) risk in experimental Navy divers with a patent foramen ovale/right-to-left shunt (PFO/RLS) and in age-matched experimental Navy divers without a PFO/RLS. Eighty-eight subjects completed 432 man-dives in an empirical evaluation of extensions to air diving no-stop limits at Navy Experimental Diving Unit. Six subjects experienced neurologic DCS. Review of transthoracic echocardiography and transcranial Doppler imaging discovered a PFO/RLS in five of those subjects who experienced DCS and in six of 24 divers who had no DCS and had been recruited from the 82 remaining divers participating in the evaluation of the air diving no-stop limits. Logistic regression analysis indicates that after completing selected experimental no-stop dives to at least 150 feet of seawater with bottom times extended beyond current Navy diving limits, divers with a PFO/RLS are at increased risk for DCS.				
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INTRODUCTION

In recent years passage of venous gas emboli (VGE) through right-to-left shunts (RLS) of the cardiovascular system has been increasingly scrutinized as a possible source of arterial bubbles in decompression sickness (DCS) and arterial gas embolism (AGE). Particular attention has been directed at a common intracardiac shunt called patent foramen ovale (PFO). Several cross-sectional studies suggest that divers who have experienced central nervous system (CNS) DCS have a prevalence of PFO higher than that of the control population.¹⁻⁵ For example, in a study of 37 Belgian sport divers with a history of CNS DCS, 60% (22/37) had PFOs, while controls matched in age and dive experience had the expected PFO prevalence of 36% (13/36).⁵ From these observations many have inferred that having a PFO is a factor in developing some forms of CNS DCS.

In the normal individual, venous bubbles can form during decompression after a dive or hyperbaric exposure. The bubbles enter the heart at the right atrium, move to the right ventricle, and are then pumped to the lungs. The pulmonary capillaries, very small blood vessels within the lungs, filter the bubbles from the blood. Very few, if any, bubbles are then returned to the left side of the heart and subsequently pumped into the systemic circulation.

It is theorized that in the diver with a PFO, bubbles in the right atrium can pass through the PFO directly to the left atrium and thus bypass the pulmonary capillary filter. Such bubbles may then be pumped directly to the systemic circulation, become arterial gas emboli, and precipitate symptoms of CNS DCS. PFO is the most commonly recognized type of RLS, and it has drawn attention because it may occur in otherwise healthy, asymptomatic people. Other possible sources of RLS include atrial septal defects, ventricular septal defects, arteriovenous malformations of the pulmonary vasculature, or functional arteriovenous shunts within the pulmonary vasculature. While these theories of the pathogenesis of CNS DCS are commonly discussed in the diving medical literature, the pathophysiology has not been documented in either animal models or human studies.

The presence of a PFO is not disqualifying for diving duty. The U.S. Navy does not currently evaluate asymptomatic diving candidates for RLS and PFO, but divers who have experienced CNS DCS or AGE are evaluated for PFO/RLS according to guidelines issued by the Navy Bureau of Medicine and Surgery (BUMED) and judgments of cognizant Diving Medical Officers (DMOs). Some divers who have served as experimental subjects in diving studies have also been screened for PFO as part of research projects.

In a series of 432 dives designed to evaluate extensions to the air diving no-stop limits and completed by 88 divers at the Navy Experimental Diving Unit (NEDU), resulting rates of DCS were within current U.S. Navy standards but of greater than anticipated severity. Six cases of DCS resulting from the experimental dive profiles with no decompression stops at depths from 130 to 190 feet of seawater (fsw) occurred: all were Type II CNS DCS requiring immediate recompression therapy. Five of these six subjects were subsequently found to have PFO/RLS by the screening technique described in the

PFO/RLS TESTING PROTOCOL section of this report. The same screening method discovered that six of 24 age-matched controls recruited from the remaining 82 divers who completed the same dive profile and did not develop DCS also had PFO/RLS. DCS risk in PFO/RLS positive divers and the PFO/RLS negative divers were compared.

TASKING

In 2002 Naval Sea Systems Command (NAVSEA 00C) had tasked NEDU to "perform a case control study in Navy divers to establish the excess risk, if any, of decompression sickness and arterial gas embolism in divers with patent foramen ovale (PFO) and other right to left shunts."⁶ That task had been originally addressed in NEDU Protocol 03-17/32130, *Case Control Study of DCS Risk Associated with Patent Foramen Ovale in Navy Diving*. Intended to build on data initially obtained in that study, the current study also includes observations of subsequent DCS cases among some participants in that study. NAVSEA 00C Task Assignment 07-08 provided specific tasking for the current study,⁷ which was carried out under NEDU Protocol 07-17⁸ after being approved by the NEDU Institutional Review Board.

