Patent Foramen Ovale Closure for Hypoxemia

Jonathan Marvin Tobis, MD[a,*], Deepika Narasimha, MD[b], Islam Abudayyeh, MD[b]

KEYWORDS

• Patent foramen ovale • Hypoxemia • Right-to-left shunting • COPD • Platypnea-orthodeoxia

KEY POINTS

- Hypoxemia may occur in the presence of a patent foramen ovale (PFO) caused by right-to-left shunt across the interatrial septum.
- Right-to-left shunting can be exacerbated by clinical conditions that alter the relative pressure between the right and left atria (eg, obstructive sleep apnea, chronic obstructive pulmonary disease, and pulmonary hypertension) or by changes in the anatomic relationship between the inferior vena cava and the foramen ovale caused by surgery or other conditions that may cause cardiac rotation.
- A PFO is one cause of platypnea-orthodeoxia (dyspnea and hypoxemia while upright, which improves in the recumbent position), in addition to liver and lung disease.
- PFO closure may successfully treat hypoxemia in selected cases.

INTRODUCTION

Several clinical syndromes are associated with patent foramen ovale (PFO), including stroke caused by paradoxic embolism, migraine headaches with aura, and decompression sickness. Although the link between these disorders and PFO has been studied extensively, the associations between hypoxemia-related conditions such as chronic obstructive pulmonary disease (COPD), obstructive sleep apnea (OSA), and the platypnea-orthodeoxia syndrome (POS) are not fully defined. Case reports linking PFO to hypoxemia that is out of proportion to the severity of lung disease have been described over the last 2 decades.[1–3] This article describes the mechanisms linking hypoxemia with the presence of a PFO, the clinical conditions in which PFO may play a role in contributing to hypoxemia, and the role of PFO closure in management.

PATENT FORAMEN OVALE AND HYPOXEMIA

In the fetal circulation, blood from the inferior vena cava (IVC) flows from the right atrium (RA) into the left atrium (LA) through the foramen ovale, which acts as a one-way valve. This valve ensures that oxygenated blood from the placenta directly enters the systemic circulation, and bypasses the nonaerated, amniotic fluid–filled lungs. The remaining oxygenated blood that gets into the right ventricle (RV) is directed through the ductus arteriosus into the descending aorta, thus also bypassing the nonfunctional lungs. The IVC is aligned with the PFO by the eustachian valve, which facilitates the IVC flow directly across the septum. Blood from the superior vena cava (SVC) meanwhile is directed down into the RA and across the tricuspid valve (Fig. 1). After birth, the pulmonary vascular resistance decreases, leading to a decrease in RA

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[a] Division of Cardiology, UCLA David Geffen School of Medicine, 10833 Le Conte Avenue, Factor Building, Room B-976, Los Angeles, CA 90095, USA; [b] Division of Cardiology, Interventional Cardiology, Loma Linda University Health, 11234 Anderson Street, MC 2434, Loma Linda, CA 92354, USA

* Corresponding author.

E-mail address: jtobis@mednet.ucla.edu

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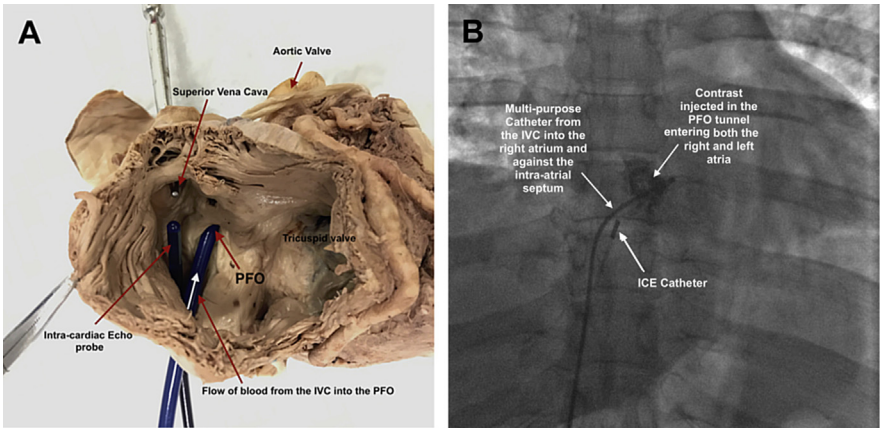

Fig. 1. (*A*) Gross anatomy showing the atrial septum from the right atrial side. The delivery catheter follows the course of the IVC and into the PFO. The SVC is angled away from the PFO and points toward the tricuspid valve. (*B*) Fluoroscopic image showing the course of a catheter as it enters the RA via the IVC and is directed toward the PFO. ICE, intracardiac echocardiography.

pressure. As the LA pressure exceeds the RA pressure, the septum primum closes against the septum secundum. With time, the septum primum and secundum fuse leaving behind the fossa ovalis. For approximately 20% of the population this fusion remains incomplete, resulting in a PFO.[4–6] In most people who have a PFO, it is an incidental finding and not associated with symptoms or hypoxemia. However, right-to-left shunting (RLS) of blood can occur during any activity that increases venous return and right atrial pressure, such as the release of the Valsalva maneuver. It is estimated that a cryptogenic stroke occurs in 1 in 1000 people per year who have a PFO. Of the patients with PFO who have some related symptom, only 3% present with symptomatic hypoxemia.

Although RLS causing hypoxemia is rare in patients without increased right-sided pressures, there have been reports of significant hypoxemia in patients with normal right-sided pressures.[7] Godart and colleagues[7] report a series of 11 patients with PFO who presented with significant dyspnea and cyanosis, which subsided after percutaneous closure of the atrial defect. Six of the 11 patients also had POS, in which the hypoxemia occurs on sitting or standing up. Various theories exist to explain RLS in patients with normal right-sided pressures. These theories include preferential blood flow streaming from the IVC to the LA because of the presence of a large eustachian valve.[8,9] Another theory describes the presence of a systolic right-to-left atrial pressure gradient in conditions such as RV infarction, right atrial myxoma, and mechanical ventilation.[10,11] In these cases, inspiration, the Valsalva maneuver, and changes in posture exacerbate RLS. In the 11 patients described earlier, the investigators observed that all of the patients had rotated atrial septa toward the horizontal axis such that the PFO was more directly in line with the blood flow from the IVC. Note that this phenomenon has also been noted in patients with ascending aortic aneurysms that may distort the septum, or after pneumonectomy and abdominal surgery, which are thought to alter the anatomic orientation or opening height of the PFO. The incidence and degree of shunting also increase when the septum primum is aneurysmal or highly mobile (Fig. 2).[9,12]

Establishing that an RLS through a PFO is primarily responsible for hypoxemia or cyanosis in patients without increased right-sided pressures can be challenging. Persistent desaturations despite administration of 100% oxygen therapy should alert clinicians to the possibility of an RLS. A transesophageal echocardiogram (TEE) showing cross-septal flow on color Doppler or contrast administration can help confirm the diagnosis. In addition, on cardiac catheterization, there is a step-down in the oxygen saturation levels in the LA compared with the pulmonary veins, with return to normal blood saturation after occlusion of the PFO using a soft balloon sizing catheter.[7] Once identified, quantifying the degree of hypoxemia caused by RLS can be difficult. A low pulmonary vein oxygen saturation might indicate a mixed picture in which pulmonary disease is a contributor to hypoxemia, whereas a step-down of saturations in the LA compared with the pulmonary veins should indicate that an RLS through the PFO plays a larger role. Crossing the PFO to obtain