

Cardiothoracic Anesthesia, Respiration and Airway

Partial inferior vena cava snaring to control ischemic left ventricular dysfunction

[Constriction partielle de la veine cave inférieure pour contrôler une dysfonction ventriculaire gauche]

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Purpose: To describe the hemodynamic and intraoperative transesophageal echocardiographic evaluation of cardiac systolic and diastolic function in a patient undergoing off-pump coronary artery bypass graft (OP-CABG) surgery who developed pulmonary artery hypertension controlled by inferior vena cava (IVC) snaring.

Clinical features: A 63-yr-old man was referred to our hospital for elective OP-CABG surgery. His preoperative ventriculopathy revealed a decreased systolic function (ejection fraction of 35%), and a mild mitral regurgitation. Intraoperatively, after application of the stabilizer and clamping of the diagonal artery, he developed a marked increase in pulmonary artery pressure (PAP) with a decrease in systemic arterial pressure, non responsive to *iv* norepinephrine and nitroglycerin. Transesophageal echocardiographic evaluation revealed a marked decrease in systolic function and presence of a restrictive diastolic filling pattern. Partial IVC snaring decreased the venous return and PAP decreased cardiac chamber dimensions, improved systolic function and improved diastolic filling pattern.

Conclusion: Partial IVC snaring was used successfully to treat a hemodynamically unstable patient with sudden increase in PAP caused by ischemic left ventricular failure during OP-CABG.

Objectif : Décrire l'évaluation hémodynamique et échocardiographique transœsophagienne peropératoire de la fonction cardiaque systolique et diastolique chez un patient opéré pour un pontage aortocoronarien à cœur battant (PAC-CB) et qui a présenté une hypertension artérielle pulmonaire contrôlée par un clampage partiel de la veine cave inférieure (VCI).

Éléments cliniques : Un homme de 63 ans a été dirigé vers notre hôpital pour un PAC-CB non urgent. Sa ventriculopathie préopératoire

a révélé une diminution de la fonction systolique (fraction d'éjection de 35 %) et une régurgitation mitrale légère. Pendant l'opération, après l'application du stabilisateur et le clampage de l'artère diagonale, une hausse marquée de la pression artérielle pulmonaire (PAP) s'est développée, accompagnée d'une baisse de la tension artérielle générale, non corrigée par l'administration *iv* de norépinéphrine et de nitroglycérine. L'échocardiographie transœsophagienne a révélé une baisse marquée de la fonction systolique et la présence d'un tracé de remplissage diastolique restrictif. Le clampage partiel de la VCI a fait baisser le retour veineux et la PAP a réduit les dimensions de la cavité cardiaque, a amélioré la fonction systolique et le tracé de remplissage diastolique.

Conclusion : Le clampage partiel de la VCI a été utilisée avec succès pour traiter l'hémodynamie instable accompagnée d'une hausse soudaine de la PAP causée par une défaillance ischémique ventriculaire gauche pendant le PAC-CB.

OFF-PUMP coronary artery bypass grafting (OP-CABG) is becoming a widely applied procedure, in part because it may avoid cardiopulmonary bypass-related complications.^{1,2} However, in patients with low-to-moderate mitral insufficiency, or in patients with severe ventricular dysfunction, OP-CABG may exacerbate mitral regurgitation or left ventricular (LV) dysfunction and induce pulmonary hypertension and hemodynamic instability.³

To control unexpected pulmonary hypertension during OP-CABG, Dagenais and Cartier³ recently

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reported their technique of intermittent inferior vena cava (IVC) snaring to decrease pulmonary artery pressure (PAP). However, the hemodynamic and echocardiographic documentation of the effects of IVC snaring have never been reported in the literature.

In this report, we describe the hemodynamic and intraoperative transesophageal echocardiographic (TEE) evaluation of cardiac function and mitral valve insufficiency in a patient undergoing OP-CABG who developed pulmonary artery hypertension controlled by IVC snaring.

