Patent foramen ovale, a normal variant or a congenital abnormality requiring treatment?

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Abstract

It has now become possible to close a patent foramen ovale (PFO) using a percutaneous device. In addition, it has become increasingly clear that right-to-left shunting through a PFO can cause both stroke and decompression illness, due to paradoxical embolism of blood clots or gas bubbles. For these reasons, diagnosis of large PFO with significant right-to-left shunts has become important. The diagnosis can be made by transthoracic echocardiography with injection of bubble contrast, combined with multiple sustained Valsalva manoeuvres. Whilst transoesophageal echocardiography provides detailed anatomical information, functional information (with regard to right-to-left shunting) is better provided by transthoracic studies where a Valsalva can be properly performed. Device closure can prevent right-to-left shunting and can be achieved using a number of different devices. However, device closure has yet to be proven beneficial in a randomised trial. In light of the clear evidence implicating PFO, we undertake closure procedures in selected patients.

Key words: patent foramen ovale, congenital heart disease, catheter devices.

Introduction

A patent foramen ovale (PFO) is a relatively common finding: it is present in approximately 25% of the population. A large PFO is much less common but may occur in as many as 5% of the population. The advent of catheter devices that can be used to close these communications safely, without the need for open surgery, has focused attention recently on the indications for closure.

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Two examples of patients who have undergone catheter closure of their PFO are described and the issues surrounding PFO closure are discussed.

Patient 1

A 56 year old man awoke after hip replacement surgery to find that he had right-sided weakness and difficulty with his speech. A clinical diagnosis of stroke was made. His symptoms resolved over the following week. He had no clinical evidence of vascular disease, no evidence of atrial fibrillation and a normal carotid ultrasound examination. The orthopaedic surgeon (GT) referred him for cardiological assessment, being of the opinion that a paradoxical fat embolism had occurred. Transthoracic echocardiography showed a mobile atrial septum and bubble contrast (8 ml saline agitated with 1 ml of blood and 1 ml of air) crossed from the right atrium to the left. A transoesophageal echocardiogram (TOE) confirmed that the atrial septum was aneurysmal (defined as a floppy septum that moves > 1 cm through the cardiac cycle) with an associated patent foramen ovale (PFO).

It was considered likely that he would need further orthopaedic surgery, which has been shown in studies using TOE to cause venous thromboemboli. As these emboli lodge in the pulmonary capillaries, the right heart pressure rises and shunting of emboli into the systemic circulation through the PFO occurs. The presence of an atrial septal aneurysm is associated with PFO and an increased incidence of stroke.

Because the patient had already suffered one stroke and had both a PFO and an atrial septal aneurysm, percutaneous closure was deemed appropriate. The patient's PFO allowed the passage of a 12 mm balloon catheter across the atrial septum, confirming a moderately large defect (see figure 1). The defect was then successfully closed using a 35mm Amplatzer PFO occluder (see figure 2). He was anticoagulated with warfarin for six months and then maintained on aspirin. His subsequent course has been uncomplicated.

Patient 2

A 52 year old man had been scuba diving on a wreck at 45 metres. As he climbed aboard his diving boat he fell to the ground, with leg weakness and a reduced conscious level. His dive buddies recognised that he was suffering from an acute neurological decompression illness (DCI) so they treated him with oxygen and called the coastguard. He was transported by air to the Hyperbaric Medical Centre in Plymouth for recompression therapy. He made a complete neurological recovery and was keen to return to diving.

Figure 1. Transoesophageal echocardiographic image showing the spherical balloon sizing catheter (12 mm diameter) passing between right atrium (RA) and left atrium (LA) through the patent foramen ovale. The flap-like atrial septum is resting on top of the catheter. This illustrates how a large paradoxical embolus could occur

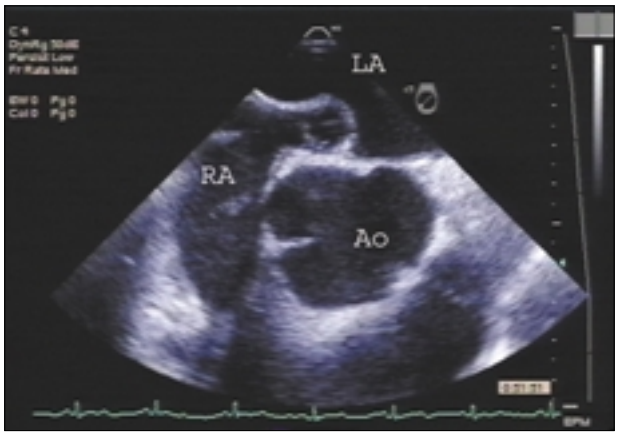
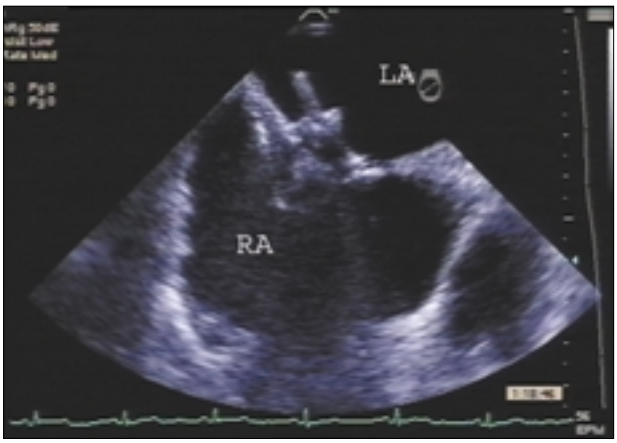