METHODS

GENERAL

A sample of experimental subjects diving under NAVSEA Task 04-12 in two phases of NEDU Protocols 04-41/32158 and 06-28/32194, *Empirical Evaluation of Extensions to Air Diving No-Stop Limits*,^{9,10} was asked to undergo testing for PFO/RLS. The current investigation is a case control study of how PFO/RLS affects DCS risk in these experimental divers.

SUBJECT SELECTION

Six of 88 divers who completed the dives in NEDU Protocols under NAVSEA Task 04-12 developed DCS. Seventy-four of 82 subjects who did not develop DCS were recruited to participate in this study as controls. They were not asked to participate in any further diving as part of this particular study. Sixty-one of these 74 responded to the initial inquiry, and 59 of the 61 agreed to participate by filling out a questionnaire and undergoing a series of diagnostic tests. Twenty-four of the 59 were selected as control subjects on a basis of age matching to those six subjects with DCS. All six subjects who had developed DCS during the experimental NEDU dives were recruited and agreed to participate in the study.

Because of subject concerns that a significant RLS or PFO might become a disqualifying condition for future diving duty, this study was designed to stringently protect subject anonymity. All subjects were assigned numerical codes at the outset, and all subsequent paperwork, data recordings, and computer files used these number codes. The appropriate numerical codes were entered for the diver survey

questionnaire after the study was begun, and subject names and identifying information were stripped from the tops of all questionnaires. A codebook was maintained in the NEDU library vault until the study was completed; the codebook was then destroyed. No personnel maintained any specific identifying information — e.g., exact age, date of birth, height, etc. — about participants in this study. However, some of the recruited controls — fearing that their careers as divers might be affected — may have known their PFO status and opted out of participation.

The ultrasonographer and Principal Investigator (PI) were not blinded to either the transthoracic echocardiography (TTE) or transcranial Doppler (TCD) examination results.

From testing conducted either as part of a previous study or as medical follow-up for DCS, the PFO/RLS status of the six divers who had developed DCS was already known. The PFO/RLS status of some other divers who had participated was also known from part of a previous study's testing, from medical follow-up, or from diver request. To ensure consistency of testing methods, this known data was not used, and all divers who became subjects of the current study were asked to undergo repeat determinations of PFO/RLS.

To seek permission for releasing the names of each participant to the investigators of the current study, investigators responsible for the no-stop studies sent requests to all their participants. Only those subjects who consented to having their names released were included on a list of potential participants for the current study. An impartial investigator (who was an investigator on neither the no-stop nor this current study's protocols, and who had no knowledge of the PFO/RLS status of individual divers) reviewed diver demographics and exposure records and selected potential divers whose ages and exposure histories were most similar to those of the divers who had developed DCS. The selected divers were then asked to voluntarily participate in this study and to submit to testing for PFO/RLS, with the results held as confidential. Current study investigators anticipated that some divers would not volunteer to be tested; thus, the number of divers in the initial request was greater than the minimum needed.

DEFINITION OF DECOMPRESSION SICKNESS

Focused on the relationship between PFO/RLS and DCS, this study involved the central nervous system (CNS DCS). For this study's purposes, CNS DCS was determined by an NEDU DMO following completion of an experimental dive and treatment with recompression therapy. According to the diagnosing DMO, all cases of suspected DCS that occurred during the no-stop studies under NAVSEA Task 04-12 were CNS DCS.

STATISTICAL TESTS AND SAMPLE SIZE

This case control study tested the null hypothesis that the DCS risk among PFO/RLS positive divers is the same as that among PFO/RLS negative divers. Since the subjects varied in number of experimental no-stop dives completed and in PFO status, two

models were proposed for comparison to the null. Model 1 considered both number of dives and PFO/RLS status. Model 2 considered only PFO/RLS status. A likelihood ratio test indicated that both models fit the data equally. By convention, the simpler model (Model 2) was chosen for comparison to the null. The null hypothesis would be rejected if the DCS risk for PFO/RLS positive divers were different than that for PFO/RLS negative divers by Fisher's Exact Test (two-sided $\alpha = 0.05$). Alpha was the upper bound for the probability of incorrectly rejecting the null hypothesis. Statistical power was the probability of correctly rejecting the null hypothesis at a given level of significance and specified difference between the groups. The controls in this study were those divers who did not develop DCS after completing the same dive profile as the DCS positive divers. The cases are those of the DCS positive divers. A sample size calculation was performed: 23 control subjects were required for a statistical power of 80%. Twenty-four control subjects participated in the study.