Case report

A 63-yr-old man with a past medical history of hypertension and a prior inferior wall infarction was referred to our hospital for elective OP-CABG. Preoperative cardiac catheterization revealed a 60% left anterior descending artery, a 80% circumflex artery lesion, a severe stenosis of the diagonal artery, and an occluded right coronary artery. Left ventriculography revealed a decreased systolic function with ejection fraction of 35%. The preoperative electrocardiogram showed a left bundle branch block. No preoperative transthoracic echocardiography was performed. The medication taken by the patient included atenolol and enalapril.

After the induction of anesthesia with midazolam $0.04 \text{ mg}\cdot\text{kg}^{-1}$, sufentanil $1 \text{ }\mu\text{g}\cdot\text{kg}^{-1}$, and pancuronium $0.1 \text{ mg}\cdot\text{kg}^{-1}$, systemic blood pressure was 102/53 mmHg, PAP was 36/20 mmHg, central venous pressure was 11 mmHg, and heart rate (HR) was 69 $\text{beats}\cdot\text{min}^{-1}$. Intraoperative multiplane TEE (Sonos 1000, Hewlett-Packard, Andover, MA, USA) confirmed the decreased systolic function, with akinesia of the infero-basal and postero-basal walls and also documented mild-central mitral regurgitation (1/4) and a patent foramen ovale with a small right to left shunt which remained unchanged during the surgery. LV and right ventricular (RV) end-diastolic dimensions measured at mid-ventricular level in four-chamber view were 6.3 and 3.6 cm respectively. Measurements were taken at end-expiration. Transmitral flow was recorded with the sample volume positioned between the tips of the mitral leaflets. The peak velocity in early diastole (E wave) and at atrial contraction (A wave) were measured. The deceleration time (dt) of the E wave was obtained by extrapolating the initial slope of the E wave to the zero line. The ratio of the peak velocities of the E and A waves was derived. Peak systolic and diastolic pulmonary vein velocities were recorded in the left upper pulmonary vein. We also measured peak velocity of the E and A waves of the tricuspid flow, and derived the E/A ratio. Finally, we measured peak systolic and diastolic velocities in the

hepatic veins. We used standard definitions of diastolic function: normal ($E/A > 1$, $dt < 220 \text{ msec}$, and systolic $>$ diastolic), impaired relaxation ($E/A < 1$, $dt > 240 \text{ msec}$, systolic $>$ diastolic) and restrictive LV filling ($E/A \text{ ratio} > 2$ or 1 to 2 with an E wave $dt < 150 \text{ msec}$, and systolic $<$ diastolic).^{4,5} Initial Doppler examination of the mitral and tricuspid inflow, as well as the left pulmonary vein and hepatic venous flow are shown in Figure 1. The E/A ratio > 1 of the mitral and tricuspid flow with systolic/diastolic ratio of the pulmonary and hepatic venous flow > 1 are compatible with normal right and left diastolic function.⁵

Details of the surgical technique have been published elsewhere.^{6,7} The surgery was performed through a full median sternotomy. Target artery immobilization was achieved through mechanical stabilization (Cor-vasc System; Coronéo Inc., Montreal, QC, Canada).

The posterior descending artery was grafted first, without hemodynamic instability. However, after application of the stabilizer and clamping of the diagonal coronary artery, we observed a marked increase in PAP (42/29 mmHg) and central venous pressure (23 mmHg) with a decrease in systemic arterial pressure (86/55 mmHg) despite a flush drip of phenylephrine ($10 \text{ mg}\cdot 100 \text{ mL}^{-1} \text{ NaCl } 0.9\%$), and a norepinephrine infusion of $10 \text{ }\mu\text{g}\cdot\text{min}^{-1}$ before the IVC snaring. A nitroglycerine infusion of $10 \text{ }\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ also failed to decrease the PAP. The HR remained stable at 67 $\text{beats}\cdot\text{min}^{-1}$. At this time, TEE examination revealed a decrease in systolic function, with akinetic inferior and posterior walls, and hypokinetic anterior and antero-lateral walls. The LV and RV dimensions measured at mid-ventricular level in four-chamber view were 6.4 and 3.7 cm respectively. Colour Doppler examination of the mitral valve showed moderate mitral valve regurgitation (2/4; Figure 2). The pulsed Doppler examination of the mitral valve was compatible with a restrictive diastolic filling pattern. The E/A ratio of the tricuspid valve was < 1 , as was the systolic/diastolic ratio of the hepatic venous flow, which is compatible with abnormal RV diastolic filling pattern (Figure 2).

