

Patent foramen ovale and decompression sickness in sports divers

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Germonpré, P., P. Dendale, P. Unger, and C. Balestra. Patent foramen ovale and decompression sickness in sports divers. *J. Appl. Physiol.* 84(5): 1622–1626, 1998.—Patency of the foramen ovale (PFO) may be a cause of unexplained decompression sickness (DCS) in sports divers. To assess the relationship between PFO and DCS, a case-control study was undertaken in a population of Belgian sports divers. Thirty-seven divers who suffered from neurological DCS were compared with matched control divers who never had DCS. All divers were investigated with transesophageal contrast echocardiography for the presence of PFO. PFO size was semiquantified on the basis of the amount of contrast passage. Divers with DCS with lesions localized in the high cervical spinal cord, cerebellum, inner ear organs, or cerebrum had a significantly higher prevalence of PFO than divers with DCS localizations in the lower spinal cord. For unexplained DCS (DCS without commission of any diving procedural errors), this difference was significant for large PFOs only. We conclude that PFO plays a significant role in the occurrence of unexplained cerebral DCS, but not of spinal DCS. We further stress the importance of standardization and semiquantification of future PFO studies that use transesophageal contrast echocardiography.

diving adverse effects; transesophageal echocardiography; paradoxical embolism

DECOMPRESSION SICKNESS (DCS) may occur in sports divers even after uneventful dives, without any (reported) error in the standardized accepted decompression procedures. Patency of the foramen ovale (PFO) is frequently found in these divers.

In animal studies in which a severe dive profile yields a high venous nitrogen bubble load, PFO may induce paradoxical arterial nitrogen bubble emboli and be the cause of DCS (12). Unlike in the normal situation, where no significant blood shunting occurs, a rise in pulmonary arterial pressure, and retrograde rise in right atrial pressure, secondary to the pulmonary embolization of nitrogen bubbles, might be responsible for a significant right-to-left shunt through the PFO. In divers, this phenomenon could also occur, accounting for at least some of the “unexplained” DCS episodes. It has been hypothesized that these paradoxical nitrogen emboli migrate into the brain, owing to both the aortic cross-flow patterns and the upright position of the diver during and immediately after ascent, thus causing high-spinal, cerebral, cerebellar, vestibular, or cochlear DCS symptoms.

In the present study, we determined the prevalence of PFO in a population of Belgian divers with neurological DCS and compared it with the prevalence in a matched population of control divers without a history of DCS. The relationship between PFO and spinal or cerebral DCS was examined, as were the severity of the dive profile and the circumstances of the dive and accident.

METHODS

Study Protocol

All Belgian divers who suffered from neurological DCS in the period from 1991 to 1995 and who were treated in either the Ostend Naval or Brussels Military Hospital Hyperbaric Centre were reviewed for participation in the study.

The study protocol was approved by the Ethics Committee of our institutions, and informed consent was obtained from all subjects.

Exclusion criteria were the following.

Uncertain diagnosis of DCS as judged by history, clinical presentation, or evolution. DCS is, fortunately, a rare complication of scuba diving; in Belgium, some 30 cases are reported annually. The majority of these cases present with neurological symptoms. Sometimes, because of the minor, vague, and subjective symptoms reported, the diagnosis of neurological DCS is only tentative. Most of these cases, despite rapid and aggressive hyperbaric treatment, do not respond well, shedding more doubt as to the initial diagnosis. These cases, accounting for ~50% of all DCS, were excluded.

Cerebral air embolism, arising from microscopic pulmonary barotrauma, has been described as a possible cause of unexplained DCS (13). Although it is not possible to rule out this diagnosis with certainty, cases in which this was considered likely were rejected. Careful clinical examination and dive profile review were carried out at the time of the DCS episode by hyperbaric medicine specialists, who were aware of this possibility. Whenever the slightest doubt existed that pulmonary barotrauma could have contributed to the origin of the decompression pathology, pulmonary high-resolution computed tomography was carried out, as well as spirometry (including flow-volume loops). Only when these examinations revealed no abnormalities was the diver included. This was the case for 13 divers. Furthermore, all subjects underwent, immediately before transesophageal echocardiography (TEE), a comprehensive transthoracic echo-doppler examination. None of the subjects showed evidence of pulmonary hypertension (pulmonary artery acceleration time was <120 ms, maximal velocity of regurgitant tricuspid flow, when present, was below 2.5 m/s) or any sign of cardiac dysfunction, which would have led to the exclusion of the diver.

