Learning Objectives
At the conclusion of this lecture, the participant should be able to:
1. Identify normal and abnormal left ventricular systolic function
2. Perform qualitative and quantitative assessment of left ventricular systolic function
3. Recognize the LV segments and their corresponding coronary distribution
4. Assess regional wall motion in these segments and differentiate between ischemic and non-ischemic etiologies

Assessment of Global LV Function

The standard approach to assessing global LV function is to consider a variety of “load dependent” indices of systolic function (eg. fractional shortening, fractional area change, ejection fraction, cardiac output and dP/dt, defined below). “Load independent” indices (including a mixed bag of assessment of either contractility or afterload from rate corrected mean velocity of fiber shortening, circumferential wall stress, meridional wall stress, and pressure volume or area loops generated during changing loading conditions) can be assessed in research situations, but are not used clinically due to their complexity. The rapid growth of new echo technologies: Doppler tissue imaging (DTI), “speckle tracking”, vector velocity imaging and 3D echo, along with cardiac magnetic resonance imaging, have been instrumental in “rewriting the textbooks” on the mechanics of ventricular function. Thus, it is now appreciated that filling and ejection from the LV is based on a “vortex” flow pattern with an inner rapidly descending swirl and an outer slower ascending rotation based on a right handed helical arrangement of myofibrils in the subendocardium (which is close to longitudinally oriented at an 80 degree angle) and a left handed arrangement in the subepicardium (with an oblique orientation of about -60 degrees) with the midwall fibers being oriented circumferentially at a zero degree angle. (1,2) Thus, once can predict that the subendocardium contributes significantly to longitudinal contraction while the mid and subepicardium contribute more to rotational/radial motion. (3) Nowadays, the cardiac anesthesiologist must be aware of the 3 major vectors of cardiac motion during systole, namely longitudinal, radial and circumferential although in routine clinical practice visual appreciation of longitudinal and inward radial motion is usually all that is required. The concept that descent of the mitral annulus (“descent of the base”, measured by M mode echo) along the longitudinal axis of the LV is a sensitive measure of ventricular function was first articulated in the late 1980’s. It has been markedly reinvigorated by precise measurement of mitral annular velocity/displacement or velocity of the basal annular segments that are relatively easily obtained with DTI and speckle tracking on most new echo machines in the cardiac OR and in fact, constitute an important part of any clinicians “eyeball assessment” of overall ventricular function. (4-6) Given the importance of this topic and the linkage of methods to technical evolution, the American Society of Echocardiography continues to establish freely accessible guidelines (via the ASE website) for how best to measure key variables that are invaluable to the busy clinician. (3,7)

Assumption of symmetrical geometry and contraction are required for application of single dimensions (planes) to determine ventricular volumes. Use of biplane methods (eg. 2 orthogonal imaging planes) is more accurate in the abnormal ventricle with wall motion abnormalities or an abnormal shape (eg. increasing sphericity due to adverse remodeling from CHF or MI). (8) TEE derived measurements have been reported to underestimating LV volumes relative to either TTE or angiography, although a more recent “simultaneous” TEE and TTE study disputes this. (9) The most commonly cited factor for any discrepancy is “foreshortening” of the LV apex with TEE, yielding a shorter LV long axis dimension (note: long axis dimension is assumed to start at apex and run to the midplane mitral valve). With TTE,
the sonographer is able to move the transducer over a wide area to find the true apex, while with TEE this is not possible (or harder to do from inside the esophagus). In fact, the differences are usually pretty minor (particularly for perioperative applications). However, you do need to be diligent to avoid grossly erroneous long axis dimensions (however, keeping an eye on mitral annular or basal motion can give you usable info even when you are foreshortened).

The prolate ellipse method (or cube formula) is quite simple in that it states that volume approximates the internal diameter (short axis at the tips of the mitral valve by TTE) to the third power. This formula has been shown to overestimate LV volumes as in larger ventricles the LV dilates to a more spherical shape (eg. along the short axis dimension). The more commonly considered area-length formula (in fact developed for angiography) assumes a bullet shaped LV. The formula is \( V = 0.85 (\text{area of the LV squared})/\text{long axis length} \). Area can be traced manually using onboard software or it can be determined using the short axis diameter at the level of the papillary muscles. Acquisition of a “true” long axis is of course critical. It is said to be preferable to Simpson’s method if only one apical view is available (although in fact is rarely used anymore).

