Intraoperative TEE for the Hypertrophic Left Ventricle

Bonnie L. Milas, MD

February 12, 2013

Considerable time and effort is concentrated on left ventricular systolic dysfunction, yet the hypertrophied left ventricle causes considerable morbidity and mortality, as well as poses clinical and diagnostic challenges. Dealing with a left ventricle with an ejection fraction of 80-100% is far from ideal when the stroke volume is markedly reduced. This discussion will review the scenarios leading to the development of left ventricular hypertrophy, concentrating on the TEE characterization of the left ventricle and supporting structures, gradient quantification, and intraoperative decision making.

Ventricular Remodeling

The left ventricular remodels in response to myocardial injury or overload through chamber dilation and/or hypertrophy. This physiologic phenomenon occurs in health and in disease states. LV hypertrophy and remodeling is determined by the type of overload. Concentric change generally occurs due to pressure overload, while eccentric change is the consequence of volume overload. Classification of such states can be achieved by evaluating LV mass, LV volume, the ratio of LV mass/ volume (M/V), and relative LV wall thickness (RWT). The generally accepted scheme of classification follows as normal, concentric hypertrophy, concentric remodeling, and eccentric hypertrophy. The term “concentric” refers to ventricles that are without chamber dilation and therefore have a normal end-diastolic volume. Concentric remodeling is classified as increased RWT (or M/V) pattern, yet with normal LV mass. Once the LV mass is increased it is referred to as concentric hypertrophy. Both concentric processes are typically in response to LV pressure overload, with the earliest response exhibited as concentric remodeling. This hypertrophic response is an attempt to limit wall stress to allow for maintenance of normal LV systolic function and performance. Such compensatory response eventually yields LV diastolic dysfunction and diastolic heart failure. Systemic hypertension and aortic stenosis are disease entities that lead to a concentric physiologic response. Eccentric hypertrophy is applied to those ventricles with increased LV mass and dilated ventricular cavities. A classic example of eccentric hypertrophy is mitral regurgitation (MR) where in early-stage both the chamber size and LV mass are increased to enhance stroke volume, while normal RWT is maintained. As MR progresses to late-stage and the LV dilates further, RWT ranges normal to low. Investigators have suggested additional categories to classify LV hypertrophy as “mixed” or “physiologic”. Physiologic hypertrophy is characterized as LV chamber enlargement and increased LV mass as in the pregnant woman due to volume overload and the athlete due to pump performance, where an increase LV stroke volume is required. In physiologic hypertrophy diastolic function remains normal. Mixed hypertrophic responses include aortic insufficiency due to both systolic pressure and volume overload. This classification system of LVH excludes those processes in which wall thickness is non-uniform, such as myocardial infarction and hypertrophic cardiomyopathy.¹
LV mass = 0.8 \times (1.04[(LVId + PWId + SWId)^3 - (LVId)^3]) + 0.6 \text{ g}

Normal s: women 65-165 g., men 85-225 g.

LVMI = LV mass/BSA

Normals: women ≤ 95 g./m.², men ≤ 115 g

RWT = (2 \times PWId)/LVId

Normal ; 0.32 – 0.42

TEE Evaluation

Hypertensive Heart Disease

In the initial phases of hypertensive heart disease the LV undergoes concentric remodeling (normal LV mass and increased relative wall thickness) progressing to concentric hypertrophy (increased LV mass and increased relative wall thickness), then in end-stage form eccentric hypertrophy (increased LV mass, normal relative wall thickness) due to LV cavitary dilation and wall thinning. Since the largest local radius of the LV ventricular curvature is located at the basal septum, a septal bulge develops early in the hypertensive process. This may be particular prominent in the elderly, which is typically sigmoidal in shape-this shape is less common in the hypertrophic obstructive cardiomyopathy (HOCM) population.
LV wall thickness is generally < 16mm in mild-moderate hypertensives. These patients typically have a history of treatment for hypertension and are lacking in a history of familial cardiac hypertrophy. Systolic anterior motion (SAM), left ventricular outflow tract obstruction (LVOTO), and mid-cavitary obliteration can be seen in patients with isolated hypertension, but generally occur with provocation such as volume depletion, tachycardia, or atrial fibrillation. Peak systolic strain rate and to a lesser extent systolic strain are reduced. Global systolic function is typically normal or supra-normal until end-stage disease, and diastolic dysfunction develops early in the process. LV remodeling seen in the setting of aortic stenosis or sub-aortic membrane (fixed outlet obstructions) is similar to that seen in hypertension. The maximum acceptable LV outflow gradient in the non-HOCM population has yet to be defined. Very meticulous interrogation of the anatomic site of fixed or dynamic obstruction is required with color-flow Doppler (CFD), 2-dimensional imaging, and spectral Doppler as described below.* Contribution of mitral valve pathology would also require careful evaluation. Late-peaking gradients would be typical of dynamic obstruction. In order to avoid post-operative unmasking of dynamic LVOTO and to improve LV regression following AVR, some surgeons advocate for prophylactic myomectomy during aortic valve replacement in patients with either demonstrable dynamic subaortic gradient or marked septal hypertrophy.2-4 In a series of patients, there was a greater predilection of myomectomies performed at the time of AVR in women.2 It is not known whether gender differences in hypertrophic remodeling, or whether a smaller LVOT diameter is the primary factor.

