Does persistent (patent) foramen ovale closure reduce the risk of recurrent decompression sickness in scuba divers?

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Abstract
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Introduction: Interatrial communication is associated with an increased risk of decompression sickness (DCS) in scuba diving. It has been proposed that there would be a decreased risk of DCS after closure of the interatrial communication, i.e., persistent (patent) foramen ovale (PFO). However, the clinical evidence supporting this is limited.
Methods: Medical records were reviewed to identify Swedish scuba divers with a history of DCS and catheter closure of an interatrial communication. Thereafter, phone interviews were conducted with questions regarding diving and DCS. All Swedish divers who had had catheter-based PFO-closure because of DCS were followed up, assessing post-closure diving habits and recurrent DCS.
Results: Nine divers, all with a PFO, were included. Eight were diving post-closure. These divers had performed 6,835 dives (median 410, range 140–2,200) before closure, and 4,708 dives (median 413, range 11–2,000) after closure. Seven cases with mild and 10 with serious DCS symptoms were reported before the PFO closure. One diver with a small residual shunt suffered serious DCS post-closure; however, that dive was performed with a provocative diving profile.
Conclusion: Divers with PFO and DCS continue to dive after PFO closure and this seems to be fairly safe. Our study suggests a conservative diving profile when there is a residual shunt after PFO closure, to prevent recurrent DCS events.

Introduction

Divers use different mixtures of breathing gas depending on the depth and duration of the activity. The most common are: air, with approximately 78% nitrogen and 21% oxygen; nitrox which is oxygen-enriched air; and trimix, which is a mix of oxygen, nitrogen and helium (gas mixtures containing helium are used at greater depths). Nitrogen and helium are inert gases not involved in physiological processes, and when breathing compressed gas underwater, the inert gas dissolves at higher partial pressure in the tissues and blood vessels.[1] With reduction in ambient pressure gas can come out of solution causing bubble formation in the blood and extravascular tissues, and this can result in decompression sickness (DCS).[2,3] The underlying causes of DCS symptoms are principally local effects and pressure exerted by the bubbles, manifested for instance as cutaneous itching, marbled skin and joint pain, complex biochemical reactions in the brain and the spinal cord affecting neurological function.[2–5] The optimal treatment for DCS is 100% oxygen therapy as soon as possible, in combination with intravenous fluids and hyperbaric oxygen treatment (HBOT) in a recompression chamber.[6]

By making stops at certain depths determined by a decompression table or dive computer during the ascent from great depths or after a long dive duration, divers try to minimise the risk of DCS. However, even when diving in line with recommendations for safe diving profiles with decompression stops, studies have shown bubble formation in the venous circulation.[7,8] Normally, these bubbles are filtered and exhaled by the lungs without causing DCS.[3] Both atrial septal defects[9] and persistent (patent) foramen ovale (PFO)[1,10–12] have been associated with an increased risk of DCS due to a right-to-left shunt of venous decompression bubbles into the arterial circulation. Moreover, it has been suggested that divers with PFO are more likely to suffer severe neurological forms of DCS and require longer

treatment with hyperbaric oxygen therapy (HBOT).[12] The Valsalva manoeuvre used to equalise the middle ear pressure, and resistance lifting of heavy diving equipment, have been proposed to cause an increased pressure in the right atrium, which can facilitate the shunting of bubbles.[13]

There are a limited number of studies on diving habits and DCS incidence following PFO closure,[14-16] and such a study had not been performed in Sweden previously. The main aims were to investigate whether patients who had suffered DCS events that led to catheter-based closure of a PFO continued to dive after the closure and if there were any DCS events after the closure.

Methods

The study was approved by the local ethical committee (Dnr 2017/572). All participants were informed about the study in writing before being interviewed, and signed a written informed consent form. The study was registered at clinicaltrials.gov, NCT03997084.

All five centres performing PFO closure in Sweden were asked to participate in the study, however one of them was not able to provide data. All patients who had had a catheter-based closure of PFO or an atrial septal defect (ASD) following DCS at these four centres were identified in SWEDCON, a national registry on congenital heart disease also covering catheter interventions including PFO. In accordance with international consensus,[17] DCS symptoms considered 'mild' were musculoskeletal pain, patchy non-dermatomal paraesthesiae, rash, lymphatic swelling, and constitutional symptoms such as fatigue. 'Serious' symptoms were objective neurological deficits and cardiopulmonary symptoms. Based on information in medical charts, patients who had had the closure because of DCS were selected according to the following criteria: at least one DCS event that led to investigation for a PFO or ASD; PFO or ASD verified with contrast echocardiography; and a completed catheter-based intervention to close the PFO or ASD with the indication being DCS prevention.