Unreliability of reported dive profile, defined as persistent inconsistencies in the diver's history. This was judged from discrepancies in the dive profile reports in the medical file,

the insurance files, and the personal interrogation of the diver at the initial interview for this study. These excluded cases accounted for up to 20% of the total number of cases evaluated.

Unwillingness to cooperate. Six divers were excluded because of refusal to undergo a TEE, as specified by the study protocol (see below). Some of these divers did undergo transthoracic contrast echocardiography (TTE), but, because of the low sensitivity of TTE compared with TEE, especially when semiquantification of the PFO was being tried (7), they were nevertheless excluded from analysis.

Evidence of cardiac or pulmonary disease at the time of the investigation. Arrested diving activity or history of previous DCS was not considered an exclusion criterion.

Thirty-seven divers with neurological DCS were finally included. According to the symptoms, they were classified as having suffered from "spinal" DCS (uni- or bilateral lower extremity paresthesia, paresis, or paralysis, bladder or bowel dysfunction, or a combination of these, often with middorsal pain as the first presenting symptom) or "cerebral" DCS (cerebral, cerebellar, high-spinal, vestibular, or cochlear symptoms). The dive profile characteristics recorded were the following: dive depth, bottom time, successiveness of dive, type of dive computer or set of dive tables used, necessity for decompression stops, omission of any decompression stops, and rapidity of ascent (according to the dive planner used).

Also, "minor" risk factors were noted, such as predive fatigue, stress, alcohol consumption, or possibility of predive dehydration (inadequate fluid intake), physical exertion, or feeling of cold during the dive, and postdive exercise. A DCS episode was classified as unexplained when no errors were made as to ascent rate or decompression stops, with a maximum of three of these "minor" risk factors.

For each participating diver, a matched control diver, who never suffered DCS, was selected from the population of Belgian divers. A great deal of attention was given to this selection of matched control divers. Because DCS is, in most cases, a multifactorial event, all possible interfering parameters were sought to be matched with regard to factors influencing nitrogen uptake during a dive (such as age, gender, body mass index, physical fitness), factors influencing pulmonary integrity (smoking), and factors generally influencing DCS risk (such as lack of diving experience). Anticipating the hypothesis that diving and/or repeated Valsalva maneuvers could be associated with a failure to fuse or secondary reopening of a foramen ovale (see DISCUSSION), we also selected control divers on the basis of years of diving experience and ear equalization method used. Thus matching was performed on the basis of the following criteria: age (± 5 yr); gender; height and weight (wt/ht^2 : ± 2 kg/m^2); smoking habits [classified into three categories according to the number of pack years ($\text{pack}\cdot\text{yr}$; calculated as $\text{packs}/\text{day} \times \text{yr}$ of smoking) smoked: <5 , 5 – 10 , or >10 $\text{pack}\cdot\text{yr}$]; physical condition, as judged by diver and examiner (roughly estimated as bad, moderate, or good); diving experience (no. of yr diving, total no. of dives, $\pm 10\%$); and tubar permeability (method used for ear equalization). Thirty-six control divers were finally selected.

All divers underwent TEE with the use of agitated saline for contrast. In brief, TEE was performed by means of a multiplane echocardiographic probe (HP Sonos 2500) in the awake or mildly sedated patient. The interatrial septum was located, and the ultrasound probe was positioned to allow a clear view of both right and left atrium. Via an antibrachial vein perfusion, agitated saline (9.5 ml saline with 0.5 ml air, pushed back and forth 10 times in a double-syringe system) was rapidly injected to obtain contrast generation. Correct

injection of this volume resulted in a massive opacification of the right atrium. The number of bubbles appearing in the left atrium within three heart cycles after complete opacification of the right atrium was noted.

After at least two injections, each with a 1-min interval to clear the right atrium completely of remaining bubbles, the patient was asked to perform a high-strain Valsalva maneuver, which was held for ~ 10 s before release. Agitated saline was injected during this maneuver. At the arrival of the first bubbles in the right atrium, the patient was instructed to release the strain. This resulted in a brisk leftward bulging of the interatrial septum. Again, any passage of bubbles to the left atrium was noted.