SENSITIVITY OF PFO DETECTION METHOD

The recognized standard for definitively diagnosing atrial-septal defects (including PFO) of all sizes is widely considered to be contrast-enhanced transesophageal echocardiography (TEE), which involves placing an ultrasound (US) probe into the esophagus to generate high-resolution images of the heart. Unfortunately, TEE is invasive, normally requires sedation of the patient, and can be associated (rarely) with major complications. Furthermore, sedation of the patient limits his or her ability to perform a vigorous Valsalva maneuver, a limitation that may restrict his or her ability to detect a PFO/RLS that depends upon a transiently elevated right atrial pressure. The technically easier TTE, in which the US transducer is held against the skin of the chest just below and lateral to the left nipple, provides a less detailed anatomical image — but it allows the patient a greater degree of cooperation in performing a Valsalva maneuver and may therefore be better able to detect some functional PFO/RLS. Sensitivity and specificity are variable and depend at least partially on operator factors including patient instruction regarding the Valsalva maneuver, timing the injection of the bubble contrast solution, and the choice of injection site of the contrast solution.¹¹

TCD involves placing specialized Doppler US probes over the temporal region of the skull to detect flow in the underlying arteries. This technique is highly sensitive for detecting intra-arterial bubble emboli. With appropriate timing of bubble contrast and Valsalva maneuver, this technique has been shown to be highly sensitive and specific for detecting, diagnosing, and grading of most RLS. We chose to use simultaneous TTE and TCD to provide the best diagnostic evidence available without sedation.

Testing for PFO/RLS was performed as detailed in the **PFO/RLS TESTING PROTOCOL** subsection below, with TTE and TCD monitoring during injections of bubble contrast. One contrast injection was performed with the subject breathing normally, and a second injection of contrast was performed at the end of a ten-second period with the subject performing a Valsalva maneuver. As outlined in that same subsection, PFO/RLS was detected and graded by direct observation of the echocardiogram for bubbles in the left atrium and left ventricle. TCD detection of embolic signals was recorded. Timing of TCD

embolic signals in relation to echocardiographic evidence of bubbles was recorded. TTE imaging was collected and stored in a data file under the test subject's numerical code. TCD images and data were similarly collected and stored.

To ensure that determination of PFO/RLS was as objective and unbiased as possible, a cardiologist experienced in diagnosing PFO/RLS reviewed data files including echocardiographic images, TCD summary, and notes on timing of Valsalva, contrast injection, and other relevant information. This reviewer was not informed of the opinions of the technician and investigator, and he did not know whether the subject had ever suffered DCS. This expert reviewer's determination of PFO/RLS prevailed if his opinion differed from that of the investigator.

EQUIPMENT AND INSTRUMENTATION

The following equipment was used during testing:

- a. Medasonics Model CDS Transcranial Doppler with head fixation device, and
- b. Acuson Cypress Ultrasound System with color Doppler.

PFO/RLS TESTING PROTOCOL

While bilateral TCD signals in the anterior cerebral artery (ACA) and middle cerebral artery (MCA) were monitored through the temporal bone, a saline bubble contrast was injected into a right arm vein during normal respiration, and a second contrast injection was made during a Valsalva strain. Bubble embolic tracks (ETs) appearing on the power m-mode display of the Medasonics Model CDS TCD were counted and recorded according to right or left distribution. With the unilateral logarithmic scale from grade 0 to V for both normal respiration and Valsalva, the TCD recordings were graded according to the side with the greatest number of tracks. Concurrent with the TCD study, up to five minutes of a four-chamber apical TTE image was collected. Observation of bubbles in the left atrium or ventricle at times corresponding to bubble presence in the right atrium and ventricle was considered to be positive evidence for PFO/RLS. TCD ETs were considered to be evidence confirming RLS.