A letter with information on the purpose of the study and a consent form was sent to the potential subjects. Subsequently, a phone interview was conducted. The interview was based on a questionnaire with eight main questions focusing on the dive habits and DCS event(s):

- Type of diving certificate: for recreational diving or for professional diving;
- The total number of dives before closure separated into the breathing gas used: compressed air, nitrox or trimix;
- The date of the DCS event/events prior to the closure, together with additional facts regarding each event: breathing gas used, depth, if oxygen therapy was used, if recompression therapy was used, remaining symptoms after the therapy, and symptoms of DCS. The symptoms were divided into mild and serious as described above.
- The date and location of catheter-based closure.
- The total number of dives after closure categorised by the breathing gas used, and the maximum dive depth post-closure;
- The date of DCS events after closure, together with the additional facts stated in question 3;
- The number of dives performed during the last year.
- Optional comments from the participants regarding their diving history.

Data about the treatment and DCS symptoms were collected at each centre in medical records. All DCS cases were diagnosed medically. None of the subjects had any evidence of barotrauma. Information about the result of the closure together with data about the dives that lead to DCS events, including breathing gas and diving depth were compiled. The SWEDCON registry and medical records provided information about the date of closure, the patient's height and weight at the time of intervention, the size of the defect measured with a sizing balloon, the type of closure device, complications post-intervention and the result of the closure. The result was measured by the number of agitated NaCl contrast bubbles found in the left heart, when provoked with Valsalva manoeuvre that was visible on echocardiography 24 hours and one year after the closure. Results were categorized as either no detected bubbles, 1–10 bubbles, more than 10 bubbles or an incalculable amount at each of the follow-ups respectively.

There were no patients with ASD that met the criteria after the medical record review. Hence, the following data pertain only to PFO patients.

Table 1

Defect size (mean SD) and closing device. Echo result after closure based on the number of agitated saline contrast bubbles found in the left heart when provoked with a Valsalva manoeuvre. * One patient did not have a follow-up after one year. In that case no residual shunt was detected with echocardiography after 24 h and after seven days.

Parameter	
Initial defect size (mm)	7 (SD 3)
Complications after closure	Nil
Closing device (*n*)	
Amplatzer PFO Occluder 25 mm	6
Gore Septal Occluder 25 mm	2
Noble Stitch	1
Echo at 24 h (*n*)	
1–10 bubbles	3
No residual shunt	6
Echo at one year*(*n*)	
1–10 bubbles	2
No residual shunt	6

Table 2
The total number of dives and median number of dives, separated by the breathing gas used, performed by the nine divers before closure, and by the eight divers that dived post-closure

Period	Total, median (range)	Compressed air *n* (%)	Nitrox *n* (%)	Trimix *n* (%)
Dives before closure	6,835, 410 (140–2,200)	4,950 (72)	1,320 (19)	565 (8)
Dives after closure	4,708, 413 (11–2,000)	2,639 (56)	1,507 (32)	562 (12)
Dives in the last year	539, 19 (0–250)	269 (50)	250 (46)	20 (4)

Table 3
The number of decompression sickness (DCS) events and depths of incident dives before and after closure, separated by the breathing gas used. * = Median (range). ** = Range. Six of the seven incident dives during use of trimix were performed by the same diver, and depth data could only be provided for one of these six dives (76 m)

Parameter	Total	Air	Nitrox	Trimix
DCS before closure	17 1 (1–6)*	9 (53%)	1 (6%)	7 (41%)
DCS after closure	1	0	1	0
Depth (m) of incident dive before closure		31 (15–49)*	36	73–76 **
Depth (m) of incident dive after closure		–	19	–

Results

From 1997 up until the end of 2017, a total of 603 PFO were closed; 13 of these were performed because of previous DCS. Out of these 13 PFO patients, four chose not to participate in the phone interviews, leaving nine persons who agreed to participate. Five of them were professional divers and four were recreational divers (one female). The subjects had a mean age of 29 (SD 4) years and a mean BMI 26 (3) $kg \cdot m^{-2}$ at the time of PFO closure. A sizing balloon was positioned in the PFO in all cases to measure the stretched diameter of the defect. The PFO defects were mean 7 (SD 3, range 4–10) mm (Table 1).

There were no complications associated with the closure. At one-year follow-up six patients demonstrated complete closure while two patients had a residual shunt of 1–10 bubbles (Table 1). The median time after closure at which interview for this study was conducted was seven years (range 2–18). A total of 6,835 dives were performed before PFO closure (median per subject 410, range 140–2,200). One diver did not dive after the closing procedure. The other eight divers performed a total of 4,708 dives after closure (median 413, range 11–2,000) ranging in depth from 20–100 metres (m) (Table 2). One stopped diving six years after the PFO closure, and one diver stopped after another incident of DCS. Six divers were still diving at the time of the interview.

