

# Acute Respiratory Failure: A Right-to-left Intracardiac Shunt

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## Abstract

Right-to-left intracardiac shunting through a patent foramen ovale [PFO] is well described in the medical literature and usually relates to an elevated right atrial pressure. However, this abnormal flow pattern has also been identified in the setting of mediastinal distortion. We have identified a similar example where ascending aortic enlargement produced a right-to-left intracardiac shunt through a patent foramen ovale; despite normal cardiac pressures, the patient clinically progressed to respiratory failure requiring surgical closure of the PFO

## INTRODUCTION

Depending on the method of selection, a patent foramen ovale [PFO] can be identified in up to 35% of the population.<sup>(1)</sup> Patency can persist into adulthood yet remain asymptomatic - without abnormalities on physical examination, electrocardiogram, or chest radiograph.<sup>(2)</sup> Complications associated with a PFO usually include paradoxical embolus and, in the presence of pulmonary hypertension, subsequent development of a right-to-left shunt. However, this abnormal-flow pattern can occur despite normal intracardiac pressures. Strunk et al. proposed 4 mechanisms to explain this “upstream” phenomenon: (1) the development of transient (rather than mean) interatrial pressure differentials during the cardiac cycle; (2) preferential flow direction from the inferior vena cava toward the foramen ovale (i.e. a persistent eustachian valve); (3) a decrease in right ventricular compliance; and (4) accentuation of interatrial gradients during respiration and the Valsalva maneuver.<sup>(1)</sup> Relative distortion of mediastinal structures leading to an elevated right atrial pressure has also been reported to cause right-to-left shunting. Savage et al. described two such cases wherein right atrial compression from a dilated, elongated aorta caused shunting through a PFO which led to intermittent positional hypoxia.<sup>(3)</sup> We have identified a similar case of ascending aortic enlargement that produced a right-to-left intracardiac shunt. With progression to respiratory failure, the patient required endotracheal intubation, mechanical ventilation, and surgical closure of the foramen ovale.

## CASE REPORT

A 72 year-old female with a history of myocardial infarction nine years prior presented with dyspnea and intermittent chest pain; recent illnesses included a transient ischemic attack (left-sided upper extremity weakness) and one episode of supraventricular tachycardia which resolved with medical therapy. Her dyspnea had progressed over a 6 month period - leading to an outpatient catheterization. This revealed normal coronary arteries and left ventricular function. Passage of the right-sided catheter into the left atrium, compatible with a patent foramen ovale, was also incidentally noted. During the examination, intracardiac pressures were normal and an “oxygen step-up” could not be appreciated. However, two weeks later, she presented to the emergency department in acute respiratory distress. Following endotracheal intubation and the initiation of mechanical support, a transesophageal echocardiogram demonstrated dilatation of the aortic root (diameter 3.5 cm). Impingement of the right atrium with a significant right-to-left shunt was also noted. A ventilation-perfusion scan and pulmonary angiogram did not reveal an acute embolus. Repeat catheterization again failed to reveal a significant oxygen step-up.[Table 1] A contrast bubble study through the cephalic vein demonstrated direct right-to-left shunting at the inter-atrial septum. In view of her incapacitating arterial hypoxemia, closure of the foramen ovale was advised. At operation, the aortic root was noted to be slightly dilated, causing a mild impingement on the right atrium. Upon entering the right atrium, a patent foramen ovale was identified and closed primarily with a prolene suture. Further

intracardiac abnormalities could not be appreciated. The patient's postoperative course was uneventful and her arterial saturations quickly returned to normal allowing extubation and eventual discharge 5 days later.

### Figure 1

Table 1: Right Heart Catheterization, Pressure & Saturations

Wedge, mean	9 mmHg	RA	71 %	LV	89 %
RA, mean	10 mmHg	RV	71 %	Aorta	95 %
RV	28 / 11 mmHg	PA	68 %		
PA	23 / 13 (17) mmHg				

## DISCUSSION

Acute right-to-left intracardiac shunting has been well described in the medical literature and usually relates to an elevation in right atrial pressure. The foramen ovale, a membranous structure, functions as a unidirectional valve and is critical to the success of in-utero circulation. However, following birth and the associated elevation in left atrial pressure, this membranous structure "closes" to prevent further inter-atrial flow. Given that 20 - 25% of normal individuals have a patent foramen ovale(4), any condition which elevates right-sided pressure can theoretically produce a right-to-left shunt. These conditions include, but are not limited to, congenital heart disease associated with right atrial and ventricular hypertension (i.e. tricuspid atresia, tricuspid stenosis, tetralogy of Fallot, pulmonic stenosis, and septal defects), pulmonary emboli, right ventricular infarction, pulmonary hypertension from lobectomies, pneumonectomies, or chronic obstructive disease, tumors in the form of right atrial myxomas or metastatic implants causing relative flow obstruction to the right ventricle, pericardial tamponade, ARDS, and the application of extrinsic positive end-expiratory pressure [PEEP].(1, 4, 5)

Several mechanisms have been postulated to explain the occurrence of right-to-left shunting in the setting of normal cardiac pressures.(1) First, during the normal cardiac cycle there is a brief moment in early ventricular systole where the right atrial pressure is greater than the left atrial pressure. This gradient can be accentuated with release from the Valsalva maneuver, which suddenly increases systemic venous return and thus, right atrial pressure. Second, a preferential flow direction from a persistent Eustachian valve; with this, blood returning through the inferior vena cava follows an in-utero circulatory pattern to create a significant shunt. In our patient, this physical abnormality was not identified by echocardiography or at the time of operative exploration. The third mechanism consists of a

change in right ventricular compliance - either from a progressive loss of distensibility (i.e. chronic fibrosis) or from an acute ischemic event. Silver et al. reported, in their review of the literature, ten such cases of intracardiac shunting related to right ventricular infarction - only four patients survived until hospital discharge.(5) The last proposed theory involves respiratory variation in the intra-atrial pressures. With inspiration, there is an increase in venous return and thus, an increase in right atrial pressure. There is also a simultaneous decrease in thoracic pressure and, as such, an increase in pulmonary blood volume which decreases left atrial pressure.(1) This combination may enhance a pre-existing right-to-left pressure gradient. However, in our patient, the severity of her symptoms seemed to be related to a mechanical distortion of the right atrium - despite normal intracardiac pressures. As demonstrated by echocardiography, her ascending aorta was slightly dilated and appeared to be interfering with right atrial function; given the angle of associated aortic compression, subcostal and apical views were required to better visualize the interatrial septum. There exists only one prior report in the English literature of right-to-left shunting through a patent foramen ovale caused by ascending aortic compression. In 1996, Savage et al. described their experience with 2 cases of ascending aortic aneurysm producing extrinsic compression of the right atrium as well as left-sided rotational displacement [Patient 1: 7.6 cm. aortic aneurysm; Patient 2: 8.7 cm. aortic aneurysm].(3) While our patient did not have a true aortic aneurysm, the observed aortic root dilatation must have been of sufficient caliber to cause elevation in the right atrial pressure and possibly alter flow dynamics - though we did not appreciate this in her preoperative diagnostic studies. Given that primary closure of the patent foramen ovale restored normal arterial saturations and abolished her symptoms, this clinical entity should be considered in the differential diagnosis of refractory hypoxemia and respiratory failure.

## References

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