Commentary: Now you see me, now you don’t—the peek-and-boo left ventricular outflow obstruction gradient

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Septal myectomy provides proven surgical therapy for left ventricular outflow obstruction (LVOT) associated with hypertrophic cardiomyopathy (HCM),\(^1\) with reduction of symptoms and normalization of life expectancy.\(^2\) However, outflow tract obstruction in HCM is not always evident, as only 10% of patients afflicted with the disease require surgical intervention. Within this cohort, the dynamic nature of the obstruction often poses a diagnostic challenge, and clinical outcomes after myectomy of patients with only provokable gradients are unknown. In the current issue of the Journal, Cui and colleagues\(^3\) from the Mayo Clinic present a large cohort of patients with HCM who underwent surgical myectomy for latent outflow tract obstruction defined as a provoked LVOT gradient of greater than 30 mm Hg. Although these patients had decreased septal thickness and low baseline gradients, their provoked gradients were impressively reduced with surgery, as was their New York Heart Association functional class.

The complex interplay of anatomic and functional factors that contribute to LVOT obstruction in HCM is often difficult to tease out in the symptomatology of patients with HCM.\(^4\) Factors as subtle as degree of myocardial fibrosis may have an influence on LVOT obstruction.\(^5\) Rarely, dynamic LVOT obstruction may be present without overt diagnosis of HCM due to a sigmoid septum, prominent papillary muscles, or concentric LV hypertrophy.\(^6\) Lack of a baseline gradient in symptomatic patients, however, should not dull the diagnostic senses, as emphasized by the current authors. The dynamic nature of LVOT obstruction in patients with HCM may not infrequently lead to intraoperative consternation when transesophageal echocardiographic assessment under general anesthesia does not reveal significant outflow obstruction, systolic anterior motion (SAM), or mitral regurgitation that were all visualized on preoperative transthoracic echocardiography. In 293 patients with HCM undergoing septal myectomy, Esayes and colleagues\(^7\) reported an almost 20-mm Hg difference in LVOT gradients between transthoracic echocardiography and intraoperative transesophageal echocardiography. In patients who had a resting preoperative gradient, 41.6% demonstrated no or mild obstruction after induction of anesthesia. Provocative testing with inotropic agents, pre- and postmyectomy, unmasked the obstruction and provided guidance for adequacy of resection. Similar observations were reported by Bonanno and colleagues\(^8\) with 43% of patients with HCM and LVOT obstruction revealing only mild gradients (<30 mm Hg) after induction of anesthesia. Again, dobutamine testing provided clarity as “normal” intraoperative resting gradients postmyectomy give little comfort for therapeutic success. In the current study, the patients with latent obstruction had a median provocative intraoperative gradient of 96 mm Hg, which was essentially resolved with myectomy.

CENTRAL MESSAGE
Symptomatic hypertrophic cardiomyopathy patients without baseline obstruction but with a provokable left ventricular outflow tract gradient benefit from septal myectomy.

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It is noteworthy that great majority of patients with latent obstruction who underwent myectomy demonstrated SAM with provocative maneuvers. There was a small subset who required additional transapical myectomy due to predominantly mid-ventricular obstruction. The unmasking of SAM is an important diagnostic clue as even patients with latent obstruction and relatively thin septa benefit from resection as much as those with baseline gradients. In the past, patients with septal thickness of less than 18 mm were not recommended for myectomy and usually received a mitral prosthesis, a strategy that would have been of disservice to majority of the latent patients presented in the current study. The latent cohort patients did, however, more frequently require mitral valve repair, although the preoperative burden of mitral regurgitation was significantly lower in that group. These data suggest that abnormalities of the mitral apparatus may also modulate latent obstruction.

The presented data behoove the clinician to dig deeper in patients with HCM with disproportionate symptomatology to the level of LVOT obstruction. Provocative maneuvers and inotropic agents serve this purpose well, both pre- and intraoperatively, and other methods are on the horizon. Computer-assisted analysis of baseline beat-to-beat gradient variability may prove useful in characterizing severity of LVOT obstruction and a magnetic resonance imaging–derived anatomic ratio has been shown to identify patients with latent obstruction.

Further technological advancements will surely aid determination of surgical candidacy for these challenging patients. Surgeons and cardiologists alike should remain diligent in using the available diagnostic armamentarium to guide appropriate therapy.

References