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Left Ventricular Distention during Pulmonary Artery Venting in a Patient Undergoing Coronary Artery Bypass Surgery

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Prevention of left ventricular distention during cardiac surgery, particularly during ventricular fibrillation and reperfusion is crucial.[1–5] Decompression of the ventricle by venting reduces the risk of left ventricular distention but is not without complications. Venting *via* the ventricular apex or the superior pulmonary vein have been associated with numerous adverse events. Alternatively, venting *via* the pulmonary artery has been reported to be simple, effective and relatively free of complications.[6] We present a case of severe left ventricular distention during venting *via* the pulmonary artery during coronary artery bypass graft surgery. In addition, during cardiopulmonary bypass, our patient demonstrated low pulmonary artery pressures (PAP) in the presence of severe left ventricular distention, although PAP is often monitored as an indicator of left ventricular distention.

CASE REPORT

A 79-yr-old man with a history of mild hypertension and 25 yr of stable exertional angina developed pulmonary edema following 4 h of angina unresponsive to medication. A diagnosis of myocardial infarction was excluded by enzyme analysis and serial electrographic data revealing no change from a baseline pattern of left bundle branch block. Cardiac catheterization demonstrated severe three-vessel disease, an ejection fraction of 0.4–0.45 with diffuse hypokinesis, a left ventricular end-diastolic pressure of 23–24 mmHg, trivial mitral regurgitation, and no pressure gradient across the aortic valve. A supravalvular injection of dye was not used to evaluate aortic regurgitation because the patient had no history or physical findings suggesting this condition. Elective coronary artery bypass graft surgery was scheduled. Preoperatively, the patient's medications included diltiazem, 90 mg three times daily; isosorbide dinitrate 40 mg three times daily; nitroglycerin ointment 1.5 inches four times daily; and diethylstilbestrol 1 mg twice daily. Physical examination was remarkable only for a blood pressure of 164/70 mmHg. Electrocardiography revealed sinus rhythm with left bundle branch block.

Intraoperatively, routine monitoring included radial and pulmonary artery catheters placed with local anesthesia prior to induction of general anesthesia with sufentanil and neuromuscular blockade with pancuronium. The PAP prior to induction was 34/12 mmHg decreasing to 24/11 mmHg with induction. After tracheal intubation, a two-dimensional transesophageal echocardiography probe was placed to permit imaging of the aortic valve, mitral valve, and regional wall motion *via* a view of the left ventricular short axis at the level of the midpapillary muscles. Two-dimensional echocardiography revealed thickened aortic valve leaflets, posterior wall akinesis, and septal and anterior wall hypokinesis. Color flow imaging demonstrated mild aortic valve insufficiency and mild mitral regurgitation. During aortic cross-clamping, the left ventricle was vented *via* the aortic root.

Just before removal of the aortic cross-clamp, the mean arterial pressure was 58 mmHg and mean PAP was 4 mmHg; after clamp removal, mean arterial pressure decreased to 40 mmHg while PAP increased to 42 mmHg. The surgeon was notified of the increased PAP, and visual inspection confirmed distention of the left ventricle. Transesophageal echocardiographic imaging confirmed left ventricular distention. The patient's cardiac rhythm at the time was asystole. The surgeon elected to place a pulmonary artery vent to decompress the heart while the proximal grafts were anastomosed to the aorta. After the pulmonary artery vent was placed, PAP decreased to −6 mmHg while the mean arterial pressure increased to 46 mmHg. Despite the reduction in PAP, the echocardiogram continued to show severe left ventricular distention (fig. 1). The surgeon was requested to place ventricular pacing leads, and after V-pacing was established at a rate of 60, the echocardiogram showed a reduction in left ventricular end-diastolic volume (fig. 2). Mean arterial pressure at this time was 44 mmHg and PAP was −4 mmHg. A brief interruption of pacing was associated with rapid left ventricular distention that resolved with resumption of pacing.

Separation from cardiopulmonary bypass required bolus injections of calcium chloride, 700 mg, and ephedrine, 5 mg, plus a dopamine infusion at a rate of 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Postoperatively, inotropic support was discontinued by day 1, when creatine phosphokinase MB isoenzyme levels peaked at 16.6 U/l, and the electrocardiogram showed no change from the preoperative baseline pattern of left bundle branch block. The patient was discharged on postoperative day 7.

