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Preintervention imaging and intraoperative management care of the hypertrophic obstructive cardiomyopathy patient

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Abstract

With an estimated overall mortality of less than 1 percent per year, hypertrophic cardiomyopathy, is the most common genetic cardiomyopathy. Intraoperative transesophageal echocardiography is the standard of care for assessing patients with hypertrophic obstructive cardiomyopathy undergoing surgical septal myectomy, allowing surgical planning, intraoperative hemodynamic monitoring, and postprocedural assessment of the repair, including detection of immediate complications. At various phases during surgical septal myectomy, the changing hemodynamic conditions may lead to worsening or improvement in left ventricle outflow tract obstruction by change in preload or afterload, systolic anterior motion of the mitral valve, or sympathetic stimulation. These characteristics represent unique challenges in the management of these patients, requiring a comprehensive understanding of the management of all the conditions required to decrease the left ventricle outflow tract gradient avoiding obstruction, which include the maintenance of sinus rhythm, adequate rate avoiding tachycardia and bradycardia, and avoidance of systemic hypotension preserving preload and afterload, with adequate vasoactive agents. The aim of this review is to summarize the perioperative assessment and management of patients undergoing hypertrophic obstructive myopathy surgery.

Keywords

Hypertrophic obstructive cardiomyopathy, cardiac, transesophageal echocardiography, septal myectomy, mitral systolic anterior motion

Intraoperative preprocedural transesophageal echocardiographic evaluation of hypertrophic obstructive cardiomyopathy (HOCM)

Recommendations

Intraoperative transesophageal echocardiography (TEE) is the standard of care for assessing patients with hypertrophic obstructive cardiomyopathy undergoing surgical septal myectomy.[1–3] Periprocedural TEE is a Class 2B recommendation, it allows surgical planning, intraoperative hemodynamic monitoring and set a baseline to assess postprocedural adequacy of the repair and detection of immediate complications.[1]

Preprocedural TEE examination

Preprocedural TEE provides information about the complex interaction between the interventricular septum (IVS), the mitral valve, the subvalvular apparatus within the left ventricular outflow tract (LVOT). It is paramount to define the anatomy of the LVOT and identify the presence of dynamic obstruction of the LVOT with or without mitral regurgitation (MR) (Figure 1).

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Presence of mitral valve regurgitation. As a result of LVOT flow acceleration through systole, the anterior mitral valve leaflet (AML) is pulled towards the inter-ventricular septum, which is commonly referred to as systolic anterior motion of the mitral valve (SAM), reducing the effective LVOT cross sectional area, increasing the peak pressure gradients (PGs) and almost invariably leading to associated significant MR.[4–6] TEE should confirm SAM as the main mechanism for MR, excluding coexisting intrinsic valvular abnormalities which may be present in 10–20% of the cases, and would change the surgical plan.[3]

As the main mechanism for MR in HOCM, SAM is favored for three main reasons. First, the left ventricle outflow stream is redirected posteriorly and laterally due to the presence of a mid-septal bulge, second, the mitral valve has a high drag coefficient, and third, four specific anatomic characteristics of HOCM patients that may enhance the effect of drag forces. These anatomic features include: 1) anterior displacement of the coaptation point of the mitral valve due to papillary muscles (PM) abnormalities, that places it into the left ventricle outflow stream; 2) elongated chordae tendineae that predisposes for mitral valve displacement by drag forces; 3) elongated AML or presence of residual leaflet, and 4) abnormal connections between the PM and the anterior left ventricular wall which would shift the coaptation point more anteriorly.[7] In this context, MR is caused by poor leaflet coaptation with presence of an interleaflet gap due to inability of the anterior leaflet to meet the posterior. The resulting MR jet is eccentric, posteriorly directed and appears in mid to late systole. The severity of the MR depends on the extent of the interleaflet gap, and it is accepted that the severity of SAM and MR is correlated with the severity of the dynamic PGs. SAM might be worsened by decreased intravascular volume, hypercontractility or low afterload and might be present in clinical scenarios other than HOCM (i.e. post- mitral valve repair or patients on ionotropic drugs).