Simpson’s MOD is clearly the most common used method and is standard on all echo machines. It is based on the assumption that the ventricle can be modeled as a stack of circular discs that may vary in diameter based on the shape of the ventricle. The discs are automatically determined based on perpendicular radii from the long axis dimension (which is determined from tracing the endocardial borders at end systole and diastole). Usually 20 discs are calculated, although in fact it is not known precisely how many discs are optimal. Likewise, a flaw in the assumption that this technique is better than other older methods in patients with abnormal anatomy or contraction patterns, is that the discs must be modeled as perfectly circular, which is not always the case anatomically. Nonetheless, this method is the current gold standard. Certain echo machines make this really easy to using an automated template while in others you have to trace it manually using the trackball (which is often a pain). When using the biplane Simpson’s MOD (which probably is not commonly done in the OR anyway but is in the echo lab), it is usually stated that you should not perform the analysis if the long axis dimensions vary by more than 20% between the 2 orthogonal imaging planes. An obvious limitation of any technique using long axis dimensions is that longitudinal scanning results in poorer endocardial resolution than axial (short axis) scanning and tracing of these images can involve a fair amount of “artistic license”.

Once you have made the appropriate measurements in both end-systole and end-diastole, it is very simple to determine what you can call “ejection fraction” paradigm indices since the concept of some measure of area or volume ejected divided by the residual measurement left in the pump is the same for all listed below:

1. M-mode echo single plane: End diastolic internal diameter – End systolic internal diameter/End diastolic internal diameter x 100%. This is termed \textit{Fractional Shortening} (FS), mean 33% (range 28 – 41%) and is usually made with TTE in a parasternal plane. It is quite easy to measure with TEE using M-mode in the TGSAX view from inferior to anterior wall.

2. 2D echo single plane: End diastolic area – End systolic area/End diastolic area x 100%. This is termed \textit{Fractional Area Change} (FAC), mean 57% (range 37 – 76%). Most commonly done in the TGSAX view. The lack of contribution of the apex, which is commonly abnormal in patients with severe CAD is a limitation.

3. 2D echo multiplane: End diastolic volume – End systolic volume/End diastolic volume x 100%. This is \textit{Ejection fraction} (EF), mean 62% (range 45 – 90%). Volume is measured using one or more long axis dimensions using Simpson’s MOD.

Usually EDA and ESA are estimated qualitatively as the largest and smallest areas during contraction respectively. For EDA, the first frame at or before systolic coaptation of the mitral valve or the first frame on the ECG QRS complex (most common and easy to do assuming you use ECG leads); for ESA the frame preceding early diastolic mitral opening is mentioned, but in fact, the smallest cavitary size is easiest (there is no precise ECG event associated with end systole other than it usually falls within the
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The ASE recommends that when obtaining any of these measurements that the LV be displayed on the screen in its most magnified setting possible. They discuss the fact that there is a trade-off here, since a smaller image on the screen will likely have higher resolution for tracing of the endocardial borders, but the smaller dimensions would be more likely to subject to measurement errors. They also note as do numerous articles that reproducibility of EF determinations has likely at very best 7% error and often much worse. Clinicians should realize that for most epidemiologic and risk assessment purposes that EF’s are usually grouped into wide categories (eg. < 30%, 30 – 50%, > 50%) due to measurement errors and different types of technology used.

Physiologic bases for ischemia detection

Animal and clinical data well document the high sensitivity of echocardiographic methods for measuring the effects of myocardial ischemia on regional and global ventricular function. (10) Most often this is assessed by 2D imaging, focusing on visually observed “gross” changes in regional systolic function. Newer methods can evaluate more complex changes (e.g. velocity of a particular myocardial segment throughout systole and diastole) at higher temporal resolution (as small as 5 msec increments for color M-mode TDI) in multiple regions (which is usually too much information for the busy practitioner). The basic physiologic principle behind ischemia detection is the rapid reduction in regional myocardial function with an acute reduction in myocardial blood flow in the affected coronary artery territory (no secret here!). Regional or segmental wall motion abnormalities (R or SWMA’s) may occur sooner than ST-segment changes, potentially allowing earlier diagnosis and therapy (although the bigger issue is its value when the ECG is grossly insensitive such as with conduction abnormalities or LVH strain pattern, etc.). TEE also allows recognition of important related anatomic factors (a chronically infarcted segment consistent with prior MI, VSD or associated mitral regurgitation).

