

# Profound Hypoxemia Resulting from Shunting Across an Inadvertent Atrial Septal Tear After Left Ventricular Assist Device Placement

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Defects within the interatrial septum (IAS) can be a source of significant right-to-left shunting and hypoxemia, particularly after placement of a left ventricular assist device (LVAD). We report a case of LVAD placement in which an unrecognized IAS tear occurred intraoperatively, leading to profound arterial desaturation. Transesophageal echocardiography (TEE) was instrumental in making the diagnosis. Certain intraoperative

events increased the pressure gradient between the right and left atria, aggravating hypoxemia. We recommend that patients undergoing LVAD placement be screened intraoperatively with TEE for unrecognized IAS defects. Re-examination of the IAS should occur on weaning from cardiopulmonary bypass.

(Anesth Analg 2004;98:937–40)

**C**omplications relating to left ventricular assist devices (LVAD) are borne at a time of minimal tolerance for instability. In particular, patients may experience life-threatening hypoxemia despite adequate pulmonary function. Right-to-left shunting may occur across the interatrial septum (IAS) via unrecognized defects. Shunting may be insignificant before LVAD placement, whereas device operation may decrease left atrial pressure (LAP) sufficiently to cause considerable conduction of venous blood into the left atrium (LA).

Shunting with LVAD occurs across PFO (patent foramen ovale) (1–3). We report a case of severe hypoxemia after placement of the Abiomed BVS5000® (Abiomed, Danvers, MA) in a patient without prior evidence of interatrial communication. Specific events during the procedure account for the timing of onset of desaturation, and we discuss maneuvers we found effective in minimizing the shunting.

## Case Report

A 42-yr-old man was admitted for high-risk repeat coronary artery bypass grafting. Preoperative echocardiography showed severe LA and LV enlargement with severe systolic dysfunction (ejection fraction <25%), moderate right ventricular (RV) dysfunction and moderate mitral regurgitation. No IAS defects were noted.

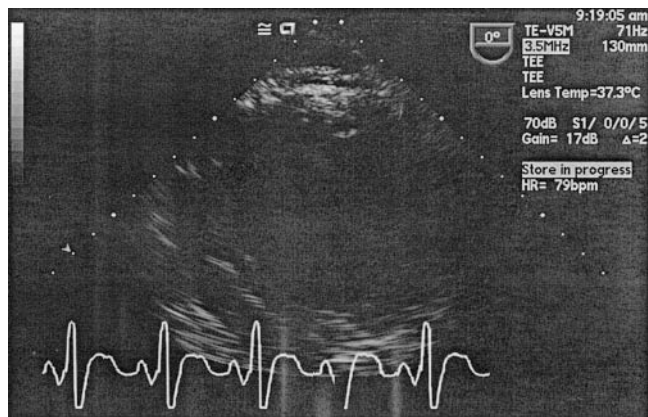
Intraoperative transesophageal echocardiography (TEE) revealed severe LV dysfunction (Fig. 1, clip 1). The IAS was imaged with color flow Doppler (CFD) without evidence of atrial septal defect (ASD) or PFO (Figs. 2, 3, clips 2, 3). Placement of the retrograde cardioplegia cannula was difficult and required several attempts. During cardiopulmonary bypass (CPB), the posterior descending artery was grafted and the mitral valve was repaired. After CPB, LV support was provided with three inotropic drugs and an intraaortic balloon pump. Arterial blood gas measurements revealed an initial Pao<sub>2</sub> of 75 mm Hg (Fio<sub>2</sub>, 1.0) with improvement to 276 mm Hg over 45 min. By two-dimensional (2D) TEE, no defect was observed within the IAS nor was the septum bowed toward either atrium. Because of hemodynamic instability (severe LV dysfunction by TEE, systolic blood pressure <70 mm Hg, metabolic acidosis), the LA and aorta were cannulated and LVAD support initiated.

