IS SCREENING FOR PATENT FORAMEN OVALE FEASIBLE?

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Key Words

Cardiovascular, decompression illness, equipment, medical conditions and problems, medicals, risk, safety.

Introduction

More than 50 % of decompression illness (DCI) are unexpected, which means after regular dives without incidents or rapid pressure changes. As bubbles arise from the veins after most of the dives, cerebral embolism from bubbles by-passing the lungs, for instance by shunts through a patent foramen ovale (PFO), has been discussed. There are however other shunts possible and cerebral arterial gas embolism (CAGE) arising from air trapping in divers with lung pathology has also been proposed.[1] In a recent study Wilmshurst demonstrated that only a negligible part of unexpected DCI cases could not be “explained” by either a PFO or a lung pathology.[2]

Rationale

Bove, in a retrospective study, and Germonpré, in a case control study, recently determined the increase in probability for PFO related DCI as, respectively, 2.5 and 3.7 times non-PFO probability.[3,4] As almost 30% of all divers could have a patent foramen ovale, the non-diving population prevalence, one can wonder why DCI is not much more frequent than it is. Balestra demonstrated that, even with significant shunts, there is a need for an increased right-left atrial pressure gradient for bubbles to pass through shunts.[5] These gradients can be caused by intra thoracic pressure (ITP) rises. The investigators measured ITP by inserting a balloon tube into the oesophagus, filling the system with water and measuring the pressure in cm of water above the pressure during normal respiration. One of the most effective methods of raising ITP is a prolonged, forced Valsalva manoeuvre, but larger rises in ITP are produced by knee bends while performing a Valsalva as well as coughing and pressing down as hard as possible while holding one’s breath. Figure 1 (page 86) compares the intra thoracic pressures reached.

The test manoeuvres charted in Figure 1 were:

Control

Maximal isometric arm and chest muscles exercises: while sitting in a standard position (with knees and hips in 90° flexion) with the hands one above the other and the arms extended forward horizontally, the subject had to push down as hard as possible on a set of scales , placed on the ground, by means of a wooden stick while holding his or her breath. This test was performed three times; the mean push-down force was noted, and the mean ITP reached was used as the control ITP value for the other tested manoeuvres.

Gentle Valsalva

Valsalva manoeuvre (as usually performed by the diver to equalise middle ear pressure).

Forced Valsalva

Valsalva manoeuvre with the subject blowing as hard as possible.

Calibrated Valsalva

Valsalva manoeuvre gradually increasing ITP until the ITP reached the level of the first maximal isometric exercise.

Cough

Forceful coughing.

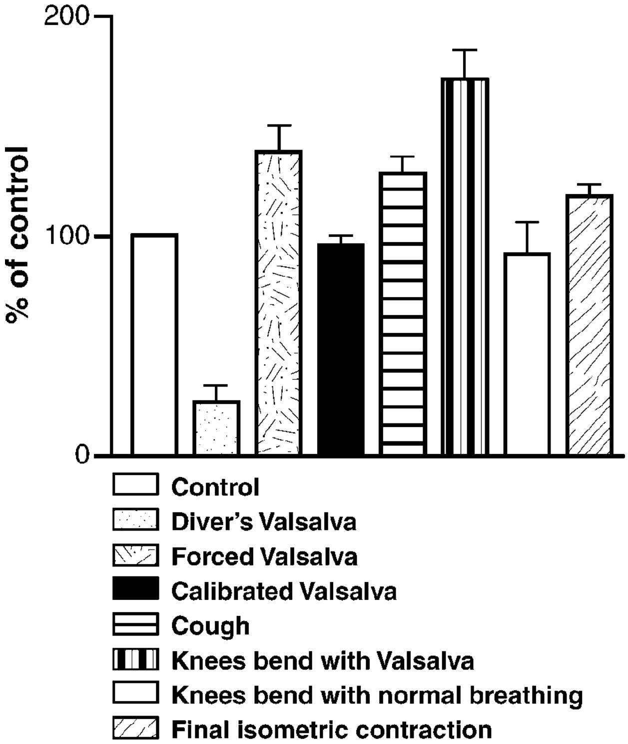

Figure 1. Intrathoracic pressure increase produced by different manoeuvres (from[5]).

Knees bend with Valsalva

Knees bend (squatting) with the subject performing a forced Valsalva.

Knees bend with normal breathing

Knees bend while the subject breathed normally

Final isometric contraction

A repeat of procedure 1 after the subject had completed tests 2-7. Care was taken to ensure that the same push-down force was reached.