The most sensitive changes with ischemia are reduction (hypokinesia) or cessation (akinesia) of systolic wall thickening (which normally thickens by 50% of the end-diastolic value). With complete cessation of coronary flow, systolic wall thinning will occur in a previously normal segment causing outward bulging (dyskinesia) although this is rarely encountered outside the medical STEMI setting. However, “pseudo-dyskinesia” is relatively common in chronically infarcted segments particular when loading conditions are altered acutely. Since the function of the heart as a pump is to eject blood, requiring inward motion of the endocardial surface during systole to reduce cross sectional area, a reduction in endocardial excursion is the most obvious and easily observed visual sign of dysfunction. Adjacent segments in the acutely ischemic ventricle often develop exaggerated endocardial excursion (“compensatory hyperkinesis”), offsetting the adverse effects of regional dysfunction on “global” stroke volume. Thus, changes in systemic hemodynamics are usually a late and ominous sign occurring with a large ischemic region (i.e. left main or proximal LAD stenosis) or with global subendocardial ischemia in the absence of CAD as may occur with aortic stenosis.(11) In the clinical setting, systolic wall thickening and endocardial excursion are assessed jointly to arrive at a wall motion “grade”.

Animal studies demonstrate that changes in endocardial excursion overestimate the actual amount of hypoperfused ischemic myocardium, while systolic wall thickening more closely approximates it .(12) This is likely due to “tethering” of the abnormal myocardium to adjacent segments which imposes complex mechanical effects, either horizontally or vertically oriented. Other factors (e.g. endocardial pacing, left bundle branch block, septal changes due to release of pericardial restraint, etc.) can reduce the specificity of abnormal endocardial excursion as a marker of ischemia. However, in these situations systolic wall thickening should be normal. The magnitude of endocardial excursion and wall thickening has been documented to vary between different regions of the heart and between “normal” individuals. Thus, the patient’s baseline status should serve as a control, which is particularly important when evaluating subtle changes (hypokinesia).

The response of the myocardium to inotropic stimulation, as occurs with dobutamine stress testing, should be appreciated by all clinicians. Regional function may start to deteriorate with only a 20%
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reduction in transmural flow and at least one clinical study found akinesis occurring at only 25% reduction. With low doses of dobutamine, normal myocardium becomes hyperkinetic as coronary blood flow increases. Development of hypo- or akinesis in a previously normal segment represents an ischemic response. Chronic transmural infarction will not respond to either low or high doses. Improvement of function in a baseline akinetic segment at a low dose suggests contractile reserve. A biphasic response with improvement at a low dose followed by deterioration at higher doses, is characteristic of hibernating myocardium. End-diastolic wall thickness of $\leq 0.6$ cm has been shown to virtually exclude the potential for functional recovery with myocardial revascularization. (13) Development of short-term myocardial stunning during CPB often complicates early postCPB prediction of long-term viability.

Abnormal loading conditions commonly encountered perioperatively, at either end of the volume or pressure spectrum, complicate interpretation of RWMA’s. Wall motion can appear severely hypokinetic with hypervolemia or with elevated afterload accompanying severe hypertension (the latter being a manifestation of the direct relation between increasing afterload and decreased fractional area change). However, in otherwise normal patients, changes occur in all walls equally and motion returns to normal with reduction in pressure or volume. Hypovolemia accentuates normal regional disparity in motion and may cause gross disparity, particularly in previously abnormal segments. Thus, a previously akinetic segment may appear dyskinetic. However, wall thinning will not be present. (14)

Interpreting wall motion in patients with severe left ventricular hypertrophy (LVH) is challenging since cross-sectional area may already be significantly reduced (particularly with concentric hypertrophy) making appreciation of subtle changes difficult. Ventricular pacing, particularly via endocardial wires during open chest cardiac procedures can be problematic, simulating LBBB, which is known to cause a distinctive type of septal dyskinesia (so called “double bounce” pattern, described long ago on M-mode imaging). Release of pericardial restraint with sternotomy has been reported to cause abnormal septal motion, which on occasion may be confused with ischemia.

Localization of Ischemia: The 16 and 17 segment system

Accurate anatomic classification of RWMA’s is essential to guide clinical management, localize coronary artery pathology and to gauge therapy or need for diagnostic intervention (i.e. cardiac catheterization). Documentation in the medical record using accepted terminology for quality improvement, billing and for communication with cardiologists and primary care providers is essential.