Over the next 75 min, Spo<sub>2</sub> exceeded 95%; however, Pao<sub>2</sub> declined significantly (57–76 mm Hg). Fio<sub>2</sub> was maintained at 1.0 throughout this period, with volume-cycled ventilation providing tidal volumes of 680–750 mL and with peak inspiratory pressure of 26–33 cm H<sub>2</sub>O (no added positive end-expiratory pressure). Within 5 min after sternal approximation, Spo<sub>2</sub> declined to 80%, (Pao<sub>2</sub>, 42 mm Hg). TEE showed the IAS bowing toward the LA

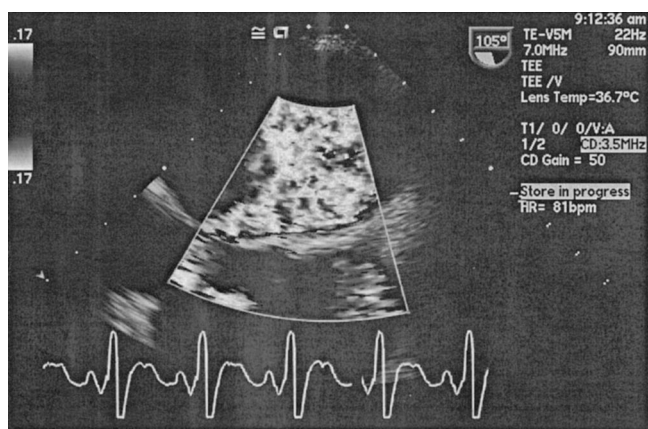
Supplemental material available at [www.anesthesia-analgia.org](http://www.anesthesia-analgia.org). Accepted for publication October 24, 2003.

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DOI: 10.1213/01.ANE.0000105861.99795.00



**Figure 1.** Large dilated left ventricle with severe global systolic dysfunction before cardiopulmonary bypass. See online clip #1 at <http://www.anesthesia-analgesia.org/> for corresponding moving picture clip.

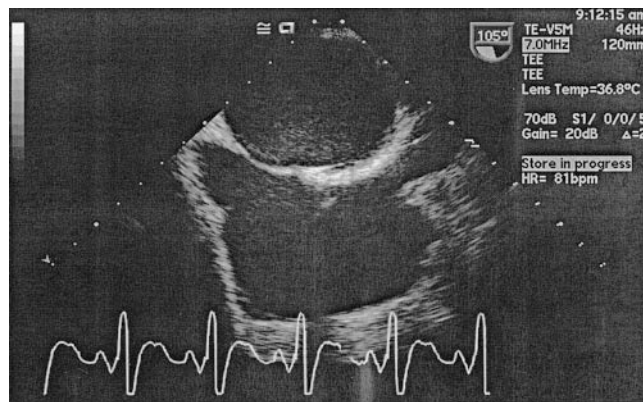


**Figure 2.** Bi-atrial view showing representative image in which no atrial defects were found using color Doppler. See corresponding online clip #2 at <http://www.anesthesia-analgesia.org/>.

and a 4–5 mm defect centrally within the fossa ovalis (Fig. 4, clip 4). CFD revealed obvious right-to-left shunting via the defect (Fig. 5, clip 5).

For 30 min,  $SpO_2$  remained stable (80%–85%). Skin closure was completed with intention to repair the IAS by percutaneous methods. Shortly after applying suction (–20 cm  $H_2O$ ) to pleural drainage tubes,  $SpO_2$  decreased to 8%—clearly indicative of profound hypoxemia, despite a wide range of error associated with pulse oximetry at saturations less than 90%. Central venous pressure (CVP) increased from 10–15 mm Hg to 23 mm Hg. The chest was promptly reopened with intent to reimplement CPB and repair the defect. Immediately after reopening,  $SpO_2$  improved to 100% ( $Pao_2$ , 70 mm Hg). TEE showed the IAS no longer bowing toward the right atrium (RA) but moving bidirectionally with each cardiac cycle (Figs. 6, 7, clips 6, 7).

With poor hemostasis, further CPB was felt to be undesirable and the chest was stented open and covered with synthetic pericardial patch. Drainage tubes were connected to underwater seal only, and the patient was transferred to the intensive care unit, maintaining  $SpO_2$  between 85% and 90%.