The TEE method was applied in a strictly standardized fashion by two experienced cardiologists. All TEE sessions were recorded onto high-resolution videotape (super-VHS) and reviewed at a later stage by both cardiologists together, in a blinded manner. Bubble counts were performed manually on high-quality still-frame video images. Care was taken to exclude nonsmoke respiratory spontaneous contrast (11).

PFO was classified into three grades: *grade 0*, no contrast passage at rest or after Valsalva strain; *grade 1*, no or slight (<20 bubbles) contrast passage at rest or after Valsalva strain; and *grade 2*, important (≥ 20 bubbles) contrast passage at rest or after Valsalva strain.

Statistical Analysis

Results were analyzed by means of a standard statistical software package on an IBM PC (SPSS for Windows, version 6), the null hypothesis being that there would be no difference in PFO prevalence between DCS divers and control divers in any of the subgroups. *P* values were calculated by using Fisher's exact test. Biometric and dive data were analyzed by using Student's unpaired *t*-test or Fisher's exact test where appropriate.

RESULTS

The overall prevalence of PFO in DCS divers was 22 of 37 (59.5%), which tended to be higher than in the matched control divers: 13 of 36 (36.1%, $P = 0.06$) (Table 1).

In the subgroup of divers with cerebral DCS, the prevalence of PFO was significantly higher than in

Table 1. Prevalence of PFO

	No. of Divers With PFO	No. of Divers With Grade 2 PFO
All types of DCS ($n = 37$)	22 (59.5)	19 (51.3)
All control ($n = 36$)	13 (36.1)	9 (25)
<i>P</i>	0.06	0.03
Cerebral DCS ($n = 20$)	16 (80)	14 (70)
Matched control ($n = 20$)	5 (25)	3 (15)
<i>P</i>	0.012	0.002
Spinal DCS ($n = 17$)	6 (35.2)	5 (29.4)
Matched control ($n = 16$)	8 (50)	6 (37.5)
<i>P</i>	0.49	0.29

PFO, patent foramen ovale; *n*, no. of divers; *grade 2* PFO, important (≥ 20 bubbles) contrast passage at rest or after Valsalva strain; DCS, decompression sickness. Nos. in parentheses are %total. Divers with cerebral DCS had a significantly higher prevalence of PFO than did control divers without DCS. Prevalence of PFO in divers with spinal DCS is not significantly different from that in control population.

Table 2. Dive analysis

	Depth of Dive Leading to DCS, m	No. of Divers Using a Computer (mean depth, m)	No. of Divers Using a Table (mean depth, m)	No. of Divers Making "No-Fault" Dives	Time to Onset of Symptoms, min
Cerebral DCS ($n=20$)	35 ± 11	10 (37.4 ± 10.2)	10 (31.4 ± 10.1)	12	32 ± 48
Spinal DCS ($n=17$)	41 ± 8	6 (45.0 ± 8.8)	11 (39.0 ± 6.8)	14	79 ± 187
<i>P</i>	0.07	0.15	0.06	0.17	0.33

Values are means ± SD; n , no. of divers. Divers with spinal DCS tended to dive slightly deeper than divers with cerebral DCS. No. of "unexplained" DCS episodes tended to be slightly higher in divers with spinal DCS than in those with cerebral DNS. Time to onset of symptoms tended to be longer.

control divers. In contrast, for the subgroup of divers with spinal DCS, PFO prevalence was comparable to the prevalence in their control group. The difference between the two control groups (5 of 20 vs. 8 of 16 divers) was not significant ($P = 0.17$).

To exclude confounding factors that could possibly have predisposed for either cerebral or spinal DCS, an analysis was made of the dives leading to the DCS episodes (Table 2). No significant differences were found in dive depth, although cerebral DCS tended to occur at shallower depth. Of cerebral DCS cases, 12 of 20 could be classified as unexplained, as were 14 of 17 spinal DCS cases. There was no significant difference in the number of divers that used a computer to plan their dive. Computer-use divers did generally deeper dives than did dive table users, in both the cerebral DCS subgroup and the spinal DCS subgroup, but none of these differences were statistically significant.