DISCUSSION

Routine venting of the left ventricle during cardiopulmonary bypass remains a controversial issue. Advocates claim that venting preserves the myocardium by preventing wall stress when the myocardium is particularly susceptible to ischemic insult; provides a bloodless surgical field, improving visibility; maintains a flaccid heart, thereby improving surgical exposure; and decreases myocardial rewarming.[7] The transmural pressure gradient during reperfusion may result in redistribution of trans-

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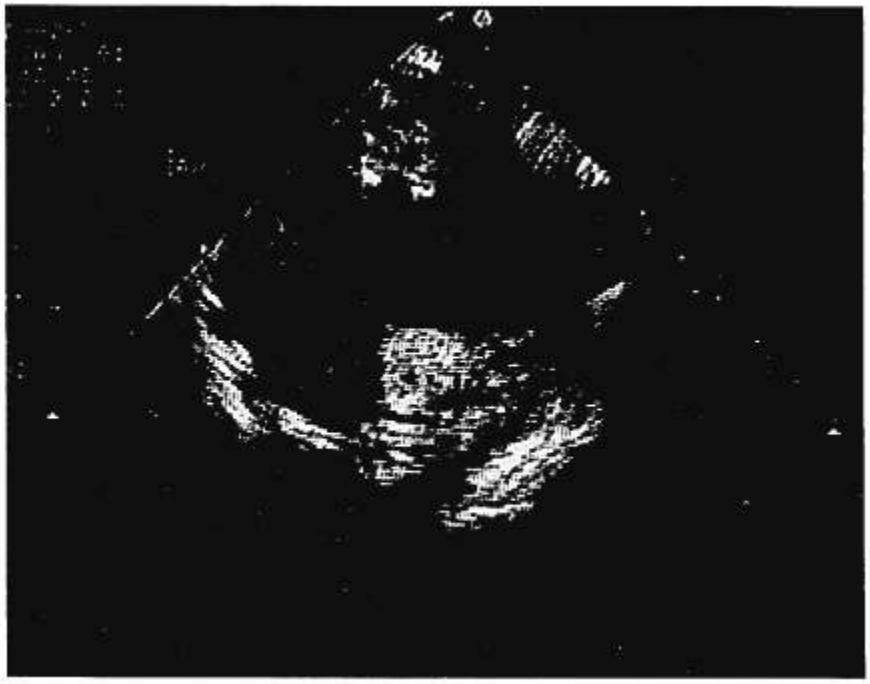

FIG. 1. Two-dimensional transesophageal echocardiographic display of the left ventricular short-axis view at the midpapillary muscle level after placement of the pulmonary artery vent. Pulmonary artery pressure (PAP) is −6 mmHg; mean arterial pressure is 44 mmHg. Despite the low PAP, severe distention is apparent.

mural blood flow.[8,9] Early postischemic perfusion correlates positively with return of ventricular function.[3] Although the use of cardioplegic ischemic arrest has reduced the risks of ventricular distention,[4] even mild ventricular distention during reperfusion can lead to decreased subendocardial perfusion with resultant deterioration in left ventricular performance.[5]

Despite the advantages of avoiding ventricular distention, use of left ventricular vents has been associated with numerous complications. These include myocardial damage[10]; arrhythmias[11]; hemorrhage[11]; left ventricular aneurysms[12,13]; apical dyskinesia[14]; focal contraction abnormalities[15]; mechanical injury of the left ventricle, atrium, and mitral valve[16]; disruption of apical collateral