Position and intravenous access

Subjects were placed recumbent on their left sides. A qualified phlebotomist (usually the investigator) placed an 18-gauge intravenous (IV) catheter with a plastic tube extension to double three-way stopcocks in the right antecubital vein. Alternatively, a more distal vein in the right arm or hand was used. A continuous IV drip of 0.9% normal saline was run at the rate of approximately 30 cc/h.

TCD Monitoring

TCD monitoring was performed with the beam directed to include the bilateral middle cerebral and anterior cerebral arteries (MCAs and ACAs). For this monitoring, the TCD

probe was located on the side of the face over the indentation immediately above the condyloid process of the mandible (the condyloid process can be felt moving during mastication) and anterior to the ear. A head frame stabilized the probe. The spectrogram sample volumes were set on the MCAs and ACAs at a depth of 5–6 cm. While listening to the MCA spectral signal, technicians observed the power mode Doppler for ETs. Using the "autodetect" mode, they noted and reported any spontaneous microembolic tracks on the power mode. Bilateral TCD recordings were obtained when possible. Because of anatomic variables or technical factors, bilateral TCD signals could not be obtained on one subject. He was PFO negative by TTE and unilateral TCD, and the variation from the desired testing method was noted.

Valsalva Strain

To improve their performance of the Valsalva maneuver during actual testing, subjects performed a simulated Valsalva during the initial monitoring before the contrast was injected. They were instructed to hold a breath at mid exhalation, forcibly tighten their abdominal muscles, and hold pressure for approximately ten seconds while the TTE display was monitored.

Agitated Saline Bubble Injection

Approximately 9.5 cc of 0.9% saline solution and 0.5 cc of air were drawn into a 10 cc syringe via the first of two three-way stopcocks in the previously placed IV line. Back and forth exchanges of the saline-air mixture to and from a second 10 cc syringe attached to the second three-way stopcock produced a suspension of microbubbles (agitated saline bubble contrast). While the microbubbles were being generated, the stopcocks were closed to the IV line. Opening the second stopcock to the IV line leading toward the subject and injecting 5 cc of the contrast suspension in a single bolus over a 1–2 s time period created the bubble contrast. After this injection of contrast, the stopcocks were turned to stop any further contrast injection and provide a continuous flow of normal saline solution from the IV bag to the subject.

At intervals of 5 min, two injections of the bubble contrast were made: the first with normal respiration, and the second with the respiratory strain (Valsalva maneuver). Before the first contrast injection, the technician positioned the US transducer head in the appropriate location to obtain a TTE apical four-chamber view of the subject's heart. When the first dose of contrast was administered, the US technician began recording TTE images and continued recording them for about one minute after each contrast injection.

After 5 min, when no evidence of bubbles remained from the first injection, the subject was asked to commence a ten-second Valsalva. The second contrast bolus was injected approximately 6–9 s after the Valsalva began and before the Valsalva was released. As the bubble contrast appeared in the right atrium, the TTE image was observed closely. Valsalva release after approximately 10 s was usually evident on the TTE display by transient enlargement of the cardiac image. Observation of bubbles in

the left atrium at the same time that bubbles were in the right atrium, and within four cardiac cycles of the release of the Valsalva, was considered a positive finding. At the discretion of the technician or investigator, a brief cough-sniff maneuver, or a second Valsalva, was sometimes performed after the first Valsalva procedure. The test was repeated after 5 min in three of 30 TTE studies because the image was poor or the results were not clear. Recording of TCD to identify the microembolic event continued for 5 min after each contrast injection.

All data and images were stored in computer files labeled with the appropriate subject numerical codes and test identifiers.

Grading of Right-to-Left Shunt Conductance

The following expanded six-level grading scale created by Spencer Vascular Laboratories (Seattle, WA) predicts the conductance of an RLS measured in ET. The conductance, which depends on both the size of the RLS and the pressure gradient, is positive if a TCD spectrogram detects any ETs in the cerebral circulation. To report both the resting conductance and the straining conductance, the numbers of all microbubble ETs were counted separately on the TCD display for the two injections.

The grading scale for unilateral TCD monitoring is as follows:

GRADE 0	0 embolic tracks
GRADE I	1 to 5 embolic tracks
GRADE II	6 to 15 embolic tracks
GRADE III	16 to 50 embolic tracks
GRADE IV	51 to 150 embolic tracks
GRADE V	>150 embolic tracks, many uncountable

These scales allow the RLS's capability to conduct embolic material directly from the venous to the cerebral circulation to be quantified.