As previously described,³ the surgeon passed a standard umbilical tape around the IVC to correct pulmonary hypertension. The IVC was partially snared until PAP returned to basal values (18–20 mmHg of diastolic PAP), while we observed an increase in systemic pressure to 110/60 mmHg, with a HR of 60 $\text{beats}\cdot\text{min}^{-1}$. The phenylephrine infusion was stopped and norepinephrine gradually decreased to $5 \text{ }\mu\text{g}\cdot\text{min}^{-1}$. As shown by TEE examination, the snaring of the IVC resulted in a significant improvement of the LV systolic function with normalization of regional wall motion of the anterior and antero-lateral walls, a decrease in LV

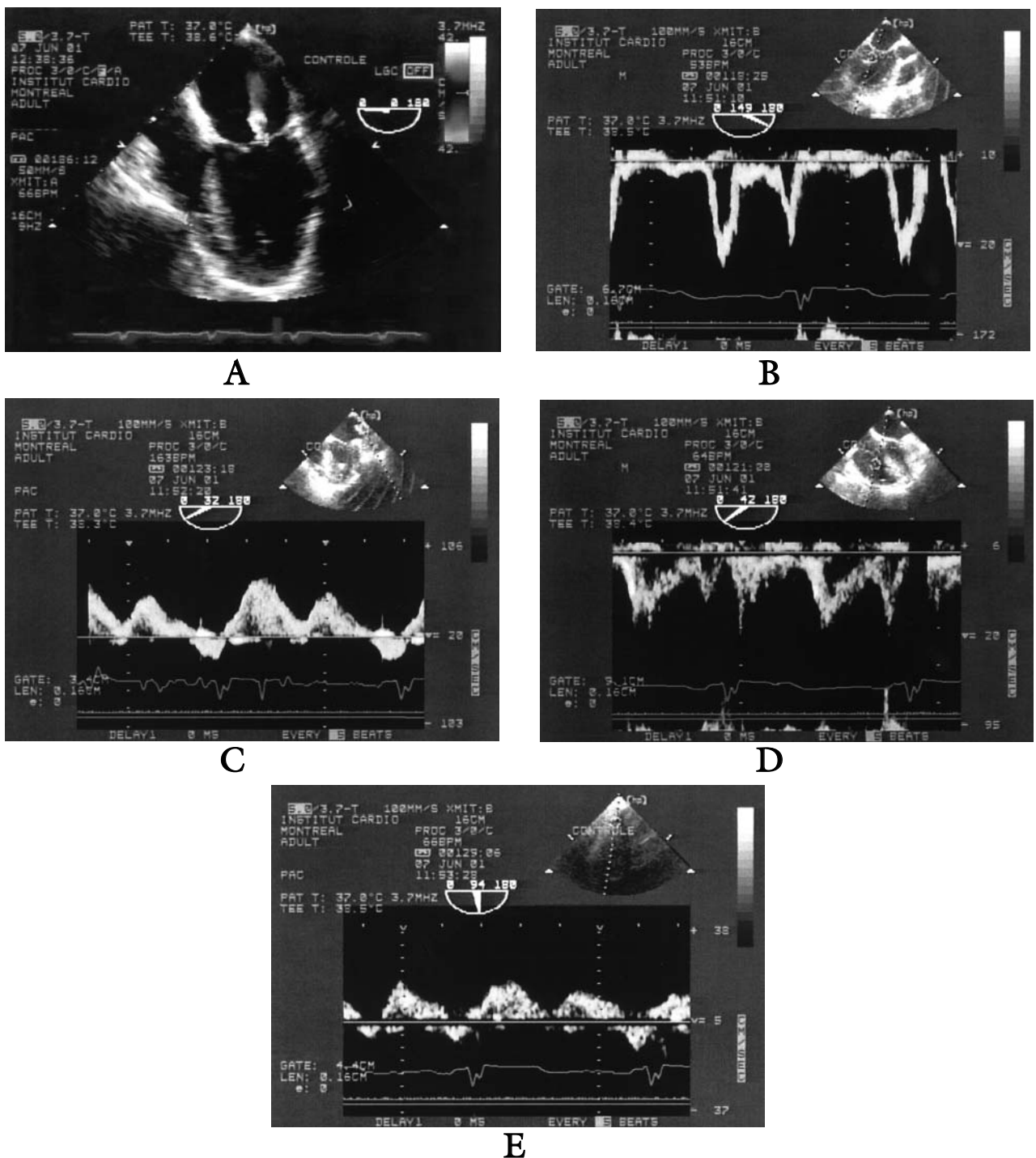


FIGURE 1 Transesophageal echocardiography findings after anesthetic induction. Findings were compatible with a normal right and left diastolic function. A) Mild mitral regurgitation; B) mitral flow: E and A wave velocities were 86.2 and 73.1 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 1.18); E wave deceleration time was 200 msec; C) pulmonary venous flow systolic and diastolic wave velocities were 60.9 and 45.2 $\text{cm}\cdot\text{sec}^{-1}$ respectively; D) tricuspid flow E and A wave velocities were 37.8 and 37 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 1.02); E) hepatic flow systolic and diastolic wave velocities were 15.3 and 11.3 $\text{cm}\cdot\text{sec}^{-1}$ respectively.