The best view to define SAM is the mid-esophageal long axis view (ME LAX).[8] In this view, the displacement of AML into the LVOT, can be seen with both 2D imaging, and M-mode. The distance from the mitral leaflet to the septum or the duration of the mitral leaflet-septal contact quantify the severity of SAM as follows: a) Mild: SAM-septal distance >10 mm or AML motion without encroachment in the LVOT, b) Moderate: SAM-septal distance <10 mm or AML motion with encroachment in the LVOT or brief mitral leaflet-septal contact, c) Severe: prolonged SAM-septal contact, lasting more than 30% of systole. By color Doppler, a mosaic pattern in LVOT indicating the presence of turbulent flow, and eccentric posteriorly directed MR at mid-late systole, can be determined in HOCM patients. When the MR jet is not posteriorly directed, it is crucial to search for intrinsic mitral valvular abnormalities, especially in central or anterior MR jets.[1,9]

Anatomic characteristics of the LVOT. The three main anatomic structures that contribute to SAM are: the IVS thickness, the length of mitral leaflets and abnormalities of the subvalvular apparatus.[10,11]

Interventricular septum. The use of TEE is key to describe the extent and location of septal thickening and thus to guide the extension of surgical resection. The anterior IVS is the target resection point in surgical septal myectomy. Since the septal bulge redirects the left ventricle outflow posteriorly and laterally contributing to SAM, the objective of the septal resection is to redirect this outflow more anteriorly and medially. The proximal resection point should be away from the aortic annulus to prevent aortic valve dysfunction, to preserve atrioventricular (AV) node and to avoid lesions of the membranous portion of the septum, which may lead to ventricular septal defects. The distal myectomy resection point should be extended beyond the contact point, between AML and the IVS, to effectively redirect the flow more anteriorly and medially.[1,7,12,13]

Despite the anterior IVS anatomy, minimal LVOT area and severity of the LVOT obstruction (LVOTO) can be well evaluated by two-dimensional echocardiography, if available, real time three-dimensional (3D) echocardiographic acquisition may improve accuracy of septal measurements by better positioning a plane perpendicular to the septum, LVOT area or obstruction, by using dynamic rendered images and multiplane reconstruction.[14,15]

Useful measurements to guide resection are measured at end-diastole and include the maximal septal thickness, distance of maximum thickness from the aortic annulus, location of the endocardial fibrous plaque (friction or impact lesion) and apical extent of the septal bulge.[1]

Anterior mitral valve leaflet. The presence of elongated mitral valve leaflet may contribute to SAM and may require surgical correction. The AML (A2 scallop) is best measured in the ME LAX view, during diastole and using zoom mode. The measurement must be done from the aortic valve annulus to the tip of the AML. It is important to include the entire leaflet, especially the portion after the coaptation point, since this is the part that will be pushed into the LVOT. 3D TEE may provide more precise identification of the A2 scallop. An AML >16 mm/m^2 or >30 mm is considered elongated and more prone to produce LVOTO in patients with HOCM, and a surgical correction may be considered.[2,13] The residual length of the AML of the mitral valve beyond the coaptation point does not participate in the coaptation and may protrude into the LVOT causing obstruction.[2]

Subvalvular apparatus. The analysis of the subvalvular apparatus including the PM and the chordae tendineae must also be part of the preprocedural TEE. Patients with

