Short communication

The impact of performing spirometry on shunting across a patent foramen ovale

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Key words
Patent foramen ovale (PFO), echocardiography, scuba diving, decompression illness, pulmonary function

Abstract

(Maddox IEC, Smart DR, Bishop WLJ. The impact of performing spirometry on shunting across a patent foramen oval. *Diving and Hyperbaric Medicine*. 2009;39(4):213-5.)
Transient changes in intrathoracic pressure can alter left and right intra-atrial pressures, and may provoke shunting of blood across a patent foramen ovale (PFO). Spirometry causes a transient rise and subsequent fall in intrathoracic pressure that, if performed following a dive on compressed air, could raise the risk of decompression illness by arterialisation of venous bubbles across a PFO. To assess whether spirometry can provoke right-to-left shunting across a patent foramen ovale, a subject with a known PFO, previously identified by bubble contrast transthoracic echocardiography, where shunting was only evident on performing a Valsalva manoeuvre, underwent re-examination whilst performing spirometry. Right-to-left shunting was not evident at rest, but was provoked by performing spirometry. If spirometry is to be performed within two hours of surfacing, this should be regarded as a potential risk for decompression illness.

Introduction

The termination of manoeuvres that transiently raise intrathoracic pressure is recognised to cause a rise in right atrial pressure relative to left atrial pressure.[1,2] This fact is utilised in the echocardiographic diagnosis of a patent foramen ovale (PFO), where bubble contrast may be seen to cross from right to left atrium following such a manoeuvre.[1–4]

An ongoing study investigating lung function pre- and post-recreational scuba diving requires volunteers to perform spirometry before a dive and in the period following surfacing.[5] Venous bubbles commonly form following recreational scuba dives on air.[6] It is possible that increased right atrial pressure relative to the left atrium may occur following spirometry. In the setting of venous bubbles, this may provoke their arterialisation across an undiagnosed PFO, increasing the risk of decompression illness (DCI). This possibility was investigated in a subject with a known PFO.

Methods

The subject (author IM, who gave informed consent) was known to have a PFO, previously identified by transthoracic bubble contrast echocardiography, where right-to-left shunting was evident only following a Valsalva manoeuvre. Re-examination by transthoracic bubble contrast echocardiogram was undertaken by the same operator (author WB). The subject was examined in the left lateral position. Bubble contrast medium was prepared by agitating 9 ml of saline with air by repeated injection and aspiration from the ampoule. One ml of the subject's blood was added to this agitated saline and visible gross air bubbles expelled from the syringe. Once good apical, four-chamber views were obtained, this agitated saline/blood mix was injected via an 18-gauge cannula into a right antecubital vein, and the echocardiogram scrutinised in real time for the appearance of bubble contrast in the chambers of the heart. The echocardiogram images were also recorded electronically. Following an interval of about five minutes, the subject performed spirometry using a Spirolab II portable spirometer, whilst remaining in the left lateral position with the four chamber views maintained. A single vital capacity inspiration was followed immediately by forced expiration through the spirometer. Agitated saline with blood was injected intravenously as before at three seconds after the onset of the forced exhalation, and the echocardiogram images viewed and recorded. After a further interval, spirometry, injection of agitated saline with blood and echocardiogram examination were repeated in the same manner. In both instances, the duration of forced expiration was almost exactly four seconds.

Results

With the subject at rest, and no airway or thoracic manoeuvres being undertaken, no bubbles were detected in the left atrium or left ventricle during the seven monitored cardiac cycles following detection of bubbles in the right atrium. No right-to-left shunt was demonstrated (Figure 1).

Figure 1
Bubble contrast is seen entering the right atrium (RA) and right ventricle (RV), but none in the left heart chambers; no thoracic or airway manoeuvres have been performed

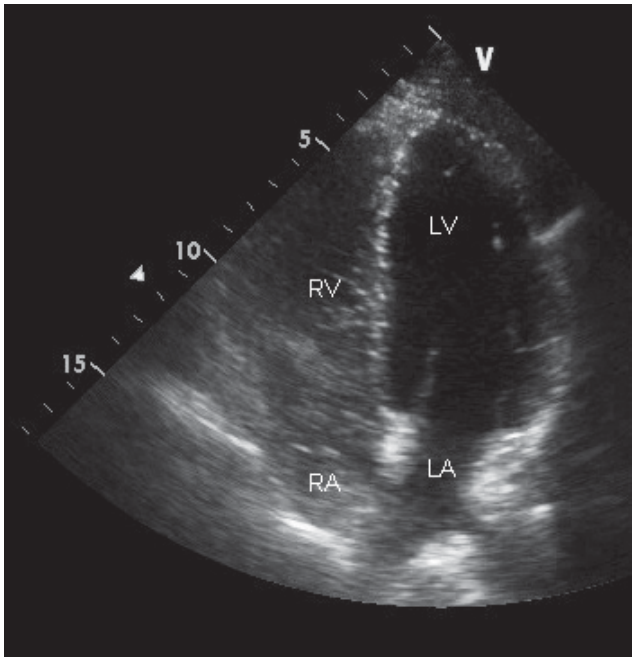
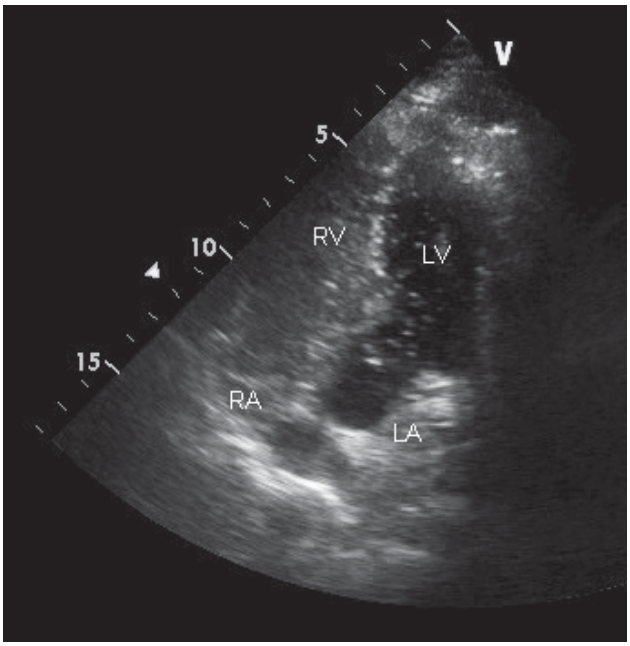