With regard to biometric data (age, body mass index), smoking habits, and diving experience, no significant differences were found between spinal and cerebral DCS subgroups (Table 3). However, there was a striking difference in the method used for middle-ear equalization. Whereas in the spinal DCS subgroup, 8 of 17 divers indicated having spontaneous tubar permeability, i.e., no difficulties for equalizing middle-ear pressure (by yawning or only a very light Valsalva maneuver), all divers from the cerebral DCS subgroup reported they had to "push hard" or "push really hard" to "clear their ears" while diving ($P = 0.006$).

Finally, we looked at those divers with an unexplained DCS episode (26 of 37, 70.3%). Of the 12 cases from the cerebral DCS subgroup, 1 had *grade 1* PFO and 9 had *grade 2* PFO (10 of 12, 83%). In the spinal DCS subgroup (14 cases), 2 had *grade 1* PFO and 4 had *grade 2* PFO (6 of 14, 43%). For PFO in general (*grade 1 + grade 2*) these differences were almost significant

($P = 0.05$). For *grade 2* PFO, significance was reached ($P = 0.047$).

Divers with PFO had significantly more cerebral DCS (16 of 22 cases, 73%) than did divers without PFO (4 of 15, 26%) ($P = 0.0084$). With regard to *grade 2* PFO, this difference was still significant (14 of 19 vs. 6 of 18 cases, $P = 0.021$). The odds ratio could be determined as 7.33 and 5.6, respectively.

DISCUSSION

PFO is present in ~30% of the normal population; the prevalence seems to decline with ascending age groups (4). The anatomic details of PFO are well known: in most cases, it consists of a narrow (1- to 6-mm), rather long (7-mm) channel, transgressing the interatrial septum from upper right posterior to lower left anterior (2). Thus it forms a functional valve through which, in normal hemodynamic conditions, no significant blood shunt occurs because the right atrial pressure is lower than the left atrial pressure. In some patients, reversal of the pressure gradient may transiently occur in the cardiac cycle, but unless there is a very wide opening, no significant shunt occurs (10). If a left-to-right shunt is present, the term "PFO" is no longer appropriate, and the term "atrial septal defect" (ASD) should be used instead.

ASD, however mostly asymptomatic, is an accepted contraindication for sports diving, whereas PFO, usually undetectable on a clinical basis, is usually not considered a contraindication by medical specialists in diving.

During recent years, several clinical studies indicated the possibility of a higher prevalence of PFO (5, 14) in divers who suffered from DCS. These authors concluded that PFO can be the cause of DCS by paradoxical embolism of nitrogen bubbles that would

Table 3. Biometric analysis

	Age, yr	BMI, kg/m ²	No. of Smokers, >10 pack·yr	Diving Experience, yr	No. of Dives	No. of STP Cases
Cerebral DCS ($n=20$)	37 ± 9	26 ± 4	8	8 ± 6	327 ± 282	0
Spinal DCS ($n=17$)	38 ± 9	25 ± 3	4	12 ± 10	481 ± 465	8
<i>P</i>	0.59	0.36	0.32	0.16	0.25	0.006

Values are means ± SD; n , no. of divers. BMI, body mass index (wt/(ht)²); pack·yr, packs/day × yr of smoking; STP, spontaneous tubar permeability. There were no statistically significant differences in biometric data. All divers with cerebral DCS had to perform strenuous Valsalva maneuvers for middle-ear equalization, as opposed to only 9 of 17 divers with spinal DCS.

otherwise have been filtered out in the lung vasculature. PFO could thus be at the root of unexplained DCS (when no diving technical errors have been committed). This hypothesis has subsequently been challenged (3, 8).

The clinical pattern of PFO-related DCS has been inconclusively described. Symptoms usually appear very shortly (<30 min) after the dive (14). Although hemodynamic considerations would dictate the occurrence of embolization in the cerebral or high-spinal region, a higher prevalence of DCS with "cerebral" symptoms was not found in a recent study (13). This has led the author of the study to postulate that another mechanism, rather than paradoxical nitrogen bubble embolization per se, might be involved.