The grading scale for TTE gas emboli is as follows:^{12,13}

GRADE 0	no bubbles detected in left heart
GRADE I	occasional bubble signal discernable, but predominately bubble free
GRADE II	many (>20) bubbles, but less than half of cardiac cycle period with bubbles
GRADE III	bubbles 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

RESULTS

Results are summarized in Tables 1A, 1B, and 2 below. Further details regarding the dive exposures and DCS events can be found in the NEDU technical report on this dive series.¹⁴

Table 1A. Data Summary, DCS Case Subjects

ID #	Age	Years Diving	Dives in protocol	DCS Dive Profile fsw:min	Presenting Symptoms	Onset Time (min)	TTE PFO: RLS Grade	TCD Grade	Comments
7349	41	24	3	190:11	"Heavy" legs, abdominal pain and numbness, hand numbness	40	Pos: II	II	Possible atrial septal aneurysm, atrial septal defect
1126	43	15	5	150:15	Visual field deficit (left eye, lower half)	25	Neg: 0	0	
6308	43	17	2	130:20	Right LE weakness and numbness	20	Pos: II	II	Persistent residual sensory deficit, prior hx DCS
6215	49	26	7	130:20	Dizziness, weakness/paralysis of arms and legs	18	Pos: II	II	Persistent residual sensory deficit
3717	42	17	7	150:12	Hip/flank pain, LE weakness, altered mental status, visual field defect	10	Pos: I	I	Prior observation RLS at rest prior hx DCS
1203	37	18	4	190:9	Dizziness, gait disturbance, altered mental status, blindness	17	Pos: III	III	Elected PFO closure

Table 1B. Data Summary, Non-DCS (Control) Subjects

Subject ID #	Age	Years diving	Dives in Protocol	TTE, PFO: RLS Grade	TCD Emboli Grade
9546	40	16	11	Neg: 0	0
5723	42	13	8	Neg: 0	0
7699	47	24	3	Neg: 0	0
1481	39	19	3	Pos: I	I
8468	41	21	5	Neg: 0	0
6302	38	16	5	Pos: I	II
7256	45	25	5	Pos: I	I
7085	48	22	8	Neg: 0	0
6176	47	24	4	Neg: 0	0
1927	40	22	1	Neg: 0	0
3751	49	19	1	Pos: I	III
3661	38	5	7	Neg: 0	0
3708	46	18	9	Neg: 0	0
3504	38	15	11	Neg: 0	I
2465	40	10	1	Neg: 0	0
4690	38	19	3	Neg: 0	0
7414	39	8	3	Neg: 0	0
6698	46	18	4	Neg: 0	0
1726	46	26	8	Neg: 0	0
5800	38	8	5	Pos: II	I
9151	37	6	3	Neg: 0	0
8066	38	7	5	Neg: 0	0
6685	43	18	10	Pos: II	II
4856	37	18	2	Neg: 0	0

Model 1 considers both number of no-stop dives and PFO status in the DCS risk model. Model 2 considers only PFO status. These models were compared by the likelihood ratio test. By convention, if two models fit the data equally well (see likelihood ratio (LL) = -11.5 and $p=0.946$ in Table 2), the simpler of the two is accepted. Therefore, Model 2 — indicating that DCS risk is dependent on PFO status and independent of number of no-stop dives — was accepted. The null model assumes equal DCS risk for all divers. By comparing Model 2 and the null model, investigators rejected the null model ($p=.008$). Therefore, Model 2 — indicating that DCS risk is dependent on PFO status — was accepted. The Model 2 odds ratio (15.0) indicates an increased DCS risk for divers with a PFO after they dove the table and schedule described in NEDU Protocols 04-41/32158 and 06-28/32194, *Empirical Evaluation of Extensions to Air Diving No-Stop Limits*.

