# Intraoperative direct measurement of left ventricular outflow tract gradients to guide surgical myectomy for hypertrophic cardiomyopathy

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**Objectives:** We sought to summarize our recent experience with intraoperative monitoring for management of patients undergoing surgical myectomy for hypertrophic obstructive cardiomyopathy with emphasis on dynamic left ventricular outflow tract obstruction. We also analyzed the impact of these data on surgical decision-making and adequacy of septal myectomy.

**Methods:** We retrospectively analyzed the medical records of 198 patients who underwent transaortic septal myectomy and evaluated baseline and provoked left ventricular outflow tract gradients obtained by Doppler echocardiography and by direct measurement of pressures in the left ventricle and aorta.

**Results:** After induction of anesthesia before myectomy, left ventricular outflow tract obstruction, assessed by direct measurement, was less than the gradient documented by preoperative Doppler echocardiography in 119 patients (60%) (41 ± 31 vs 76 ± 40 mm Hg; $P < .001$). In 75 patients (38%), the obstruction was more severe (64 ± 32 vs 35 ± 31 mm Hg; $P < .001$); 4 patients (2%) had similar left ventricular outflow tract gradients. After myectomy, left ventricular outflow tract gradient decreased markedly (49 ± 33 vs 4 ± 8 mm Hg [$P < .001$] by direct measurement; 59 ± 42 vs 4 ± 6 mm Hg [$P < .001$] by transesophageal echocardiography). Cardiopulmonary bypass was resumed for more extensive myectomy in 8 (4%) patients because of a persistent residual left ventricular outflow tract gradient of 33 ± 14 mm Hg. Of note, for 78 patients (39%) intraoperative Doppler echocardiographic assessment of left ventricular outflow tract gradient was technically inadequate.

**Conclusions:** Direct intraoperative measurement of pressures in the left ventricle and aorta provides important hemodynamic data in addition to intraoperative transesophageal echocardiography findings. This information assists the surgeon in defining the extent of myectomy. (J Thorac Cardiovasc Surg 2011;142:53-9)



Surgical myectomy is the preferred treatment for left ventricular (LV) outflow tract (LVOT) obstruction due to symptomatic hypertrophic obstructive cardiomyopathy (HOCM) when medical therapy proves unsuccessful.[1] LVOT obstruction in HOCM is a dynamic process. Changes in myocardial contractility, loading conditions, and heart rate substantially alter LVOT gradients.[2] The magnitude and potential impact of these changes on surgical decision-making within the operating room have not been well described. It is not uncommon for patients with severe symptomatic LVOT obstruction (> 30 mm Hg) to have a lower gradient under anesthesia than preoperatively; in contrast, patients with moderate LVOT gradients preoperatively may demonstrate severe obstruction in the operating room. LVOT obstruction can be ameliorated by anesthesia (eg, potent narcotics such as fentanyl induce bradycardia, and volatile anesthetics reduce contractility). LVOT obstruction also may be reduced by volume infusion and patient positioning (Trendelenburg) or, alternatively, it may be accentuated by decreased venous return due to anesthetic-induced vasodilation or hypovolemia common in patients who are fasting before surgery.

In this report we summarize our experience in the management of patients with HOCM, with an emphasis on the perioperative monitoring of the dynamics of LVOT obstruction.

## MATERIALS AND METHODS

### Study Patients

After institutional review board approval, we searched our clinic database for patients 18 years of age or older who had transaortic septal myectomy for symptomatic HOCM between 2004 and 2008. Of 549 patients identified, 198 consecutive patients had consented to participate in research and had medical records containing all the necessary information for the study: preoperative and intraoperative echocardiography reports, and scanned tracings of LV and aortic pressures measured directly in the operating room.

### Operative Technique

The standard surgical treatment of LVOT obstruction was transaortic septal myectomy.[3,4] The septum was exposed through an oblique

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**Abbreviations and Acronyms**

CPB = cardiopulmonary bypass
HOCM = hypertrophic obstructive cardiomyopathy
LV = left ventricle, left ventricular
LVOT = left ventricular outflow tract
MR = mitral regurgitation
MV = mitral valve
PVC = premature ventricular contraction
TEE = transesophageal echocardiography
TTE = transthoracic echocardiography

aortotomy, and an initial upward incision was made in the septal muscle at the nadir of the right aortic sinus. This incision was turned leftward to excise muscle over the anterior leaflet of the mitral valve (MV). The septal excision was deepened and lengthened toward the apex of the heart past the contact lesion (''scar'') on the endocardial surface. Operations were carried out with normothermic cardiopulmonary bypass (CPB), and hypothermic antegrade blood cardioplegia was used for myocardial protection.



## Anesthesia

Intraoperative management included standard monitoring (ie, electrocardiography, pulse oximetry, blood pressure cuff, direct arterial blood pressure, temperature), peripheral nerve stimulation, pulmonary artery catheterization, and transesophageal echocardiography (TEE).[5] Graphical trends of all principal hemodynamic parameters were displayed in real time and on the network computer at 1-minute intervals. For study purposes, we recorded hemodynamic parameters first after induction and then simultaneously with LVOT echocardiographic and direct measurements: (1) before myectomy 2 to 5 minutes before going on bypass; and (2) after myectomy 5 to 10 minutes postbypass after hemodynamic stabilization.