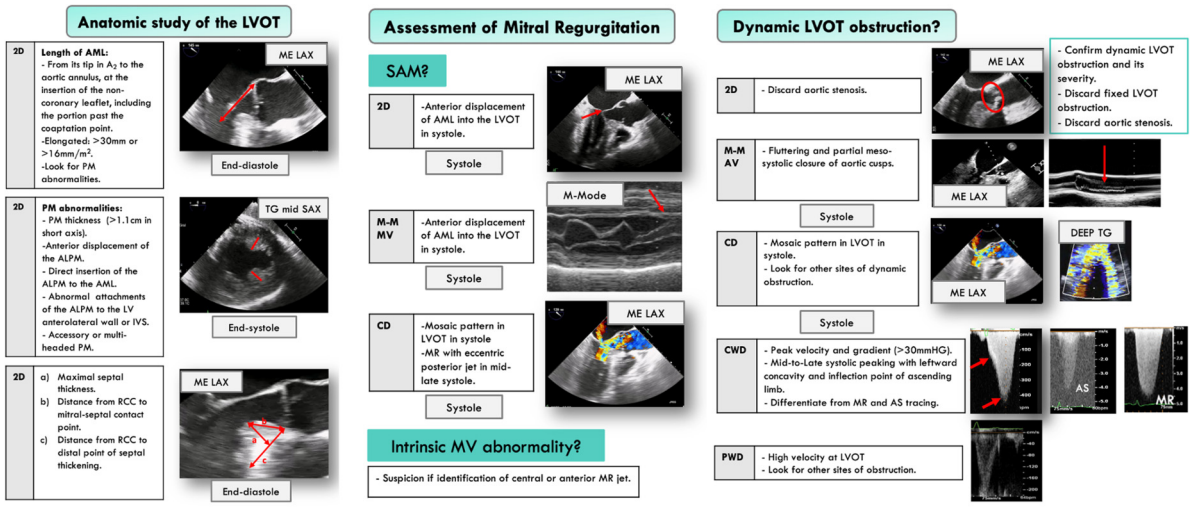

Figure 1. Preprocedural TEE examination in HOCM patients. *AML: anterior mitral valve leaflet, ALPM: antero-lateral PM, CD: Color-Doppler, CWD: continuous wave Doppler, PWD: pulsed wave Doppler, LVOT: left ventricle outflow tract, ME: mid-esophageal, M-M: M-mode, MR: mitral regurgitation, MV: mitral Valve, PM: papillary muscle, RCC: right coronary cusp, SAM: systolic anterior motion of the MV, SAX: short-axis, TG: trans-gastric.*

Figure 1. Preprocedural TEE examination in HOCM patients. TEE: transesophageal echocardiography; HOCM: hypertrophic obstructive cardiomyopathy.

HOCM may have PM abnormalities that may contribute to the development of LVOT and/or mid-cavitary obstruction. This has important therapeutic implications because, if not identified, they can be the cause of residual obstruction after septal myectomy.[2]

The most common PM abnormalities are: 1) anterior displacement of the anterolateral papillary muscle (ALPM) with respect to the IVS; 2) abnormal attachments of the ALPM to the septum or anterolateral wall; 3) direct insertion, without chordae, of ALPM into the anterior leaflet; 4) PM thickening (> 1.1 cm in short axis view), that may predispose to anterior displacement; 5) multi-headed or accessory PM.[10,16–19] These variations may cause mid-cavitary obstruction and can predispose to SAM by increasing the overlap between the left ventricular outflow stream and the mitral valve, due to a shift of the subvalvular apparatus and mitral leaflets anteriorly.

The PM are best evaluated in end-systole using the mid-esophageal commissural and 2 chamber (ME 2CH) views, as well as in the transgastric 2 chambers (TG 2CH) and transgastric mid-papillary short axis views (TG mid SAX).[9] 3D imaging may also help to identify these abnormalities.[19]

Dynamic LVOTO features

Dynamic LVOTO is defined as the presence of obstructive gradient during systole. The main goal of surgical correction is the elimination of systolic obstruction and pressure gradient, and is most closely associated with amelioration of patients' symptoms. The longer the mitral valve stays in contact with the septum, the higher the pressure gradient (PG).[7] Although SAM is the most frequent cause of dynamic LVOTO in hypertrophic cardiomyopathy, in up to 20% of the patients this obstruction is independent of SAM, and is due to anomalous insertion of the PM directly into the anterior mitral leaflet or mitral valve prolapse, calcification or fibrosis.[7,20]

Preprocedural TEE aims to confirm and assess the severity and location of dynamic LVOTO, ruling out the presence of different locations of LVOTO (subaortic, midcavitary or apical), including the presence of fixed LVOTO and aortic stenosis. Using the ME LAX view in M-mode through the aortic valve, may identify a fluttering and partial mid-systolic closure of aortic cusps. This finding is caused by the proximal obstruction to the aortic leaflets that decreases aortic flow and driving forces that keep leaflets open. In this view, color Doppler helps to identify the site of obstruction by showing a mosaic pattern in the areas with turbulent flow. The deep transgastric view is used to localize the site and severity of obstruction. Color Doppler and pulse-wave Doppler help to identify the location of obstruction by showing a mosaic pattern and high velocities respectively. Continuous-wave Doppler (CWD) through the LVOT allows the measure of LVOT peak velocity and PG. A resting PG ≥ 30 mmHg, or if < 30 mmHg at rest but ≥ 30 mmHg with provocation (latent obstruction), suggests that obstruction is present.[21] The typical CWD envelope shape seeing during a dynamic subaortic LVOTO is "dagger-shaped," with a mid to late systolic peaking, leftward concavity and inflection point of the ascending limb due to maximal flow acceleration in late systole, when the heart is empty and the LVOTO is worse due to SAM. Conversely, a severe fixed subvalvular LVOTO is characterized by an early peak LVOT signal with a CWD rounded envelope shape, or a triangular

envelope shape in mild cases (e.g. subaortic membrane). These fixed patterns are different from the late systolic peaking of the dynamic obstruction. It is also important to be cautious when analyzing the CWD through the LVOT to avoid contamination by MR jet. MR jet tracing is symmetrical with a parabolic shape and with an early systolic peaking.[1]