In total, 17 DCS events (seven mild, ten serious) were reported before closure (median 1, range 1–6) (Table 3). Oxygen therapy was used in 15 out of 18 events, the one post closure DCS included. HBOT was used in 10/18 cases. The reported diving depths for the dives that caused DCS varied between 15 and 76 m and are summarised in Table 3. One DCS event was reported after the closure procedure (Table 3). The affected diver was the only female included in the study. She was one of two patients who had a residual shunt one year after closure (Table 1). She performed 300 dives with compressed air before the closure, and had suffered three DCS events. After the intervention, she performed nine dives with compressed air and two with nitrox. The dive that caused DCS was performed with nitrox to 19 msw. She experienced a serious DCS after the PFO closure but was successfully treated with hyperbaric oxygen with complete symptomatic relief. However, the incident dive was performed with a provocative diving profile that potentially could increase the risk of DCS. It is notable that the information about the provocative diving profile was added by the diver voluntary at the end of the interview, and not as an answer to our predetermined questions. The second diver who had a residual shunt one year after closure did not suffer DCS in 125 dives post-closure.

Discussion

Divers with PFO and previous DCS events are currently recommended to dive more conservatively to reduce the risk of recurrent DCS.[18] PFO screening is generally recommended when DCS occurs after a non-provocative dive, after neurological or repetitive DCS events.[19] Several authors have suggested that PFO closure would abolish the increased risk of DCS events associated with PFO.[15,16,20–22]

This proposal is supported by the finding that chamber dives producing venous gas emboli in the majority of subjects resulted in arterial bubbles in some divers with PFO but in no divers with a catheter-based PFO closure.[23] However, 'dry' divers have been shown to produce fewer venous gas emboli than submerged dives.[19]

To our knowledge there are five studies of DCS outcomes after PFO closure. In two studies,[14,24] no episodes of major DCS were reported after PFO closure in 11 and 20 divers respectively. Another showed a decreased DCS incidence after PFO/ASD closure.[16] In the study by Billinger et al., there was one case of serious DCS after PFO closure and this occurred in the only diver out of 26 who had a residual shunt.[20] In the latest study by Honek et al. PFO closure was shown to prevent DCS.[25] In the present study one subject out of nine experienced a DCS event after the closing procedure but this subject undertook a provocative diving profile and had a residual shunt. A possible conclusion from this is the importance of the follow-up echo examination after the intervention. If a residual shunt is detected, we suggest it would be wise to recommend conservative diving profiles.

Estimates of the DCS risk per dive is 0.095% for commercial divers and 0.01–0.019% for recreational divers.[4] It has previously been proposed by two studies that PFO increases the DCS risk 2.5–5 times.[10,26] Based on the DCS risks described above, 0.01-0.095%, combined with these increased risk estimates, the expected number of DCS events pre closure in our cohort (6,835 dives), would be 2–32 events, we report 17. If the DCS risk post closure (4,708 dives) is estimated on the numbers above but without the increased risks associated with PFO 0.5–4 events would have been expected, we report one. Hence, our results correspond to the risks previously described in the literature.

In the questionnaire, we chose to include maximum diving depth after the closure. AGE can occur even after ascent from shallow diving.[4] Results from a study where divers performed saturation dives to certain depths and then ascended without decompression stops, indicate that DCS is uncommon at depths shallower than 6–9 metres.[27] All participants in our study, except one that had not performed any dives after the closure, had been diving deeper than 20 metres after the closure. Thereby, they had exposed themselves to conditions that theoretically could cause both DCS and AGE. This exposure to dives carrying a risk of DCS strengthens our conclusion that PFO-closure protects against recurrence. Among this small group of Swedish divers a large majority continued to dive after the PFO closure. This is important because if the divers would not attempt to dive again due to fear of recurrent DCS, the benefit of the intervention would have been called into question.

In deciding whether to undertake PFO closure after DCS the risks of PFO closure must be taken into account. In a study including 825 patients, overall device implantation failed in 0.2% of the interventions. Complication rate was 2.2% and most common were embolisation of the device in 0.6% of the cases.[28] The PFO closing procedure seems reasonably safe and feasible, but the risks should still be considered before recommending the intervention.

Our study cohort was small, and the number of dives performed made calculations of DCS risk precarious. However, there are no very large studies so the combined findings of these studies, including ours, could be of importance in the future recommendations regarding diving for patients with a closed PFO. Since retrospective reviews were conducted on a large number of medical charts, another potential weakness is that we cannot be entirely sure that all relevant patients were identified. There is a potential selection bias among the included patients. One could speculate that some divers, especially recreational divers, with a PFO who suffer DCS prefer to stop diving rather than have the PFO closed. We have not focused on the diving profiles in this study, because we did not do a logbook review and it is possible that divers dived more conservatively after PFO closure. A logbook review would have been time-consuming and potentially unfeasible in a retrospective study like this. In a future study it would be ideal to only include dives where the diver had strictly followed decompression tables.

Conclusions

Divers who suffer certain forms of DCS may be recommended to undergo investigation for a PFO and, if a large PFO is discovered, to close it to reduce the risk of recurrent DCS before resuming diving. After PFO closure it is important to check for residual shunting as this may be associated with a persistent increased risk of DCS. If there is a residual shunt and the diver wishes to continue diving, conservative profiles are recommended. These results suggest that divers with PFO who have experienced DCS and undergone PFO closure, don't need to cease diving after the intervention.

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