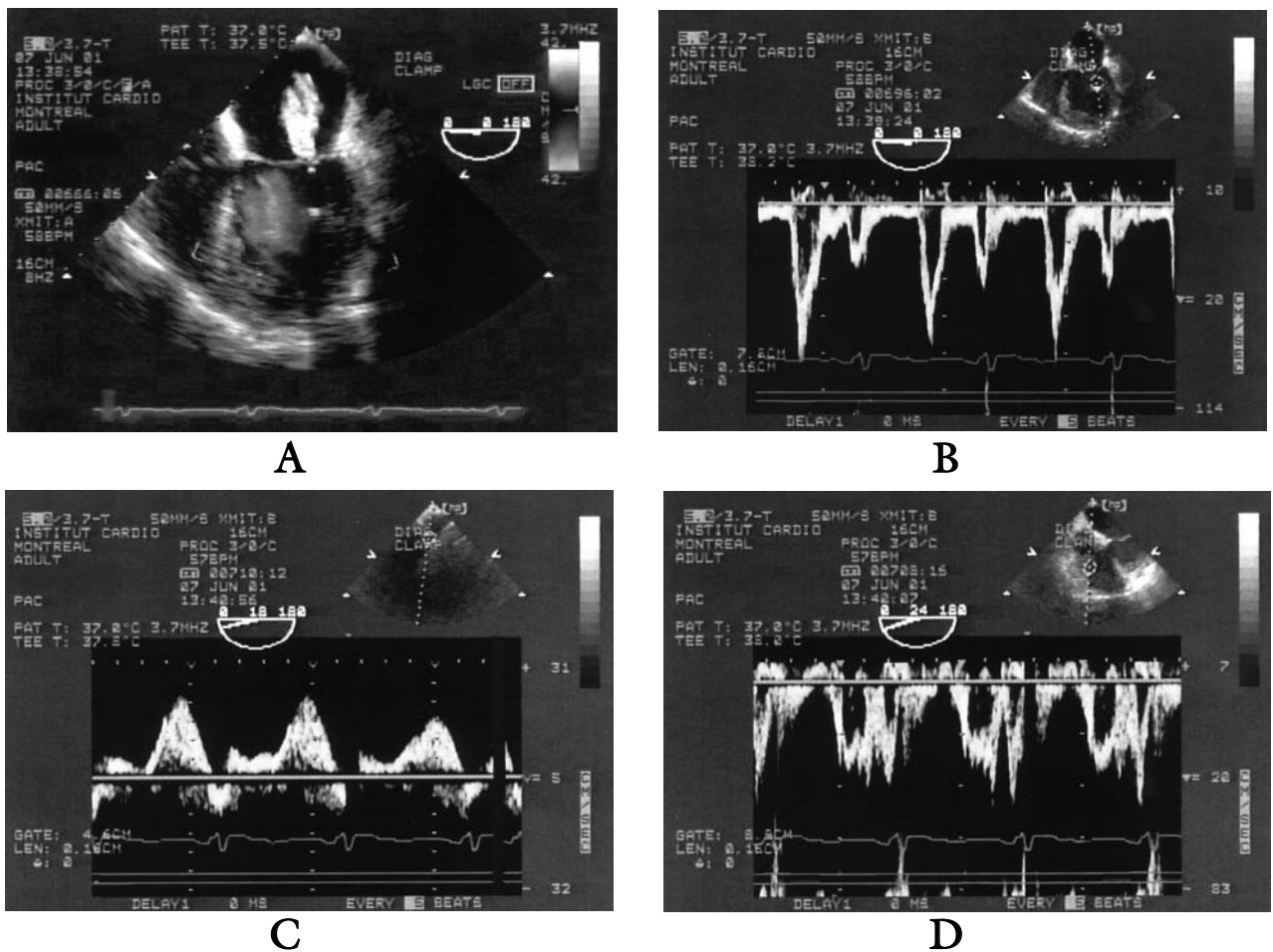


FIGURE 2 Transesophageal echocardiography findings after stabilization and clamping of the diagonal coronary artery. Hepatic flow values were missing. Findings were compatible with a restrictive left ventricular diastolic pattern and a relaxation anomaly of the right ventricular diastolic function. A) moderate mitral regurgitation; B) mitral flow E and A wave velocities were 81.6 and 30.5 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 2.68). The E wave deceleration time was 190 msec; C) pulmonary venous flow systolic and diastolic wave velocities were 8.99 and 23.6 $\text{cm}\cdot\text{sec}^{-1}$ respectively; D) tricuspid flow E and A wave velocities were 31.7 and 40.8 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 0.78).

and RV dimensions to 5.5 and 2.9 cm respectively, a decrease of the mitral regurgitation (1/4) and a return to a normal RV and LV diastolic filling pattern (Figure 3). Interestingly, we observed the disappearance of the triphasic components of the hepatic venous flow, which adopted a rather continuous flow pattern during partial IVC snaring, likely because of the lack of transmission of atrial wave pressure to the hepatic vein. Due to the hemodynamic instability observed during IVC snaring, when vasopressor, vasodilator, and fluid administration were required, we did not measure cardiac output during this busy period. In addition, fractional area change was not measured because the mid-papillary transgastric

short axis view was unobtainable during OP-CABG.

The left anterior descending and circumflex coronary arteries were later grafted without major hemodynamic variations. After completion of the last anastomosis, the IVC was gradually unclamped until reestablishment of normal hemodynamics. TEE findings at the end of the surgery are shown in Figure 4 with normalization of the LV diastolic filling pattern, persistence of an abnormal RV diastolic filling pattern, and a mild mitral regurgitation. The LV and RV dimensions were 5.1 and 2.6 cm respectively. The postoperative period was uneventful and the patient was discharged from the hospital five days later.