Even with large PFOs (type II), the length of the ITP rise is of great importance in producing a considerable shunting volume in the pressure release phase, probably due to flow pattern characteristics in the right atrium.[6] Surprisingly, the normal diver's ear clearing manoeuvre produces almost no ITP increase. Figure 2 shows that a diver's ear clearing Valsalva results in an ITP rise of 5 mmHg while a forced Valsalva reaches about 105 mmHg! Small PFOs and an insufficient bubble load result in negligible shunting. Some pressure inversion between the atria is probably present in all respiratory patterns as shown in Figure 3,[7] but may be insufficient to produce large bubble transmission because of their short duration. Besides the ITP peaks during eventual post-dive exertion, such as raising the anchor or lifting cylinders out of a boat, there is a gradual rise in mean pulmonary artery pressure (MPAP) after decompression dives, induced by bubble embolisation of the lung vasculature.[8] This retrograde increase of the right atrial pressure might open a PFO, allowing bubble shunting, if sufficient bubbles are present in the veins.

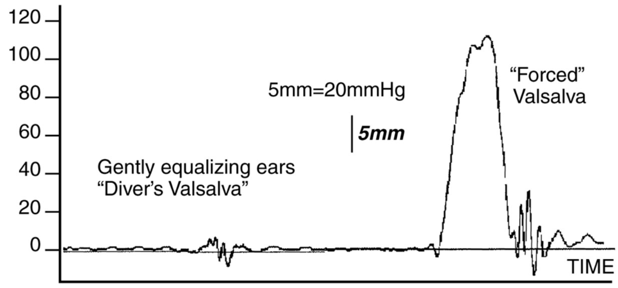

Figure 2. Intrathoracic pressure registration during normal pressure equilibration and forced valsalva manoeuvre (from[5])

The exact risk of diving with a PFO can only be determined by means of a prospective study. As DCI is a very rare event, a study with a large number of monitored divers is necessary to determine the risk increase. Such a study can only be conducted if an appropriate "screening" method for PFO could be designed. Then screening of divers could well be justified if the procedure was simple and there was no risk of unwanted consequences.

Feasibility

Beside the heavily invasive procedures like blood gas analysis and Swan Ganz catheter studies contrast-transoesophageal echo sonography (C-TEE) is recognised as the gold standard for PFO detection. Transthoracic echo (TTE) and transcranial doppler (TCD) examinations are not sensitive enough for screening purposes. Table 1 compares the sensitivity and specificity obtained with different techniques from,[6] after Di Tullio.[9]

As TEE is invasive, expensive and restricted to experienced cardiologists, we have proposed a screening technique using a much simpler Doppler bubble detector and monitoring the carotid artery.[10] As at the time no data were available, Germonpré and Balestra evaluated this technique in a prospective randomised study. They found that with their standardised procedure, described below, the sensitivity was as high as 100 % and specificity 88% when compared with TEE.[6] The intravenous injection of micro-bubbles as described here is a safe procedure and it is routinely used in TEE as a common cardiological practice.[11] However, for safety reasons, oxygen should be available and the diver should not have dived within the last 24 hours. The procedure is described below:

Standardised technique for carotid artery Doppler screening for PFO

1 Insertion of intravenous cannula to the right ante-cubital vein.

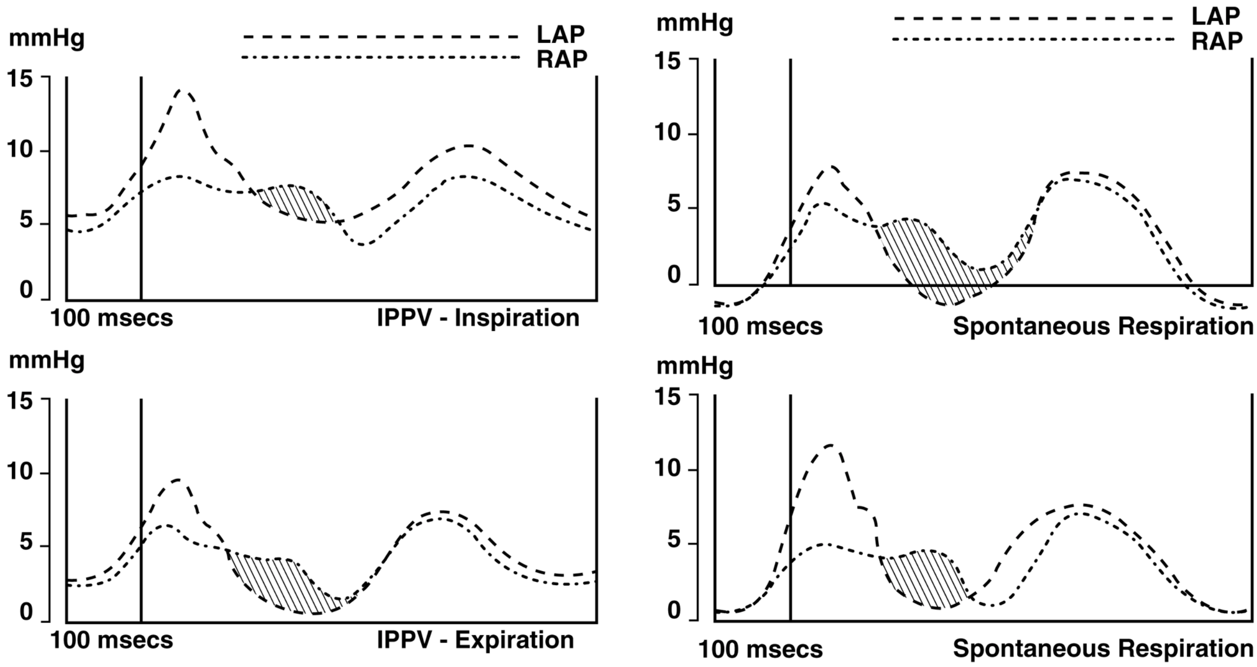

Figure 3. Left (LAP) and right (RAP) atrial pressure tracings after cardiac surgery during controlled ventilation (IPPV) (3a) and spontaneous respiration (3b); from Cambier.[7]

TABLE 1

CONTRAST ECHO-DOPPLER TECHNIQUES

Method	Abbreviation	Sensitivity	Specificicity
Contrast-transoesophageal echosonography	C-TEE	100%	100%
Contrast-transcranial Doppler	C-TCD	68%	100%
Contrast-transthoracic echosonography	C-TTE	47%	100%
Contrast Carotid artery Doppler	C-CD	100%	88%

2 Connect to a saline infusion and prepare 2 three-way taps and two syringes of 10 ml (Fig 4)

3 Fill syringe 1 with 9.5 ml normal saline + 0.5 ml air.

4 Mix by pushing the content from syringe 1 to syringe 2 and back 10 times (produces a micro-bubbles solution).

5 Inject immediately thereafter the whole content within 3 sec and continuously monitor the Doppler signal from the left carotid artery. If bubbles have been shunted through a PFO they will be detectable within 3 seconds after injection (bubbles appearing later than that will still have been shunted but through the lungs or elsewhere). During this injection, the patient should breathe normally.

6 Repeat steps 3-4 after 1 minute.

7 Perform a 10 second forced straining manoeuvre (take a deep breath, hold that breath and "push down" in the abdomen for approximately 10 sec). Inject another syringe of 10 ml with bubble mix, and instruct the patient to suddenly release the straining manoeuvre when the whole syringe is injected. Continuously monitor for bubble sounds at the carotid artery.

8 Repeat steps 6-7 twice.

The signal should be recorded on tape for later study.

Figure 4. Injection device in situ, with two three-way taps and an intravenous catheter.

Consequences of a positive screening test

At present there is no generally approved consensus about the consequences of a positive PFO finding. Some diving medicine specialists would categorically declare anyone with a known PFO unfit, however the majority would not ban further diving. Professional divers are generally (HSE in England and others) declared unfit after DCI with neurological symptoms. Recreational divers rely on the notion of "acceptable risk". From a pathophysiological point of view, the risk of diving with a PFO must be higher than without PFO. However, since the actual cause of DCI is the gas bubbles in the blood, this risk can probably be reduced to within acceptable levels by any diving procedure that reduces the bubble load after the dive. A few possibilities are:

1 Use of dive tables calculated to reduce the supersaturation threshold to 1.4 (or 1.2 or even 1.0), which results in low-bubble-diving (almost zero bubbles). A similar effect can be obtained by setting the dive computer mode to altitude. The exact effect on threshold and DCI probability still remains to be determined.

2 Breathing nitrox while diving using air tables or a computer set for air diving, which will reduce decompression stress proportionally.

3. Avoid ITP peaks, that is no effort after exiting the water, use the Frenzel manoeuvre instead of the forced Valsalva for clearing the ears (Fig.5).