Figure 2
Following spirometry, bubble contrast is seen in the left ventricle during atrial systole, within two cardiac cycles of its appearance in the right atrium



After performing spirometry, followed by inspiration, bubbles were visible in the left atrium and ventricle within two cardiac cycles of their detection in the right atrium. Repeat spirometry again demonstrated bubbles in the left atrium and ventricle within two cardiac cycles of their detection in the right atrium (Figure 2). No further study was performed as, although published data suggests bubble contrast studies are very safe, repeated injection could risk significant paradoxical air embolism in the subject.[7,8] The appearance of bubbles in the left atrium and left ventricle within three cardiac cycles of their appearance in the right atrium suggest shunting at the atrial level rather than at the transpulmonary level.[4]

Discussion

Patent foramen ovale is common and usually goes undiagnosed.[9] There is a recognised association of PFO with decompression illness.[1,3,4,9–12] In one study amongst a group of scuba divers, there was a prevalence of 22% of PFO of a physiological size that, according to the research, significantly increased the risk of major DCI.[3]

PFO-related DCI is presumed to be caused by paradoxical nitrogen bubble embolisation through the interatrial septum.[11] Nitrogen washout models support the hypothesis that it is subsequent growth of these arterial bubbles due to tissue gas supersaturation that is the cause of the link between the PFO and inner ear DCI.[12] These models also circumstantially support the suggestion that tissue supersaturation is relevant to other organ systems whose vulnerability to DCI is associated with right-to-left shunts.[12] Under normal physiological conditions, passage of blood and bubbles from right to left atrium is limited. The PFO may be a valve-like structure, which is closed during 95% of the cardiac cycle due to higher pressures in the left atrium.[11]

In using a bubble-contrast echocardiogram to investigate the presence of a PFO, the aim is to induce a right-to-left shunt. Some authors have recommended this be avoided shortly after any DCI as there is the potential to exacerbate neurological injury. It has been suggested that activities occurring during or soon after diving that cause a transient rise with subsequent release of pressure, such as manoeuvres to clear ears, straining to lift dive equipment or climbing onto a boat, may provoke right-to-left shunting of bubbles across a PFO and increase the risk of DCI.[1,10] It should be emphasised that it is the release of the raised intrathoracic pressure that causes the significant increase in right atrial pressure compared to the left.[2]

The demonstration in a single subject of the provocation of a right-to-left shunt by performance of spirometry does not fully mimic the conditions that occur when spirometry is performed after diving. Our subject lay in the left lateral position as opposed to standing upright. This may facilitate right-to-left shunting by increasing flow from the lower body into the right atrium at the release of the raised intrathoracic

pressure. Whilst sub-xiphisternal views with the subject semi-reclining may have mimicked more closely the real-life situation, we were concerned that, in this position, adequate images may not have been obtained, especially given the large thoracic excursions proposed. Nevertheless, this study raises the concern that spirometry in the period following a dive may increase the risk of DCI, especially as multiple attempts may be required to produce satisfactory respiratory data.

How might the possible increased risk of DCI by performing post-dive spirometry be minimised? Logically, spirometry should be performed in the period following surfacing when venous bubble load is at a minimum. Venous bubbles can be present for up to two hours after scuba dives on air.[13] One study of sports divers reported a Spencer Doppler bubble grade of at least III in 8 out of 28 divers within ten minutes of surfacing at a rate of 17 metres per minute, from a dive of 25 minutes at a depth of 35 metres' sea water.[6] A study examining decompression stress in hyperbaric chamber attendants by Doppler analysis found significant inter- and intra-individual variability even during a single, tightly controlled profile.[14]

Conclusion

In a single subject, spirometry induced sufficient changes in haemodynamics to provoke shunting across a PFO. This has implications for safety if spirometry is performed post-diving in subjects who may have a PFO. If spirometry is to be performed within two hours of surfacing, this should be regarded as a potential risk for DCI and preparations should be made to deal with such an event.

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Editor's note:
A link to the full echocardiographic study will be made available soon on the EUBS and SPUMS websites.