After induction of anesthesia and initiation of positive-pressure ventilation, LVOT flow velocities and gradients can vary significantly from those in awake, spontaneously ventilating patients, due to alterations in ventricular loading and contractility conditions, with LVOTO absent in up to half of the patients.[22] If LVOTO is not present at the initial evaluation, provocative measures may elicit latent obstruction. Examples of provocative maneuvers include pharmacologic stress with isoproterenol (typically 12 mcg bolus) or dobutamine 10 to 20 mcg/kg/min.[3,23] If the chest and pericardium are open, then simply tapping the heart with a forceps creates a premature ventricular contraction (PVC) and induces the Brockenbrough–Braunwald–Morrow sign with accentuation of the LVOT gradient after a PVC.[24]

Intraoperative management care of the HOCM patient

Anesthetic management

With an estimated overall mortality of less than1 percent per year, hypertrophic cardiomyopathy, is the most common genetic cardiomyopathy.[21,25] Obstruction is determined by the presence of LVOT gradient using CWD by echocardiography, under resting and/or provocable conditions.[21,26] When the resting or the provocable gradient is ≥ 50 mmHg, transaortic septal myectomy is considered the gold standard technique to treat symptomatic HOCM.[27] At various phases during surgical septal myectomy, hemodynamic condition may change and lead to worsening or improvement in LVOTO by change in preload or afterload, SAM of the mitral valve or sympathetic stimulation, although LVOT gradients during general anesthesia are commonly lower than those estimated with transthoracic echocardiography preoperatively, due to bradycardia and hypotension induced by anesthetic agents. All these characteristics represent unique challenges in the anesthetic management of this patients, requiring a comprehensive understanding of the management of all the conditions required to decrease the LVOT gradient avoiding LVOTO.

The anesthetic management of this patients is based on the maintenance of sinus rhythm, adequate rate avoiding tachycardia and bradycardia, and avoidance of systemic hypotension preserving preload and afterload, with risk of cardiac arrest or refractory shock.[28–31] To avoid decreased preload perioperatively worsening LVOTO, which will be exacerbated during induction, prolonged perioperative fasting and dehydration should be avoided.[32] Additional to standard monitoring, external defibrillator pads are required in case sinus rhythm is required to be maintained, a large bore intravenous access for rapid fluid resuscitation to preserve preload, invasive arterial line for continuous blood pressure monitoring to facilitate a rapid response to changes in arterial pressure, a central line for use of vasopressors to maintain afterload avoiding systemic hypotension, and TEE are recommended. Automated implantable cardioverter-defibrillators will be present in HOCM patients with malignant dysrhythmias, which are not to be deactivated until external defibrillator pads are available. TEE is a more reliable tool to assess fluid status intraoperatively than central venous pressure or a pulmonary artery catheter (PAC) in HOCM patients.[33] The PAC significantly overestimates the preload of this patient population, due to existing high left ventricle end-diastolic pressure or pulmonary capillary wedge pressure, induced by an existing reduced left ventricular compliance, with normal central venous pressures readings, when the left ventricle may be in fact underfilled. TEE can help guiding volume resuscitation, by directly assessing the right and left chamber or vessel size or by dynamic evaluation of blood flow. The measurement of the left ventricle end-diastolic area is recommended to assess volume responsiveness and appears to provide a better index of left ventricular preload in patients with normal left ventricle function, when compared to PAC.[33] Due to its limited diagnostic utility in this population and risk of arrhythmias on insertion, the PAC is not routinely used.[28] Size and collapsibility of the inferior vena cava can also be evaluated by TEE for estimation of central venous pressure to predict response to fluid management.[34] Because the inferior vena cava inspiratory collapse occurs in hypovolemic spontaneous breathing patients, but may not occur in mechanically ventilated patients, respiratory variation of the superior vena cava (distensibility >36%) by TEE, accurately predicts fluid responsiveness in patients during controlled mechanical ventilation.[34,35] In mechanical ventilated patients, another way to predict fluid responsiveness is to measure the variation of the peak aortic blood flow velocity during the respiratory cycle (> 12% accurately predicts fluid responsiveness), as it is directly related to stroke volume variation, but is not reliable in patients with arrhythmia, right ventricular dilation or dysfunction, an open chest, or those ventilated with low tidal volumes (< 8 ml/kg).[35,36] High tidal volume and positive end expiratory pressure should be avoided in these patients due to the reduction in the preload, that can lead to LVOTO, recommending small tidal volumes with higher respiratory rates to maintain adequate minute ventilation.[37]