Idiopathic Hypertrophic Obstructive or Non-Obstructive Cardiomyopathy (HOCM/HCM)

Approximately 25% of individuals presenting with unspecified LVH are ultimately found to have HCM. HCM is the most common genetic cardiovascular disorder with a prevalence of 1:500, inheritance is autosomal dominant.5 Asymmetric septal hypertrophy, septal to posterior wall ratio > 1.3, is diagnostic of HCM.6 Although most patients exhibit this asymmetric septal hypertrophy, the disease demonstrates significant phenotypic heterogeneity. These range from diffuse global hypertrophy to focal segmental hypertrophy of only 1 or 2 myocardial segments, often in a non-contiguous pattern. Apical HCM is one such variant. LV end-diastolic wall thickness > 15 mm. in any segment with normal wall thickness in other segments of a non-dilated LV is suspicious for idiopathic HCM. HCM patients often have significant right ventricular involvement with increased RV wall thickness and mass. Resting or provocable LVOT obstruction is present in 70% of cases.7 In the majority of patients the obstruction is directly related to the septal hypertrophy. However, in a subset of patients there is LVOTO with minimal septal hypertrophy. In this subset the obstruction is related to a variety of mitral valve or papillary muscle abnormalities. HCM patients have a higher incidence of bifid, accessory, or displaced papillary muscles contributing to their obstructive mechanism. Demonstration of a resting LVOT gradient of ≥ 30 mm. Hg. is associated with worse clinical outcomes including arrhythmia, stroke, heart failure, and death.8 Intraoperative interrogation for left ventricular outflow obstruction should be carried out methodically to determine the precise location of obstruction, whether it is predominantly apical, midcavitary, or within the LV outflow tract. *Color flow Doppler can be instrumental in identifying the sight of flow acceleration, followed by color suppression to evaluate tissue components eliciting the obstruction. Subsequently, spectral Doppler is used to quantify the obstruction. A deep transgastric or a transgastric short-axis view at 90-100⁰ can be employed to obtain a gradient, taking care to avoid
misinterpretation of a mitral regurgitant jet in the imaging sector. The deep transgastric view is preferred in order to sequentially “walk” a pulse-wave Doppler cursor through the entire LV cavity, should the LV cavity align parallel with the spectral Doppler signal. Continuous-wave Doppler can quantify the high velocity, maximum instantaneous gradient.* Interpretation of the gradient requires careful consideration, in that left ventricular outflow tract gradients under general anesthesia have been shown to decrease in 60% of HCM patients, increase in 38%, and remain unchanged in 2%. In the referenced study, 39% of HCM patients’ intraoperative gradients were technically not obtainable by TEE, and were otherwise measured by direct placement of a needle in the ascending aorta and LV to transduce pressures. If the obtained gradient is less than 30 mm. Hg., then provocation is suggested either by inducing a PVC (direct mechanical stimulation of the RV by the surgeon-following which the gradient increases) or by isoproterenol infusion. Isoproterenol is utilized to achieve a heart rate greater than 120 beats/min. or an LVOT gradient of greater than 50 mm. Hg. The most frequently seen mitral valve abnormality in HCM is systolic anterior motion of the anterior leaflet (less frequently the posterior leaflet), although elongated chordal structures are also susceptible to drag forces and can cause obstruction. Mitral annular calcification is assessed and may limit surgical repair options. MR is assessed at baseline and with provocative maneuvers. LV strain rate is markedly reduced in HCM. LV systolic function is normal or increased at baseline, but regional function may be reduced in those segments that are hypertrophied. Cardiac MRI late gadolinium enhancement images demonstrate advancing myocardial fibrosis as the disease progresses. Diastolic dysfunction is present due to inadequate LV relaxation and compliance, but is a non-specific finding.

Uncommon Causes of LV Hypertrophy

Rare causes of LV hypertrophy include neurodegenerative, non-compaction, and the infiltrative heart diseases. Friedrich’s ataxia is an autosomal recessive neurodegenerative disease which causes mitochondrial dysfunction and concentric LVH. Wall thickness is generally not > 15 mm. and the myocardium has a sparkling-granular appearance. Diastolic dysfunction is usually not present, but eventually myocardial fibrosis and necrosis lead to end-stage systolic failure, accounting for 50% of deaths from the disease. Non-compaction is a congenital process readily recognized by TEE due to the prominent LV trabeculations, due to intrauterine arrest of compaction of myocardial fibers. It typically appears as a two-layered myocardium, with the outermost layer of compacted myocardium and the inner layer of trabeculae with deep endocardial recesses. The ratio of non-compacted layer thickness end-systole to compacted thickness is generally > 2. Fabry’s disease is one of the infiltrative diseases due to an x-linked defect leading to accumulation of glycosphingolipids in organs, including the heart. Prominent papillary muscle involvement can be a distinguishing feature. Amyloidosis is a systemic disease involving the deposition of light-chain amyloid proteins in organs. In the heart this deposition leads to concentric LVH with a sparkling-granular quality, but early pseudonormal or restrictive diastolic dysfunction (“stiff heart”) distinguishes it from Friedrich’s heart disease. Sarcoidosis leads to generalized LVH and a restrictive myopathy, but asymmetric basal-septal involvement can mimic HCM. Systolic strain rate can assist in discerning the various uncommon causes of LVH.
References
