Diving and percutaneous closure of persistent (patent) foramen ovale

Efrén Martinez-Quintana, Silvia Gopar-Gopar and Fayna Rodriguez-González

Abstract

(Martinex-Quintana E, Gopar-Gopar S, Rodriguez-González F. Diving and percutaneous closure of patent foramen ovale. *Diving and Hyperbaric Medicine*. 2015 June;45(2):126-128.)
Paradoxical arterial gas embolism after diving, in patients with a persistent foramen ovale (PFO) is a potentially catastrophic complication that occurs when gas bubbles occlude blood flow at cardiac or cerebral level. Because the relationship between PFO and decompression illness is currently not clear, we should ensure that patients understand the uncertainties about the efficacy of transcatheter closure of a PFO and the possibility of complications if closure is decided upon. We report a female diver who developed temporary bradycardia, hypotension and evidence of myocardial ischaemia during a closure procedure.

Key words
Scuba diving; persistent foramen ovale; transcatheter closure; side effects; case reports

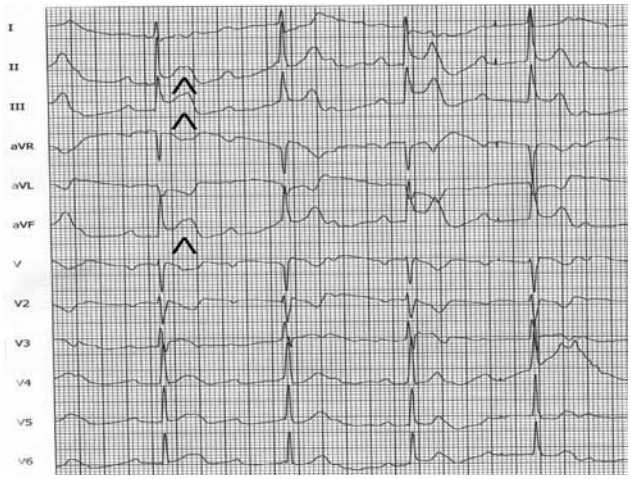
Introduction

Persistent foramen ovale (PFO) is an incomplete closure of the atrial septum that results in the creation of a flap or a valve-like opening in the atrial septal wall. In approximately 25% of the adult population the PFO remains open although symptoms are uncommon. However, in patients who have a PFO and a history of cryptogenic stroke, either aspirin or warfarin therapy is the first therapeutic choice. Meanwhile, percutaneous PFO closure is an acceptable alternative to medical therapy in those patients who have recurrent cryptogenic stroke despite optimal medical therapy.[1] In contrast, guidelines for screening for PFO in divers are difficult to create because the relationship between PFO and decompression illness (DCI) remains unclear and also because DCI is rare and percutaneous closure of PFO or atrial septal defects to prevent recurrent cerebral embolic events may have occasionally serious complications.[2,3]

Case report

A 37-year-old, female professional diver, without cardiovascular risk factors, was referred to our adult congenital heart disease unit for evaluation. The patient had a New York Heart Association functional class I/IV and a history of migraine with aura and tingling in the fingers and

Figure 1
12-lead electrocardiogram showing ST elevation in the inferior leads (arrow heads)



around the mouth in relation to diving. Physical examination revealed normal heart sounds, the extremities were normal with no oedema or venous thrombosis and respiratory and abdominal examination were unremarkable. Because DCI was suspected, transthoracic and transoephageal echocardiography were performed, showing normal left and right ventricular function, normal atria, no valvular heart disease and a PFO with a moderately positive bubble test when agitated saline contrast was administered and Valsalva manoeuvres were done.

For this reason, PFO closure was performed under fluoroscopic guidance with a 30-mm Amplatzer™ multi-fenestrated Septal Occluder ('Cribriform') (St Jude Medical, Inc.; St Paul, MN). However, following the release of the device, the patient developed chest pain, electrocardiographic inferior ST elevation (Figure 1), bradycardia and hypotension. This resolved with volume expansion and narcotic analgesia. Because of this event, coronary angiography was performed, which showed normal coronary arteries and an apparently correctly positioned device (Figures 2 and 3). Transthoracic echocardiography done during the procedure and the following day confirmed correct positioning of the device, with no residual right-to-left shunt.

Discussion

DCI includes both decompression sickness (DCS) and arterial gas embolism (AGE). DCS refers to the clinical syndrome of neurological deficits, pain or other clinical disorders resulting from the body tissues being supersaturated with inert gas after a reduction in the ambient pressure. Meanwhile, AGE describes the penetration of gas bubbles into the systemic circulation, from ruptured alveoli after lung barotrauma or migration from the venous circulation (venous gas embolism) either via a right-to-left

Figure 2
Left coronary artery in the right anterior oblique cranial projection with no significant coronary lesions (LDA – left descending artery; Cx- circumflex artery)