Cardiac medications to maintain sinus rhythm, including β-blockers, which reduce LVOTO due to their negative inotropic effect and reduction in heart rate, and calcium, sodium or potassium channel blockers, which improve diastolic compliance, should be continued throughout the

perioperative level. Premedication with glycopyrrolate and atropine should be avoided because they induce tachycardia. In patients with new-onset or poorly controlled atrial fibrillation, restoration of sinus rhythm by cardioversion or appropriate rate control should be considered before induction.[38] Pharmacological correction by intravenous bolus of either amiodarone or digoxin is less desirable, especially if LVOTO exists, due to associated hypotension with amiodarone, and positive inotropic with digoxin.[39]

It is important to prevent increases in contractility or heart rate by minimizing sympathetic stimulation. Ideally, the heart rate should be maintained between 60–65 beats per minute reducing LVOTO, and avoiding bradycardia (< 40 beats per minute) which may lead to a reduction in cardiac output and hypotension potentially resulting in myocardial ischemia, due to impaired coronary perfusion pressure in the hypertrophied, noncompliant myocardium.[28,30] It is equally important to maintain an appropriate intravascular volume (reduced preload), and avoid vasodilatation (reduced afterload). Irrespective of the agents used to induce anesthesia, the principles are the same: minimizing the sympathetic stimulation and avoidance of systemic hypotension.

The most common agents used in the induction of anesthesia in HOCM patients are summarized next. Benzodiazepines can be used as induction or co-adjuvant agents inducing hypnosis due to their limited effect on hemodynamics and prevention of intraoperative awareness, but they are associated with an increased risk of delirium, especially in the elderly.[40] Dexmedetomidine by stimulating α-2 and imidazoline receptors, is a sedative, anxiolytic, analgesic and sympatholytic agent that enhances anesthesia, which can be used as premedication in the induction of anesthesia in these patients, reducing the heart rate and attenuating hemodynamic stress response to intubation and extubation.[41] Propofol, used in induction of amnesia and hypnosis, has some myocardial depressant properties and is a potent vasodilator, predisposing to hypotension immediately following induction, hence the propofol dose and speed of administration should be reduced. This can be achieved by combining a propofol induction with a short acting opioid, for example, fentanyl or sufentanil, or higher doses of longer acting opioids, for example, methadone.[42] Etomidate is considered an option when compared to propofol for induction in this population, due its rapid onset, cardioprotective nature, and hemodynamic stability minimizing hypotension, nonetheless caution is recommended due to its increased mortality rate associated with adrenal suppression, even with a single dose.[43] Ketamine alone used as an induction agent in this population, may produce tachycardia, and may exacerbate pulmonary hypertension, predisposing to increased LVOTO and myocardial ischemia.[44] Ketamine and propofol combination has been used with success in many practices for induction of anesthesia in the high risk cardiac patient.

Induction of anesthesia with short acting opioids, as remifentanil, even at low doses (0.3–0.4 mcg kg^{-1} min^{-1}), is beneficial to mitigate potentially hazardous haemodynamic responses from stressful stimuli pre-intubation, limiting systemic hypotension post induction.[45] Conversely, longer acting opioids as sufentanil or fentanyl, first will require to wait for at least 3 to 5 min respectively, to get the onset of action to avoid sympathetic response, and second require vasopressors to support blood pressure post induction.

The ideal muscle relaxant used for induction in this population, should not produce histamine release due to the induction of vasodilatation, as rocuronium, vecuronium or cisatracurium, avoiding histamine releasing muscle relaxants as mivacurium or atracurium.[46,47] Vecuronium and rocuronium are the mainstays of induction of neuromuscular blockade in North America.

