Effects of a patent foramen ovale on arterial saturation during exercise and on cardiovascular responses to deep breathing, Valsalva manoeuvre, and passive tilt: relation to history of decompression illness in divers

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Abstract

Objective—To determine whether the presence of a patent foramen ovale could result in significant arterial desaturation and affect the responses of heart rate and blood pressure to physiological manoeuvres and to determine whether responses differed between those with and those without prior clinical events associated with a patent foramen ovale.

Design—Blind controlled study.

Setting—Teaching hospital.

Patients—Divers with a large patent foramen ovale shown by contrast echocardiography and divided into those who had neurological decompression illness that started within 30 minutes of surfacing (group 1), those who had no history of decompression illness (group 2), and age and sex matched control divers who had no evidence of intracardiac shunts (group 3).

Main outcome measures—The change in percentage haemoglobin oxygen saturation during treadmill exercise and the response of heart rate and blood pressure to physiological manoeuvres.

Results—There were no significant differences between the three groups but two divers in group 1 showed clinically important desaturation during exercise and unusual blood pressure and heart rate responses to passive tilt. These divers were notable for having the greatest number of episodes and the most severe single episode of spinal cord decompression illness.

Conclusion—A large patent foramen ovale may be associated with clinically significant arterial desaturation and unusual responses of heart rate and blood pressure in some healthy subjects.

(Br Heart J 1994;71:229-231)

The presence in divers of interatrial right to left shunts shown by contrast echocardiography, and presumed to be patent foramina ovale, is associated with neurological decompression illness with the start of symptoms within 30 minutes of surfacing.¹ The mechanism is believed to be paradoxical gas

embolism. About a quarter of the population have a patent foramen ovale² and contrast echocardiography showed shunts in a quarter of divers.¹ The British Sub-Aqua Club estimates that about 70 000 divers in the United Kingdom perform between one and two million dives annually (C Allen, personal communication). Presumably one quarter of those dives are performed by divers with a patent foramen ovale. Yet there are only about 100 cases of decompression illness treated in the United Kingdom annually. It is unclear why a conservative dive profile that allows venous bubble nucleation³ should result in decompression illness in some divers with a patent foramen ovale but not in most, but one possibility is that the shunt is functionally larger in those who have decompression illness than in those who do not.

Contrast echocardiography is occasionally associated with adverse neurological effects, which are usually transient and are presumably the result of paradoxical embolism.¹ Also, evaluation of the size of the shunt with this technique is difficult. An investigation that had no adverse effects and that aided evaluation of the physiological consequences of shunts would therefore be valuable. If the test also detected those at greatest risk of neurological decompression illness, this might provide a useful screening test for susceptibility in divers.

The presence of a patent foramen ovale can result in sufficient right to left shunting to cause clinically important arterial desaturation in some pathological conditions.^{4,5} Clinically important desaturation has also been reported occasionally in apparently healthy subjects.⁴ This study was designed to test the hypothesis that arterial desaturation detected non-invasively by transcutaneous pulse oximetry during exercise, may be a marker for a functionally important shunt across a large patent foramen ovale. Exercise was chosen as a physiological manoeuvre that might affect shunting because exercise after a dive is commonly believed to predispose to decompression illness,⁶ although the mechanism for this is unknown.

Because large atrial septal defects affect the responses of heart rate and blood pressure to physiological manoeuvres we also tested whether these responses could be used to

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Accepted for publication
13 October 1993

detect large right to left shunts across a patent foramen ovale.⁷

Patients and methods

Divers who had intracardiac right to left shunts detected by contrast echocardiography with a previously described protocol,¹ and who had had neurological decompression illness that started within 30 minutes of surfacing (group 1, $n = 7$) were studied. One diver had three episodes of spinal decompression illness and six each had a single episode of spinal decompression illness. Five had associated symptoms; definite cerebral symptoms in one, probable cerebral involvement in one, cardiorespiratory symptoms in two, and cutaneous decompression illness in one. One diver had cutaneous involvement on three further occasions and possible cerebral effects on two. In each case the shunt was seen on the first contrast injection without provocative manoeuvres and size was graded as large according to a predefined method of assessment.¹ For a shunt to be considered large at least 20 bubbles had to be shown in the left heart in any frame. Each diver had recovered from his or her neurological injury sufficiently to exercise normally on a treadmill.

They were compared with other divers who also had large shunts shown on echocardiography with a single contrast injection without provocative manoeuvres, but who had not had decompression illness (group 2, $n = 5$). Twelve divers without shunts on contrast echocardiography and without history of decompression illness, who were age and sex matched to those in groups 1 and 2, served as a further control group (group 3). The three groups had similar diving experience as assessed by the average number of dives, deepest dive performed, and the proportion of each group that had done dives that required decompression stops. The proportions of smokers and ex-smokers in the groups were comparable. No subject had past evidence of heart or lung disease or on clinical examination and each had a normal chest x ray film.