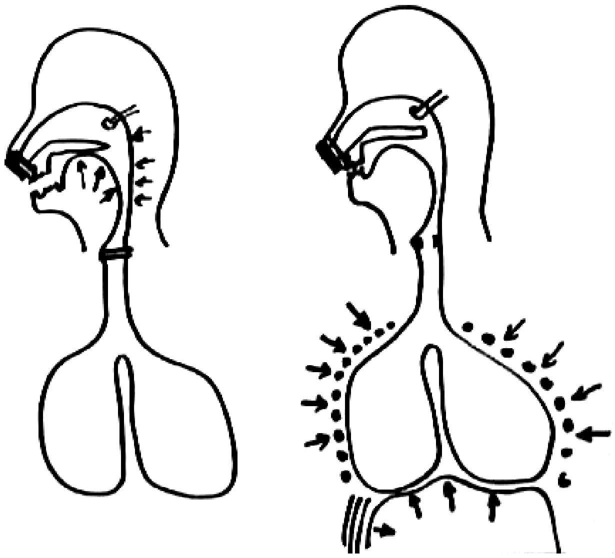

Figure 5. Two different methods for pressure equilibration in diving: the Frenzel manoeuvre (left) is performed by swallowing when the glottis is closed. The ITP remains normal. Performing a forced Valsalva (right) the thoracic and abdominal muscles produce a rise in ITP which is transferred to the ear through the open glottis and throat.

Conclusions

1 Carotid artery Doppler examination with saline bubbles injection and a standardised straining manoeuvre is a cheap, easily learned, minimally invasive and not painful screening method with very good sensitivity and specificity.

2 In victims of DCI, detection of PFO should be performed by a c-TEE, following a standardised technique.[5] For screening purposes, the Carotid artery Doppler examination provides good sensitivity and specificity.

3 PFO positive victims of DCI must be advised how to reduce the risk for decompression disorders by changing their diving practices (reduce DCI probability, special tables, altitude mode in computers) and avoiding elevation of ITP and changing their pressure equilibration practice (Frenzel manoeuvre). However, divers should stop diving if neurological DCI symptoms are due to other causes including lung disease.

4 Screening of divers should be done only in the context of a prospective longitudinal study. Data from such a study will contribute to the quality of scientific discussions about PFO. In the end we will have more evidence-based diving medicine and understanding of the pathogenesis of unexpected decompression illness.

5 A prospective study has been initiated by DAN Europe Research Division, aiming at screening 4,000 divers, with a follow up period of 5 years. Participation is encouraged (see <www.daneurope.org/research/carotid.htm>

References

1 Wilmshurst P, Davidson C, O'Connell G and Byrne C. Role of cardiorespiratory abnormalities, smoking and dive characteristics in the manifestations of neurological decompression illness. *Clin Science* 1994; 86: 297-303

2 Wilmshurst P. Cardio-vascular assessment. *Proc. "Medical Assessment of Fitness to Dive"* seminar . London, 8-9/ 4/ 2000 (study with Bryson PH at DDRC, in press)

3 Bove AA. Risk of decompression sickness with patent foramen ovale. *Undersea Hyper Med* 1998; 25 (3): 175-178

4 Germonpré P, Dendale P, Unger PH, Balestra C. Patent Foramen Ovale and decompression sickness in sports divers. *J. Appl Physiol*. 1998; 84: 1622-1626

5 Balestra C, Germonpré P and Marroni A. Intrathoracic pressure changes after Valsalva strain and other maneuvers: implications for divers with patent foramen ovale. *Undersea Hyper Med* 1998; 25 (3): 171-174

6 Germonpré P, Balestra C, Dendale P, Unger P and

Vandenhoven G. Carotid Artery Doppler as a minimally invasive screening method for patency of the Foramen Ovale. *Proc. EUBS meeting* 1999 (26-29 August) Eilat Israel

7 Cambier BA, Missault LH, Kockx MM, Vanddenbogaerde JF, Alexander JPE, Taeymans YM, Vancauwelaert PA and Brutsaert DL. Influence of the breathing mode on the time course and amplitude of the cyclic inter-atrial pressure reversal in postoperative coronary bypass patient. *Eur. Heart J* 1993; 14: 920-924

8 Vik A, Slerdahl SA, Brubakk AO. Paradoxical air embolism in pigs with a patent foramen ovale. *Undersea Biomed Res* 1992; 18: 361-374

9 Di Tullio M, Sacco RL, Venkatasubramanian N, Sherman, D, Mohr JP, Homma S. Comparison of diagnostic techniques for the detection of a patent foramen ovale in stroke patients. *Stroke* 1993,; 24 (7): 1020-1024

10 Wendling J, Wenzel J and Müller PHJ. Die Doppleruntersuchung in der Risikoabklärung für Gerätetaucher. *Caisson* 1998; 13 (2/3): 76-83 ISSN 0933-3991

11 Siostrzonek P, Zangeneh M, Grossinger H, Lang W, Rosenmayr G, Heinz G, Stümpflen A, Zeiler K, Schwarz M and Mösslacher H. Comparison of transesophageal and transthoracic contrast echocardiography for detection of a patent foramen ovale. *Am J Cardiol* 1991; 68: 1247-1249

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