All patients had general anesthesia that consisted of administration of benzodiazepines (midazolam), opioids (fentanyl), volatile anesthetics (isoflurane), and muscle relaxants (pancuronium). We used calcium chloride post-CPB; if blood pressure was low after adequate volume replacement (mean arterial pressure, < 60 mm Hg), we administered vasoactive medications such as phenylephrine or vasopressin to restore normal systemic vascular resistance. Epinephrine was reserved for rare instances of poor cardiac performance and hypotension unresponsive to vasopressors. If atrioventricular block or bradycardia persisted after reperfusion, we used dual-chamber or atrial pacing. Electrolyte balance was controlled throughout surgery; we evaluated the concentration of potassium at least three times (postinduction, on CPB, and at closure) to maintain it within the reference range (3.6–5.2 mmol/L).

## LVOT Measurements

For echocardiographic evaluation of the LVOT obstruction, the transducer was positioned as parallel as possible to the LVOT jet to obtain maximal Doppler velocities. The Doppler-derived LVOT gradient was estimated at maximal velocity of blood flow through the LVOT during ventricular contraction (ie, the maximal instantaneous gradient) (Figure 1). Doppler velocity across the aortic valve was converted into a pressure gradient between the LV and the aorta by the modified Bernoulli equation ($\Delta p = 4v^2$). The Doppler maximal instantaneous gradient by definition was expected to be higher than the peak-to-peak gradient at the same point in the same cardiac cycle (Figure 1).

Patients with hypertrophic cardiomyopathy have an initial higher aortic pressure at very early systole due to unimpeded flow, which is then followed by a decrease in pressure and a gradual increase in pressure gradient. Thus, the maximal instantaneous gradient by Doppler echocardiography correlates best with the maximal gradient obtained by cardiac catheterization. Although this gradient may be the most accurate measure of the degree of obstruction, in clinical practice in the operating room, peak-to-peak systolic gradient is the easiest to obtain and provides reliable information with minimal underestimation of the true LVOT gradient.

Preoperatively, each of the 198 patients underwent comprehensive 2-dimensional and Doppler transthoracic echocardiography (TTE). Measurement of the LVOT gradient was carried out by continuous-wave Doppler interrogation of the LVOT from the apical window.

In the operating room, the grade of LVOT obstruction was evaluated twice: before myectomy 2 to 5 minutes before going on bypass and after

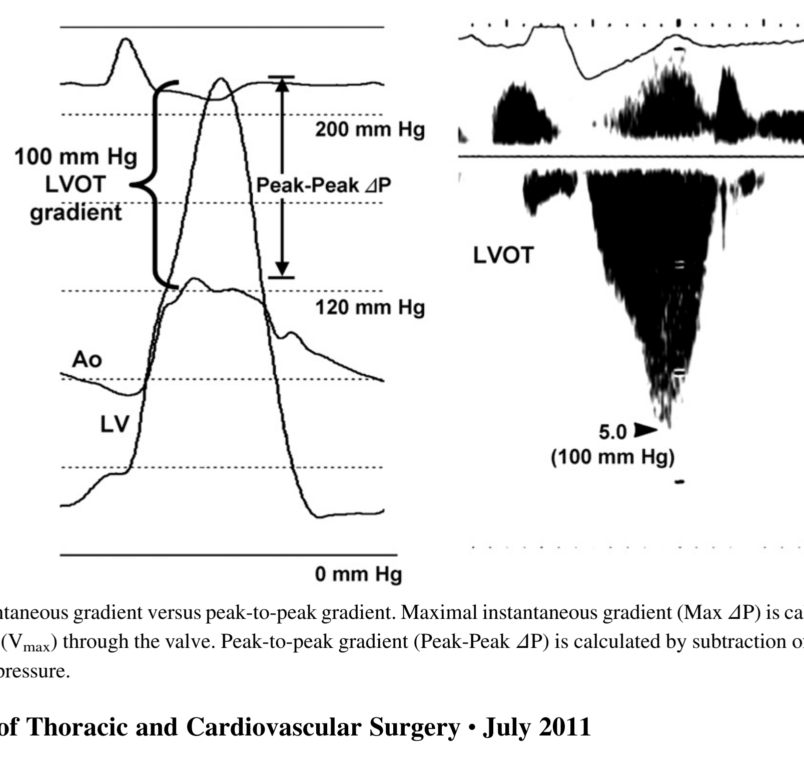


**FIGURE 1.** Maximal instantaneous gradient versus peak-to-peak gradient. Maximal instantaneous gradient (Max $\Delta P$) is calculated at the moment of maximal Ao (aortic) jet velocity ($V_{max}$) through the valve. Peak-to-peak gradient (Peak-Peak $\Delta P$) is calculated by subtraction of maximal left ventricular (LV) pressure from maximal Ao pressure.