For the maintenance of anesthesia, volatile agents have the advantage of having a negative inotropic effect, attenuating sympathetic nervous system activity and have been shown to have ischemic preconditioning properties. At high doses volatile anesthetics can induce systemic hypotension attributable to myocardial depression and peripheral vasodilation.[48] Sevoflurane due to its minimal effect on heart rate and systemic vascular resistances, is better suited for the maintenance of anesthesia than isoflurane or desflurane.[32,37] On the contrary, nitrous oxide is not recommended due to its tendency to increase pulmonary pressures.[49]

Vasoactive agents

The use of vasoactive agents should aim to prevent increases in contractility or heart rate avoiding accentuated SAM, LVOTO and consequent MR, and avert decreases in afterload by the induction of vasoconstriction, to maintain an appropriate intravascular volume increasing venous return and thus cardiac output.

The use of a short acting intravenous β-blocker such as esmolol, with a half-life of 9 min, is ideal for suppressing sympathetic stimulation, thereby, decreasing the LVOT gradient and improving the cardiac output.[50] If SAM and LVOTO are generated as a result of surgical or other types of stimulation, for example, laryngoscopy, these conditions may be reversed by esmolol.[51] Hypotension that does not respond to fluid administration in the presence of LVOTO, should be treated with either a β-blocking agent and/or pure vasoconstricting agent such as the α1-agonists (phenylephrine) or vasopressin. Phenylephrine is preferred to norepinephrine due to the mild β-adrenergic agonist activity of norepinephrine and the induced bradycardia baroreceptor-mediated reflex of the phenylephrine.[52] Vasopressin, has the advantage of being a longer acting and having a less direct coronary vasoconstrictor effect than catecholamines.[52] Its effects are preserved under

hypoxic and acidotic conditions too. In cases of HOCM with moderate to severe MR, vasopressin, due to its lesser pulmonary vasoconstrictor effect, as compared with phenylephrine and other catecholamines, may the best agent when LVOTO is present.[53] β-adrenergic agents, such as dopamine, isoproterenol, dobutamine or epinephrine that increased inotropy and chronotropy, are generally contraindicated as they will exacerbate LVOTO and increase oxygen demand.[37]

Arterial and venous vasodilators including nitrates (as nitroprusside or nitroglycerin) and phosphodiesterase inhibitors (as sildenafil) are contraindicated to treat hypertensive episodes, because they reduce preload by venodilation and lower afterload by systemic vascular dilatation.[54]

The same considerations, including maintaining sinus rhythm, adequate preload and afterload, and suppressing sympathetic stimulation, that guide the management of patients with HOCM intraoperatively, must be applied postoperatively.[28]

Diastolic dysfunction

Abnormal cellular architecture and interstitial fibrosis with patchy myocardial scarring in HOCM contribute to impaired diastolic filling and relaxation and to derangement in the electrical tissue, putting these patients at risk of fatal dysrhythmias and high pulmonary venous pressures.[55] Despite a normal ejection fraction, global longitudinal strain is diminished in this population.[1] In HOCM, a high ratio of early diastolic mitral inflow velocity to mitral septal annular tissue velocity (E/e′ ratio) has predicted death, cardiac arrest, or ventricular tachycardia.[56] Enlarged left atrium, increased atrial reversal amplitude and velocity recorded at the pulmonary veins, and a short mitral A wave duration at the level of the mitral annulus, are associated with elevated left ventricular end-diastolic pressures, but conventional Doppler parameters are unreliable for estimating left ventricular filling pressures in HOCM.[57] Advanced Doppler parameters, as mitral E velocity to flow propagation velocity and mitral E velocity to early diastolic annular velocity, can be used to predict left ventricular filling in patients with reasonable accuracy ($r = 0.76$ and 0.85 respectively).[57] Despite the presence of left ventricular and left atrial filling abnormalities, the correlation between the mitral inflow and pulmonary venous flow velocities and invasive parameters of the left ventricle diastolic function are relatively weak.[1]

Impaired diastolic filling may be minimized by atrial synchronized right ventricular pacing or atrioventricular sequential pacing, making the IVS move away from the left ventricular wall during systole, resulting in an increase in LVOT dimensions and hence reduction in LVOT blood velocity, improving symptoms and relieving LVOTO in HOCM patients.[58]

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