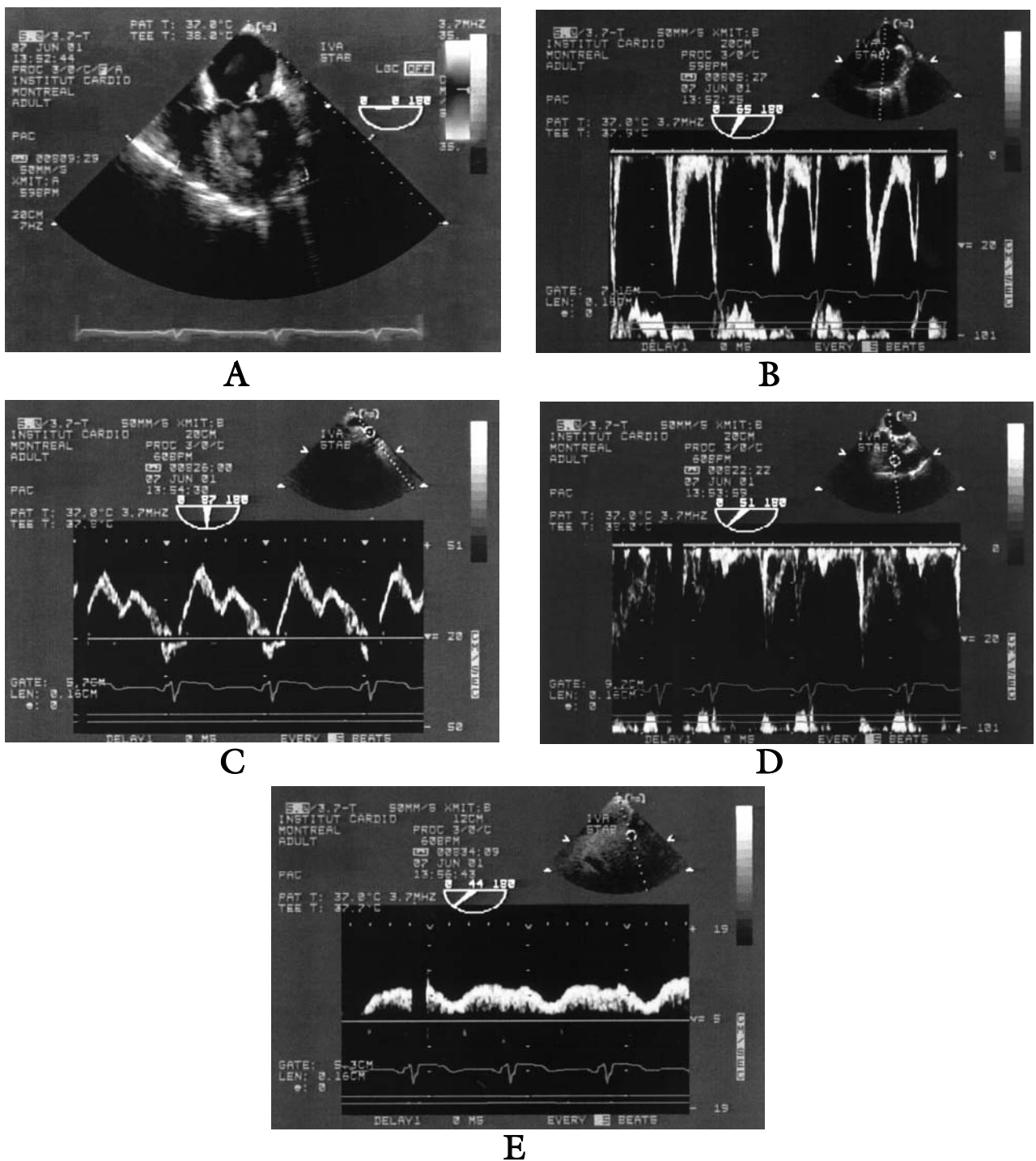


FIGURE 3 Transesophageal echocardiography findings during inferior vena cava snaring. Findings were compatible with a return to normal right and left diastolic functions. A) mild mitral regurgitation; B) mitral flow E and A wave velocities were 65.8 and 57.3 cm·sec⁻¹ respectively (E/A ratio = 1.19). The E wave deceleration time was 188 msec; C) pulmonary venous flow systolic and diastolic wave velocities were 39.1 and 27.0 cm·sec⁻¹ respectively; D) tricuspid flow E and A wave velocities were 51.5 and 33.6 cm·sec⁻¹ respectively (E/A ratio = 1.53); E) hepatic flow adopted a rather continuous flow, with absence of systolic and diastolic waves.