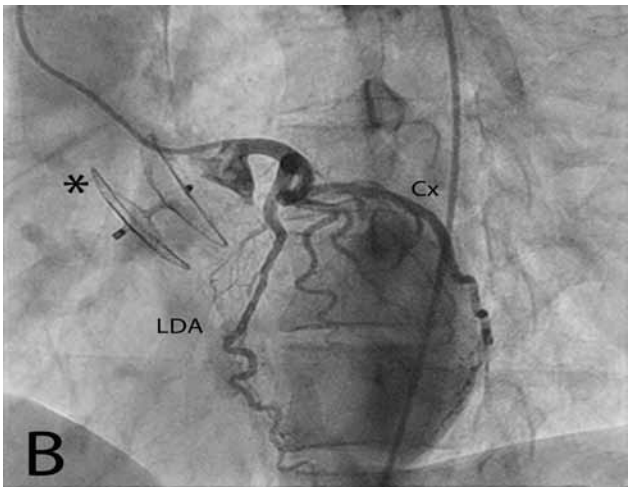
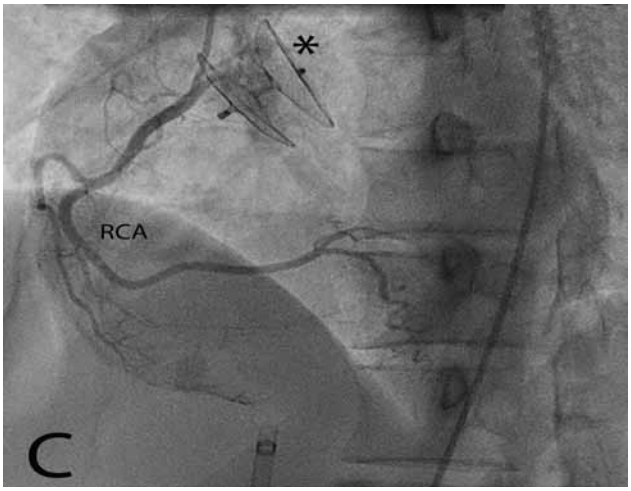


Figure 3
Dominant right coronary artery in the left anterior oblique projection (RCA- right coronary artery) without significant coronary lesions; * shows the position of the Amplatzer™ in Figures 2 and 3



shunt such as a PFO, atrial septal defect (ASD), extra-cardiac shunts or by overwhelming the filtering capacity of the lungs or from within the arterial circulation itself in severe DCS.

Diving often causes the formation of 'silent' bubbles upon decompression. A minor AGE may cause very mild symptoms or none at all. Nonetheless, if the bubble load is high, then the risks of DCS and the number of bubbles that could cross to the arterial circulation via a pulmonary shunt or PFO increase.[4] The symptoms of AGE usually occur during ascent or within a few minutes of surfacing and include mainly neurological and cardiovascular events. Neurological problems are protean, including sudden unconsciousness, motor deficits, seizures, visual

disturbances, aphasia and paraesthesiae. Meanwhile, cardiac problems include myocardial infarction, arrhythmias and sudden death.[5–7] In cases of myocardial infarction, micro-embolic gas bubbles most likely affect the terminal coronary arteries owing to their small size.[5] However, symptoms can sometimes develop more than 48 hours after diving, especially when the patient has travelled to altitude or flown after diving. In this context, paradoxical AGE of the myocardium may be seen as a result of delayed DCS.

Whilst approximately one quarter of the population have a PFO or a small ASD, the risk of paradoxical embolism in the overall sports diving population is very low. Moreover, specific DCS incidents cannot be linked to the presence of a PFO with any degree of certainty. In fact, there is no consensus on the optimal management of divers with a PFO and a history of neurological DCS. Current evidence on the efficacy of percutaneous closure of PFO for the secondary prevention of recurrent paradoxical embolism in divers is inadequate in quality and quantity, and the evidence on safety shows that there is a possibility of serious complications,[8] such as thrombus formation (mostly within the first month after device implantation), device embolization, erosion of cardiac structures leading to aorto-atrial fistula or pericardial tamponade and post- or peri-procedural arrhythmias. Notwithstanding, evaluation for PFO using echocardiography must be considered in divers with severe or repetitive neurological, cutaneous or cardiorespiratory DCS (especially with a history of migraine with aura).[9,10] If it is believed that a diver's repetitive DCS is related to a PFO, reduction of decompression stress in future diving activities by more conservative diving practice may be a better approach than PFO closure in many divers.[9] If closure is proposed for a diver, they should be advised not to dive until the closure has been performed and adequate follow up with repeat bubble contrast echocardiography demonstrates a satisfactory complete closure or only a small residual shunt.

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Conflicts of interest: nil

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Efrén Martinez-Quintana,[1] Silvia Gopar-Gopar,[1] Fayna Rodriguez-González[2]
[1]Cardiology Service, Insular-Maternao Infantil University Hospital, Las Palmas de Gran Canaria, Spain
[2]Dr. Negrin University Hospital of Gran Canaria, Las Palmas de Gran Canaria, Spain

Address for correspondence:
Efrén Martinez-Quintana, MD, PhD
Servicio de Cardiologia
Complejo Hospitalario Universitario Insular-Materno Infantil
Avda. Maritima del Sur s/n 35016
Las Palmas de Gran Canaria, Spain
E-mail: *<efrencardio@gmail.com>*