**Figure 3.** Two dimensional bi-atrial view showing intact interatrial septum. See corresponding online clip #3 at <http://www.anesthesia-analgesia.org/>.



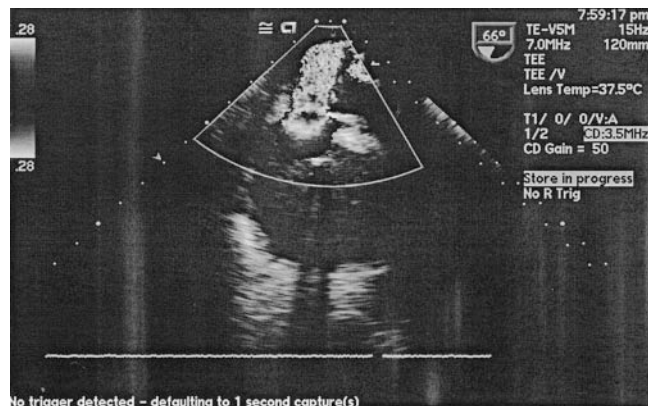
**Figure 4.** After chest closure and development of clinically significant desaturation, a defect in the inter-atrial septum is observed with prominent bowing of the inter-atrial septum toward the left atrium. See online clip #4 at <http://www.anesthesia-analgesia.org/>.

Thirty hours later, the patient returned to the operating room for definitive management. A traumatic tear within the fossa ovalis was noted, with a small co-existing PFO.

The patient subsequently developed multisystem organ failure over the ensuing 24 h; support was withdrawn 3 days after the original operation.

## Discussion

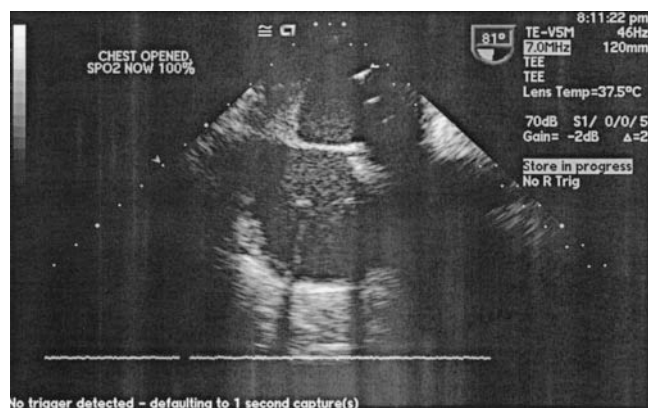
With a prevalence of 20%–35% in unselected autopsy cases (4), PFO is both common and, for most, without physiologic significance. The pressure gradient between the atria disfavors clinically significant right-to-left shunting (5–8) in healthy patients, whether awake or anesthetized and ventilated. Studies using contrast echocardiography detect most PFO using Valsalva's maneuver, creating, on release of the maneuver, a transient increase in RA pressure over LA pressure, facilitating brief passage of contrast across the defect (5,6,9). In such studies, few PFO can be detected unless elicited in this way.



**Figure 5.** Color flow Doppler image showing significant right-to-left flow across the atrial defect. Proximal isovelocity surface area (PISA) rings are noted indicating acceleration of flow toward the defect on the right atrial side. See clip #5 at <http://www.anesthesia-analgia.org/>.



**Figure 7.** After chest re-opening, the atrial septal defect is still seen, but the inter-atrial septum is no longer bowed, and moves bi-directionally. See clip #7 at <http://www.anesthesia-analgia.org/>.



**Figure 6.** The inter-atrial septum is flat and moves bidirectionally after chest re-opening, with simultaneous improvement in oxygenation, lack of significant pressure gradient across the atria. See clip #6 at <http://www.anesthesia-analgia.org/>.