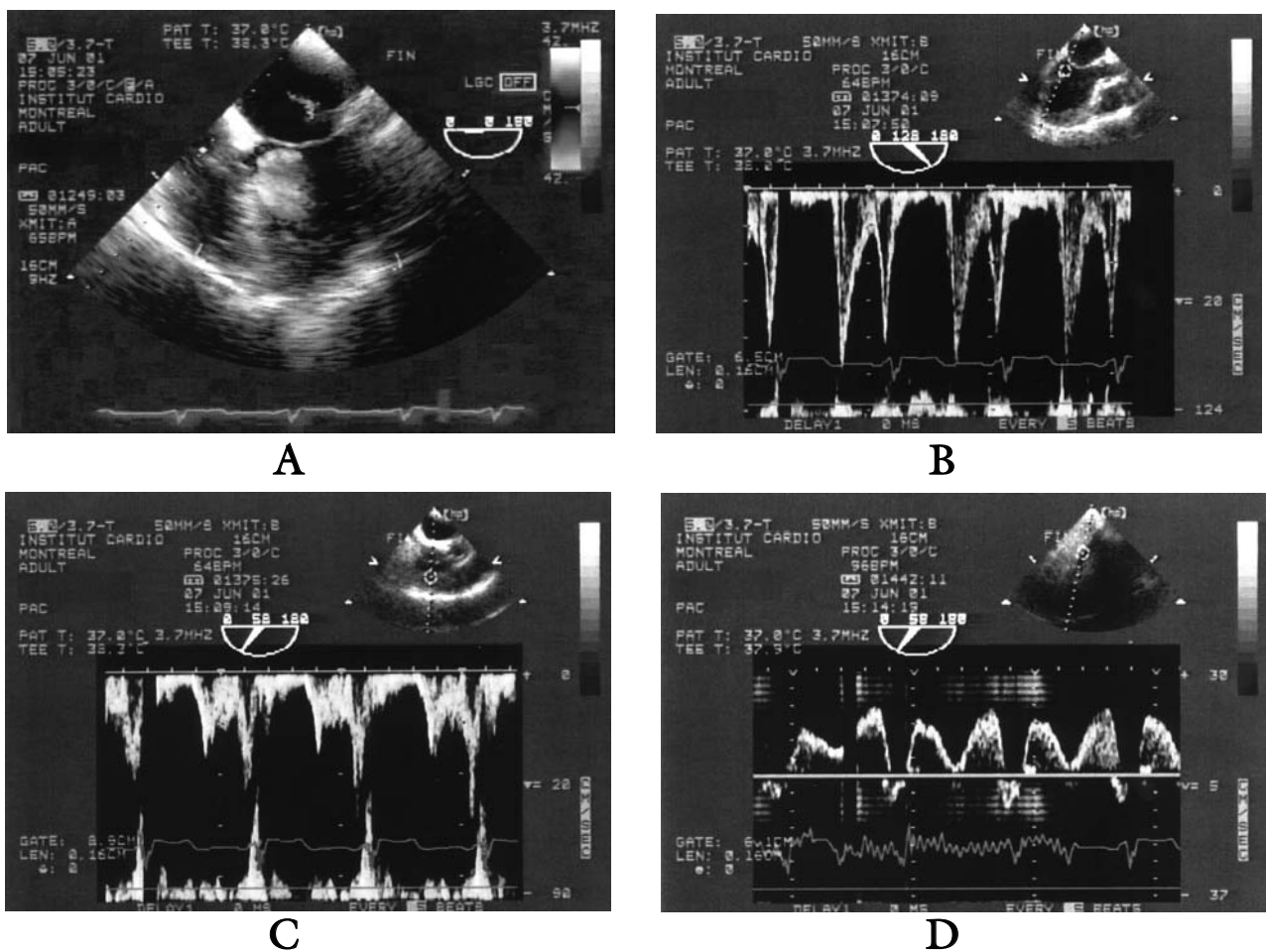


FIGURE 4 Transesophageal echocardiography findings at the end of surgery. Pulmonary venous flow values were missing. Findings were compatible with normal left diastolic function and persistence of a relaxation abnormality of the right ventricular diastolic function. A) mild-mitral regurgitation; B) mitral flow E and A wave velocities were 91.9 and 82.2 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 1.14); C) tricuspid flow E and A wave velocities were 29.8 and 43.4 $\text{cm}\cdot\text{sec}^{-1}$ respectively (E/A ratio = 0.69); D) hepatic flow systolic and diastolic wave velocities were 13.7 and 16.8 $\text{cm}\cdot\text{sec}^{-1}$ respectively.

Discussion

This case report illustrates that OP-CABG surgery may be difficult to tolerate for patients with low to moderate mitral insufficiency or LV dysfunction. In this patient, stabilization of the heart and clamping the diagonal coronary artery precipitated myocardial ischemia, as documented by new regional wall motion abnormalities, leading to myocardial dysfunction. In the face of unchanged end-diastolic dimension, change in LV diastolic function may explain the observed increase in LV end-diastolic pressure, pulmonary capillary wedge pressure, and PAP. Elevated LV end-diastolic pressure further decreased coronary

perfusion pressure, leading to additional deterioration in systolic function. An abnormal RV diastolic pattern was also observed, which may be partly explained by pulmonary hypertension, RV ischemia, and ventricular interdependence.

In this patient with acute ischemic LV dysfunction, temporary partial IVC snaring decreased the venous return, which was followed by a decrease in RV and LV dimensions and PAP. This decrease in preload likely resulted in decreased LV filling pressure, which is reflected by the normalization of diastolic filling pattern as measured with the decrease in E/A ratio. The improved coronary perfusion pressure may explain the