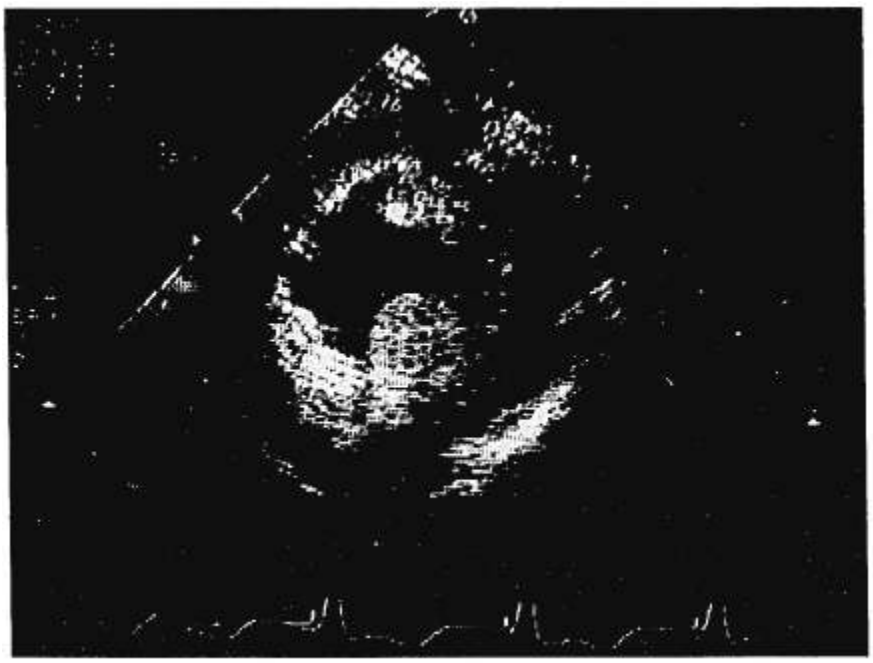

FIG. 2. Two-dimensional transesophageal echocardiographic display of the left ventricular short-axis view at the midpapillary muscle level after establishing ventricular pacing. The distention of the left ventricle is markedly reduced. Pulmonary artery pressure (PAP) is −4 mmHg; mean arterial pressure is 44 mmHg.

vessels[16]; and delayed left ventricular rupture.[17] Perhaps the most widespread concern has been the potential for introducing systemic air, shown to occur in 16% of such patients *versus* 0% of patients without left ventricular vents.[18] These concerns led several investigators to perform cardiac surgery without venting the left ventricle, demonstrating that in most cases cardiac surgery can be performed safely without venting.[11,19–21] However, most cardiac surgeons still insert a vent if the ventricle becomes distended during surgery, especially during the reperfusion.

Venting *via* the pulmonary artery has been reported to be a safe, simple, and indirect technique of left ventricular decompression[6,22,23] that reduces the risk of systemic air emboli,[16,22] myocardial rewarming, and collateral blood flow[23]; results in less washout of cardioplegic solution[23]; and retrieves right heart spillover and bronchial blood flow.[16] Although an early precautionary note advised that left ventricular distention could theoretically occur while venting *via* the pulmonary artery if aortic valve insufficiency and mitral valve competence coexisted,[6] ours is the first report of this occurrence. Aortic insufficiency was neither suspected nor documented in our patient before being detected intraoperatively by transesophageal echocardiography color flow imaging. No aortic valve gradient was apparent preoperatively, and only trivial mitral regurgitation was present, indicating predominant mitral valve competence. Although only mild, the aortic insufficiency resulted in severe ventricular distention of the nonbeating heart during the reperfusion phase of cardiopulmonary bypass.

In addition, our case demonstrates that PAP monitoring alone is not a consistently reliable measure of left ventricular distention during pulmonary artery venting. That is, an initial increase in PAP (from 4 to 42 mmHg) following cross-clamp removal accurately indicated the presence of left ventricular distention, but the subsequent decrease to −6 mmHg following pulmonary artery venting also occurred in the presence of severe ventricular distention. Two-dimensional transesophageal echocardiography detected the persistence of the left ventricular distention despite the reduction in PAP. This suggests that transesophageal echocardiography may be a useful intraoperative monitor for left ventricular distention, particularly during pulmonary artery venting since PAP is no longer a reliable indicator. Two-dimensional transesophageal echocardiography would also detect left ventricular distention during other types of venting as well, although one would also expect the PAP to reflect left ventricular distention with venting *via* the ventricular apex or superior pulmonary vein. It is likely that low, even negative, PAP will be evident despite severe left ventricular distention if active suction is applied to the pulmonary artery in the presence of a competent mitral valve or collapsed pulmonary vascular tree.

Our case also demonstrates that when venting is unsuccessful, the use of ventricular pacing to restore an effective cardiac rhythm during reperfusion can prevent severe left ventricular distention.

In summary, we suggest that 1) severe left ventricular distention can occur despite prophylactic venting *via* the pulmonary artery during cardiac surgery; 2) PAP monitoring alone may not be a reliable indicator of left ventricular distention during pulmonary venting; and 3) intraoperative transesophageal echocardiography may be a more useful monitor of left ventricular distention than PAP during pulmonary artery venting.

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