Figure 2. Transoesophageal echocardiographic image showing the patent foramen ovale occluder in the atrial septum



DCI is caused by nitrogen, dissolved in blood and tissues whilst under pressure, coming out of solution on ascent. Dive duration and maximum depth are the main determinants of the amount of nitrogen that dissolves, and hence the risk of DCI.

Early-onset neurological DCI is associated with large PFOs where, following venous bubble contrast injection, more than 25 bubbles are able to pass through the defect into the left atrium.[1] He, therefore, underwent bubble contrast echocardiography. Initially, no bubbles crossed into the left atrium but, following release of the Valsalva manoeuvre, a huge number of bubbles (more than 100) filled the left atrium. Since the patient had a history of DCI and a huge shunt across his PFO, he was advised to severely limit his diving. He was desperately keen to return to unrestricted diving, so he requested a closure procedure. This was performed using a 35 mm Amplatzer PFO occluder. A follow-up echocardiogram performed three months later confirmed adequate closure. No bubbles crossed to the left atrium despite six Valsalvas.

He has now returned to diving without restrictions, but has been advised to avoid multiple or very deep dives causing severe decompression stress (as it is still possible to suffer DCI without a PFO).

Discussion

The presence of a functionally significant PFO can be assessed by bubble contrast transthoracic echocardiography with Valsalva manoeuvres. Imaging should continue as the Valsalva is released as well as when it is initiated. Adequate bubble contrast should opacify the entire right heart. If a large PFO is present, more than 25 bubbles will cross into the left atrium.

Imaging should be performed in the apical view because, in other views, the right heart lies between the left atrium and the transducer. When the right heart fills with bubble contrast, the left heart is obscured and, therefore, the passage of bubbles cannot be detected. A false-positive test is obtained when a pulmonary arteriovenous malformation (AVM) is present but the timing of the appearance of bubbles in the left heart and the TOE findings should differentiate PFO from AVM. A false-negative result is possible if TOE is used alone as an adequate Valsalva manoeuvre is not possible in the presence of the probe.

PFO can be closed percutaneously with low risk in the vast majority of patients. The procedure is performed in the catheter laboratory with the patient under general anaesthesia. X-ray screening and TOE imaging are used to guide placement of the device. The Amplatzer device is retrievable so that if placement is unsatisfactory or if the procedure is abandoned, all of the equipment can be removed without the need for surgery.

Whether there is a long-term reduction in the risk of stroke or decompression illness has not been proven by randomised, controlled trials. However, it has been demonstrated that there is a causal relationship between the anatomical defect and paradoxical emboli[2] and DCI. This is clearly a plausible biological mechanism. For those patients with a history of stroke or DCI, it does seem rational to close the defects in the absence of another explanation. Patients who had undergone closure were found in one study to have a mean defect size of 12 mm.[3] While the prevalence of probe-patent PFO is 27%, the prevalence of a defect of > 10 mm was found to be only 1.3%.[4] It would appear, therefore, that PFOs of sufficient size to cause stroke or DCI are uncommon in the general population and should be considered pathological.

Prospective clinical trials would be helpful but inclusion criteria will need to be carefully defined. In the meantime, there will clearly be some patients for whom PFO closure seems appropriate and some who may decline their consent to inclusion in a randomised trial.

Key messages

- Right-to-left shunting through a PFO is associated with stroke and decompression illness
- Transthoracic echocardiography with bubble contrast and sustained Valsalva is the diagnostic test for right-to-left shunting
- Transoesophageal echocardiography can be useful but may be falsely negative for right-to-left shunts
- Device closure is straightforward but unproven in a randomised trial

Editors' note

This article continues our series on grown-up congenital heart (GUCH) disease, which began in February issue. Previous articles included:

- adult congenital heart disease: time for a national framework (*Br J Cardiol* 2002;**9**:65–67)
- grown-up congenital heart disease: experience in a district general hospital (*Br J Cardiol* 2002;**9**:92–98)
- atrial septal defects: a differential diagnosis for breathlessness in adults and the elderly (*Br J Cardiol* 2002;**9**:99–102).

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