Table 2. DCS Risk Model Comparison					
	Variables	Odds Ratio	95% CI	LL	P
MODEL 1	PFO	14.9	(12.5, 17.2)	-11.5	
	# dives	1	(0.6, 1.4)		
MODEL 2	PFO	15	(12.6, 17.3)	-11.5	0.946*
NULL				-15.0	0.008**
*comparison of Models 1 and 2					
**comparison of Model 2 and NULL					

TCD results were consistent with echocardiographic observations (Table 2), with embolic events detected to coincide with echocardiographic images of bubbles in the left atrium. TCD emboli were noted in every case where a PFO/RLS was detected by TTE. The grade of RLS by TCD criteria matched the TTE RLS grade in nine of eleven cases; in two cases the TCD grade was higher than the TTE grade. In one case the TTE image was poor because of anatomical factors, but markedly positive TCD signals correlating well with the Valsalva contrast injection prompted a repeated testing, which was clearly positive for PFO/RLS on TTE. In only one case did TCD indicate emboli when TTE observation did not detect PFO/RLS. In that case emboli signals were isolated, and timing of those signals did not correlate well with the bubble contrast and Valsalva maneuver. It is likely that the detected emboli resulted from late transpulmonic passage of a few bubbles.

DISCUSSION

This study contributes new evidence for an increased risk of DCS in divers with PFO/RLS. Data to conduct a statistical analysis of RLS grade were insufficient, but the data demonstrate a possible trend toward increased DCS risk with increasing TTE RLS grade. Only one of six subjects with DCS had grade 0 emboli, whereas 15 of 24 controls had such grades. Four of six subjects with DCS had a grade II or higher RLS grade, whereas only one of 24 controls had such a grade. Other studies demonstrate increased DCS risk with high RLS grades, a result indicating that additional studies in experimental diving might discover that the possible trend in DCS frequency and RLS grade is real. For example, Torti et al¹⁵ performed a retrospective study of 230 divers (28 of whom had suffered DCS) and noted an increase in DCS risk ratio with an increase in size of PFO. In their study the risk of having a CNS DCS event was nearly the same for divers without PFO and those with a small (Grade I) PFO, but that risk was increased 4.4-fold for divers with Grade II PFO and 6.6-fold for divers with Grade III PFO.

We should note that the experimental dives completed by the recruited population were outside the limits of standard Navy diving. It is unusual for a series of experimental dives to produce only CNS DCS without other forms of DCS occurring. In standard Navy diving CNS decompression injury is much less common than other types of less severe decompression injury. It is reasonable to speculate that the experimental dive profile completed— with a relatively deep exposure, a bottom time beyond the current Navy diving limits, and a direct ascent with no decompression stops — might provoke the evolution of venous gas emboli that can pass through a PFO/RLS (becoming an arterial gas embolus) to damage CNS tissues more readily than it would provoke other mechanisms of decompression injury. Further studies should be done to determine the qualitative effects that a dive profile has on PFO-associated DCS risk.

Readers should not generalize these results to normal diving activities, where other evidence indicates a modest effect of PFO on risk of DCS. In a 1998 meta-analysis of three studies, Bove estimated that the risk of CNS DCS was increased about 2.6 times by the presence of a PFO, but the increase in absolute risk is still small (from 2.3 to 5.7 cases per 10,000 dives).¹⁶ It is notable that divers in the current study, including those who had PFO/RLS and who developed CNS DCS, had undergone years of uneventful normal diving experience, including several thousand working dives — and some prior experimental no-stop dives — without developing DCS.

TCD testing was not as valuable as predicted. While its results were generally consistent with TTE findings, the additional information did not change the final determinations of PFO presence or RLS grade. It did not actually improve sensitivity or specificity of the TTE testing method, and it was found to be time consuming and technically difficult to do simultaneously with TTE. While TCD testing could be valuable as a screening test for RLS if TTE were unavailable, such testing does not differentiate the source of RLS and is thus more prone to false positive findings than TTE is. TTE proved to be as sensitive as TCD for RLS and more specific in detecting PFO than TCD is, and TTE provided additional information such as atrial septal wall motion abnormalities in some cases. If TTE is

performed by well-trained technicians in future studies, the principal investigator feels that TCD is not necessary.

CONCLUSION

This study indicates an increase in DCS risk for divers with a PFO after they have dived the table and schedule described in NEDU Protocols 04-41/32158 and 06-28/32194, *Empirical Evaluation of Extensions to Air Diving No-Stop Limits*.^{9,10}

RECOMMENDATIONS

While this study does indicate an increase in DCS cases for divers with a PFO versus for those without a PFO in experimental diving beyond the current limits of the Navy tables, these results do not apply to general Navy diving. However, the finding that DCS risk depends on PFO in some experimental dives suggests that variability of DCS risk in general Navy diving should be studied. PFO might be one of many biomedical variables that influence DCS risk, but insufficient evidence exists to require additional PFO/RLS screening to the current physical standards for qualification as a U.S. Navy diver.

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