increase in LV systolic function, stroke volume and finally arterial pressure. An additional explanation is that decreased LV end-diastolic dimension reduces afterload via the law of Laplace, thus decreasing myocardial oxygen demand. The combination of decreased demand and improved supply probably explains the benefit of reducing venous return and the decrease in mitral regurgitation. This is supported by the rapid decrease in the need for vasopressor support after IVC snaring along with the improved systolic function. The temporary IVC snaring technique enabled the surgeon to complete the OP-CABG procedure while maintaining an acceptable hemodynamic state and prevented the use of cardiopulmonary bypass. After completion of the last coronary graft, the LV systolic and diastolic functions and the mitral regurgitation returned to the pre-ischemic state. Although RV dimension and systolic function returned to pre-ischemic state, there was a persistent RV diastolic dysfunction.

As a limitation of Doppler measurements, we must consider that mitral flow and pulmonary venous flow are also affected by other factors besides diastolic function, including loading conditions, which change during OP-CABG procedures. In addition, mitral flow measurements can be difficult to measure when the heart is mobilized during OP-CABG. In this patient, we measured mitral flow at angles varying between 0 and 160° to obtain maximal values. In the context of OP-CABG, we do not know whether the effects of IVC snaring on mitral and pulmonary venous flow patterns are reproducible or not. More patients should be studied to assess the reproducibility of our findings. Also, potential adverse effects on liver, bowel, and renal blood flow have not been evaluated, particularly during prolonged occlusion.

In conclusion, this case illustrates the usefulness of temporary partial IVC snaring in reducing acute increases in PAP secondary to LV failure and in maintaining stable hemodynamics by normalization of systolic function and diastolic filling pattern. This procedure should not be seen as a surrogate for mitral valve surgery and should not interfere with the decision to repair or replace a mitral valve. However, it may enable the surgeon to complete the OP-CABG procedure or give additional time to stabilize the hemodynamics before considering cardiopulmonary bypass. More patients should be studied to evaluate its safety and confirm the reproducibility of the hemodynamic effects.

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