LVAD, however, presents a dangerous context for IAS defects. With the Abiomed<sup>®</sup>, the inflow cannula is placed within the LA or LV and fills the device by gravity. In this way, LA pressure is reduced, creating a gradient that may result in considerable shunting and hypoxemia when interatrial communications are present. Factors that increase RA pressure may compound this effect.

It has been suggested, therefore, that patients requiring LVAD undergo intraoperative TEE evaluation for PFO (3), using contrast echocardiography, after a simulated release of Valsalva's maneuver (the lungs are inflated manually to an airway pressure of 30 cm H<sub>2</sub>O, held briefly, then released; this generates a transient increase of RA pressure over LA pressure). Importantly, although most of the atrial shunting in our patient had a different explanation, a small PFO was found at surgical exploration that was not detected by

2D or CFD techniques alone. In one series of anesthetized patients, only 4 of 13 PFO were detected by Doppler echocardiography alone; the rest required contrast imaging (agitated saline) to make the diagnosis, usually with simulated Valsalva's maneuver (5). Our case confirms the importance of these methods in excluding atrial defects using TEE.

Of greater significance in this case was the presence of an atrial septal tear facilitating shunting and hypoxemia with LVAD placement. This has not previously been reported. Although the tear may have resulted during attempts at coronary sinus cannulation, the IAS was imaged and appeared normal after the initial bypass run. It is more likely, especially given the central location within the septum, that the defect occurred with LA cannulation during the second CPB period for LVAD placement. We advocate that, in cases of LVAD placement, IAS defects should be excluded by intraoperative prebypass TEE not only in the manner previously described (using saline contrast with a simulated Valsalva's maneuver) but also with brief but focussed reinspection of the septum at the time of separation from CPB. In this manner, potentially life-threatening defects may be corrected either during the initial bypass run or during an early return to bypass, before the development of significant hypoxemia.

Although widely practiced, the placement of the LVAD's inflow cannula within the LA may be substituted by placement within the LV apex, thus obviating the possibility of traumatic injury to the IAS. However, this complication is rare and may not override competing considerations that may justify LA placement, such as LV apical necrosis, significant aortic insufficiency, or significant mitral stenosis.

Although the A-a gradient was widened, SpO<sub>2</sub> remained more than 94% until chest closure; only then did saturation decrease to 80%. Moreover, there was

prompt return of  $\text{SpO}_2$  to 100% after reopening. We postulate that chest closure increased RA pressure, as confirmed by measurement of CVP, through direct transmission of increased pleural pressure to the RA, compounded by a decrease in the effective compliance of the RV, contributing to an increase in RA pressure. In this way, the interatrial pressure gradient was increased, as LAP was held comparatively constant by the LVAD inflow cannula.

The changes in the pressure gradient we have attributed to chest closure and reopening are supported by the TEE images obtained. Figure 4 and clip #4, acquired after chest closure ( $\text{SpO}_2$  80%–85%), depict the 5-mm atrial tear with the IAS consistently bowed leftward, indicating a significant pressure gradient ( $\text{RA} > \text{LA}$ ). In contrast, Figure 5 and clip #6, acquired after chest reopening ( $\text{SpO}_2$  100%), demonstrate bidirectional IAS movement, suggesting comparatively equalized pressure.

Classic and modern experiments demonstrate that negative pleural pressure increases RA pressure (7,8) owing to increased venous return. The most profound decrease in  $\text{SpO}_2$  came on application of suction to the drainage tubes. We postulate that increased shunt flow resulted from increased RA pressure that ensued from negative pleural pressure and increased venous return, compounded by decreased RV compliance.

## Conclusion

This is the first report of profound hypoxemia from shunting across a traumatic ASD in a patient with LVAD support. The importance of examining the IAS using prebypass intraoperative TEE during LVAD

placement is confirmed, and brief but focused reevaluation of the septum should be undertaken at the time of separation from CPB, particularly in instances of atrial or coronary sinus cannulation. We note that a small PFO may remain undetected by TEE unless supplemented by saline contrast and simulated Valsalva's maneuver. The degree of shunting across an IAS defect may be aggravated by chest closure or pleural suction.

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