The 16 segment left ventricular model adopted by the American Society of Echocardiography in 1989 (a simplification of an older 20 segment model) was the basis of the Society of Cardiovascular Anesthesiologists comprehensive intraoperative protocol. (15) It is based on division of the left ventricle into apical, mid and basal zones with roughly equal mass although anatomic studies demonstrate an expected decrease from base to apex. The basal and mid zones each contain 6 segments while the apical zone with its smaller area has only 4. To assess all 16 segments requires interrogation of 5 imaging planes: the mid esophageal 4 chamber, 2 chamber and long axis views along with 2 transgastric views: basal and mid.

A more recent American Heart Association/ASE committee charged with standardizing myocardial nomenclature between radionuclear, echocardiographic and magnetic resonance modalities, adopted a 17 segment model in which an additional segment, located at the ventricular apex (devoid of direct contact with the ventricular cavity), was added. (16) An additional change was deletion of the term “posterior” from the segmental nomenclature as it is only used in echocardiography and not the other modalities. They recommend exclusive use of the term “inferior”. Despite this, “posterior” is still commonly used in cardiology lingo and literature.

The basal transgastric view can be problematic to obtain and interpreting wall motion in this region adjacent to the fibrous AV skeleton can be difficult. As a result the AHA/ASE group recommends scoring of basal segments containing myocardium in all 360 degrees around the ventricle. The clinician must also remember that in order to obtain a “true” ME 4 chamber view one must rotate the multiplane
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transducer approximately 10 degrees to “remove” the LV outflow tract (normally present in the “neutral” 0 degree orientation). This allows visualization of the basal septal segment.

Assessment of the apex is accomplished nearly exclusively using the ME longitudinal orientations. A tranverse apical image is sometimes possible below the TG SAX view (obtained by posteroflexion at that level) but not commonly. Given that the apex is in the “far field” of the image, it is important to optimize gain/TGC settings in this region, place the transmit zone over the apex and use as low a frequency as is consistent with optimal resolution (usually no higher than 5 – 6 MHz). In most new systems a “zoom” or “RES” feature can also help. Recognition by the clinician that apical RMWA’s are commonly encountered in patients with significant CAD and that complications of infarction, particularly aneurysm and thrombus formation, are most commonly encountered in the apex, makes it an important area to evaluate.

The TG LAX view can be helpful in assessing anterior and inferior segments in a complementary manner to the TG SAX view, especially when imaging from the ME planes is suboptimal. However, it is not possible to image the apex in this view.

Coronary Artery Perfusion Zones

The perfusion zones or territories of the 3 main coronary arteries, although potentially variable on an individual level, have been mapped to varying combinations of adjacent myocardial segments on a population basis. (7) The most important factor influencing this mapping is the origin of the posterior descending artery from the right coronary artery in a right dominant system (the common scenario) versus its origin from the circumflex in the less common left dominant system. There is usually some degree of overlap between territories, most commonly in the posterior segments and the infero- and lateral apical segments. The LAD usually perfuses these apical segments, although the posterior descending (inferoapical) or circumflex (lateral apical) may also. In the newer 17 segment system the highest degree of variability is encountered in the true apical segment.

The population based uniformity of perfusion zones is one of the two main reasons for the popularity of the mid and basal transgastric short axis views for intraoperative monitoring (besides being easiest to continuously assess intra-cavitary area/volume) since they are the only views in which a portion of the territories of all three main coronary arteries perfusing the LV are visualized. Usually the mid papillary TG SAX is used although the basal view has similar anatomic characteristics. With reduction in flow in any (or all) of the three main coronary arteries one will usually see a new RWMA. However, if stenosis occurs distal to this region (i.e. perfusing the apical segments), wall motion will usually remain normal in this view and thus the clinician must periodically evaluate other imaging planes for a complete assessment. It should be pointed out that in the ME 4 chamber view, right coronary perfusion of the RV free wall along with LAD perfusion of the septum and LCX perfusion of the lateral wall can be evaluated.

The right coronary artery perfuses the bulk of the RV, although the conus branch of the LAD may supply a portion of the RV free wall. Right ventricular involvement during inferior LV infarction is common anatomically, but clinical manifestations (primarily RV failure manifested by systemic hypotension with clear lung fields) are infrequent.
References

7. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pelikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440-63.