All these authors used contrast TTE to diagnose PFO. The superior sensitivity of TEE has clearly been demonstrated (7), but the more invasive nature of this method renders it more reluctantly accepted in a diverse population. With the use of TTE, several important PFOs may have been "missed." Another factor that may negatively affect PFO detection is the method used for contrast generation and the Valsalva technique utilized. Valsalva maneuvers are commonly used to enhance the sensitivity of contrast echocardiography. The goal is, by augmenting the intrathoracic pressure (ITP), to temporarily obstruct the venous inflow. On release of the ITP, the inflow of the pooled blood causes a significant rise in the right atrial pressure, which, in turn, may sweep saline bubbles to the juxtaseptal region and through a PFO. Ideally, contrast should be injected into a large lower extremity vein, owing to flow-pattern characteristics in the right atrium. It was frequently observed during this study that incorrect Valsalva maneuvers do not result in a complete opacification of the right atrium, especially in the juxtaseptal region.

We have previously demonstrated that the slope of ITP reduction on release is independent of the technique used (1). For the purpose of augmenting TEE sensitivity, the duration of ITP rise before release proves to be the most important factor. It is therefore unlikely that a simple cough could be more efficient than a Valsalva maneuver (9), unless the Valsalva maneuver was improperly performed or insufficiently sustained.

In our study, the prevalence of PFO in the control divers is higher than that reported in autopsy studies (2, 4). This may be an incidental finding, but whether divers have a comparable prevalence of PFO compared with the general population can be disputed. A conclusive study on the prevalence of PFO in the diving population has, to our knowledge, not yet been published. PFO is the result of an incomplete fusion of the two leaflets of the oval fossa after the reversal of the atrial pressures after birth. The declining prevalence of PFO in advancing age groups is probably due to the secondary adhesion and solidification of the two leaflets at a later age. However, the size of the shunts persistent in older age tends to increase (4). As already suggested by these authors, but still unconfirmed by a

prospective study, stretching of the valve of the fossa ovalis may occur by transient shunt reversals during normal life and accounts for this increase in size (4, 6). Many divers frequently induce sustained Valsalva maneuvers, the release of which has been shown to induce a major rise in right atrial pressure (1). The finding that virtually all divers in the group with "cerebral DCS," in which a large number of *grade 2* PFOs have been found, had to perform severely strained Valsalva maneuvers to equalize their middle-ear pressure while diving also points in this direction.

For the gradation of PFO, a semiquantitative approach was adopted, and the difference between slight and important contrast passage was arbitrarily chosen as 20 bubbles. Because it seems unlikely that very few microbubbles can provoke clinically overt DCS, this classification would be useful in distinguishing minor patency from possibly clinically important patency. Unlike divers with spinal DCS, divers with cerebral symptoms from DCS have a significantly higher PFO prevalence than do the control divers, and this difference is even more striking when only the *grade 2* PFOs are considered.

With regard to the DCS episodes unexplained by decompression errors or multifactorial enhanced DCS susceptibility, a higher prevalence of PFO was again found in the cerebral DCS subgroup compared with the spinal DCS subgroup. Here, only when the "important" PFOs (*grade 2*) are considered, the difference was significant.

Finally, there may seem to be an exceptionally high number of unexplained DCS episodes in our study population (26 of 37, 70.3%). Overall, the "deserved-to-undeserved" DCS ratio in a population of divers treated for DCS lies ~50%. However, for the purpose of this study, only definite DCS episodes with well-reported dive profiles were included. This has led to the exclusion of a large number of "doubtful DCS cases" from the study. A great number of these "perhaps DCS cases" were actually treated for DCS, mainly because of the often serious technical errors committed by the diver rather than because of the certainty of the clinical diagnosis.

In conclusion, we found a significant correlation between the prevalence of PFO and the occurrence of cerebral, but not spinal, DCS. This is in accordance with the pathophysiological model, in which nitrogen bubbles, passing through the PFO into the arterial circulation, migrate preferably into the carotid and/or vertebral arteries.

Because all known possibly confounding factors either have been matched for or have no detected significant difference, these findings support the hypothesis that PFO is a cause of DCS with cerebral localization.

We would therefore recommend that divers with unexplained DCS and symptoms suggesting a cerebral or high-spinal localization of the lesion be investigated for the presence of PFO. If a *grade 2* PFO is present, paradoxical nitrogen bubble embolization should be considered likely, and we would advise the diver to

follow dive profiles that are very low "bubble-prone" in the future, or to give up diving.

Furthermore, for future PFO studies, we strongly recommend the following: 1) use of a standardized contrast TEE technique, with special attention to the strain and duration of the Valsalva maneuver; 2) semiquantification of the permeability of the foramen ovale; and 3) use of matched divers as control subjects.

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