Percentage haemoglobin oxygen saturation was measured at rest and during maximal symptom limited exercise with a treadmill and the Bruce protocol. The electrocardiogram of

subjects was monitored during the exercise test but blood pressure was not. Saturation was measured with a Ohmeda 3700 transcutaneous pulse oximeter, the output of which was recorded with a BBC series B computer and software designed for sleep study analysis. The output was analysed off line by a consultant chest physician who was experienced in such analysis and who was blind to the groups. Desaturation was considered to occur when there was a fall in saturation of at least 4% from the resting value and when there was a significant number of points below 95%.⁸

In separate experiments, resting sinus arrhythmia (recorded as the SD of 255 RR intervals) and the heart rate responses to deep breathing and a Valsalva manoeuvre were measured with a computer linked electrocardiogram,⁹ and blood pressure and heart rate responses to passive 60° upright tilt by sphygmomanometry and electrocardiogram. Pulse oximetry was not performed during the deep breathing, Valsalva manoeuvre, or tilt tests.

Subjects gave informed consent to the study, which had approval of the hospital Ethics Committee. Comparisons between the groups were by parametric and non-parametric statistics as appropriate.

Results

During exercise each diver reached at least stage 5 of the Bruce protocol before being limited by dyspnoea or fatigue. None had chest pain, electrocardiographic changes, or arrhythmias on exercise. Resting heart rate before the exercise test and maximum heart rate during the test were similar in the three groups.

All had normal oxygen saturations (at least 96%) at rest. Analysis of the group data showed no significant differences between the three groups in the magnitude of arterial desaturation or percentage of time spent desaturated. In 22/24 subjects saturation stayed at 93% or greater at all times with less than 4% of the exercise time at saturations in the range 93%-95%.

Two subjects, both healthy non-smokers in group 1, showed clinically important desaturation. These subjects were notable because one had had the greatest number of episodes of spinal cord decompression illness (case 1), and the other (case 2) had had the most severe single episode of spinal decompression illness associated with unprovocative dives. In case 1 arterial oxygen saturation fell to 80% at the end of exercise (fig 1). In case 2 there was a less dramatic but steady fall in arterial oxygen saturation from 99% at rest to 92% at peak exercise (fig 2). In both cases saturations were less than 95% for more than 20% of the exercise time. Figure 3 shows a representative example of an arterial oxygen saturation trace from one of the remaining 22 divers who showed no abnormal desaturation. Lung function tests (spirometry, lung volumes, flow volume loops, and transfer factor) were measured in cases 1 and 2 to exclude lung

Figure 1 Arterial oxygen saturation (SaO_2) three point average plot in case 1 showing a decrease to 80% at peak exercise. The period of exercise is shown between the arrows.

Figure 2 Arterial oxygen saturation (SaO_2) three point average plot in case 2 showing a steady decrease during exercise to 92%. The period of exercise is shown between the arrows.

Figure 3 Arterial oxygen saturation (SaO_2) three point average plot in a diver in whom desaturation did not occur. The period of exercise is shown between the arrows.

disease that might cause desaturation and could also be a reason for symptoms after diving, if pulmonary barotrauma resulted in arterial gas embolism. These tests were normal.

Sinus arrhythmia and the heart rate responses to deep breathing and a Valsalva manoeuvre were within normal age-related ranges⁹ in all the divers and there were no significant differences in response between the three groups. All responses to passive upright tilting were within the normal range except in cases 1 and 2 who had a fall in systolic pressure (>10 mm Hg) and an unusually large increase in heart rate (>23 beats/min).

Cardiac catheterisation of case 1 confirmed a catheter patent foramen ovale and he is currently awaiting closure because he wishes to resume scuba diving.

Discussion

The data suggest that transcutaneous pulse oximetry and non-invasive haemodynamic monitoring does not have a high predictive accuracy for detection of a patent foramen

ovale or susceptibility to decompression illness. However, the technique may detect those with the largest shunts and the greatest susceptibility to decompression illness: our most severely and most frequently affected divers became desaturated during exercise. This confirms that under some physiological conditions, right to left shunts across a patent foramen ovale is sufficient to produce clinically important arterial desaturation in some healthy adults.

We used exercise as a provocative physiological manoeuvre in this study for several reasons. Firstly, some forms of decompression illness are associated with the presence of a patent foramen ovale,¹ and some are believed to be precipitated by exercise after diving.⁶ If shunting could be shown to increase with exercise in those affected, this would support the hypothesis that paradoxical gas embolism is involved. Secondly, use of an exercise protocol allows standardisation of the physiological manoeuvre performed by each subject and allows reproducibility studies to be performed. Thirdly, an extended manoeuvre such as an exercise protocol means that if desaturation occurs its duration is likely to be sufficient to enable detection by pulse oximetry. Brief desaturation, as might occur during a Valsalva manoeuvre, can escape detection by pulse oximetry.

In these groups of healthy divers we found somewhat unusual blood pressure and heart rate responses to passive upright tilt in the two subjects with a patent foramen ovale who showed oxygen desaturation with exercise tests. We cannot tell whether these findings relate directly to the patent foramina ovale or are incidental.

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