LV MORPHOLOGY

The left ventricular wall comprises three layers- middle circumferential layer and superficial and deep longitudinal layers:

1. **Subepicardial longitudinal layer** (25% of the wall thickness): arise from the insertions of the cardiac valves at the cardiac base and runs obliquely down to the apex where they loop around and form the vortex cordis (spiral arrangement of muscular fibres at the apex of the heart)
2. **Subendocardial longitudinal layer** (20% of the wall thickness): at the heart vortex, the superficial fibres invaginate in a spiral pattern to give rise to the sub-endocardial layer. There is a similar continuity between superficial and deep layers at the base of the ventricle.
3. **Middle circumferential layer** (55% of the wall thickness): These circumferentially arranged fibres are confined to the LV and septum. The RV free wall has only superficial and sub-endocardial layers. The greatest thickness of circumferential fibres is found at the LV base where they encircle the inlet and outlet. They do not insert into the mitral or aortic valves, nor to the ventricular apex.

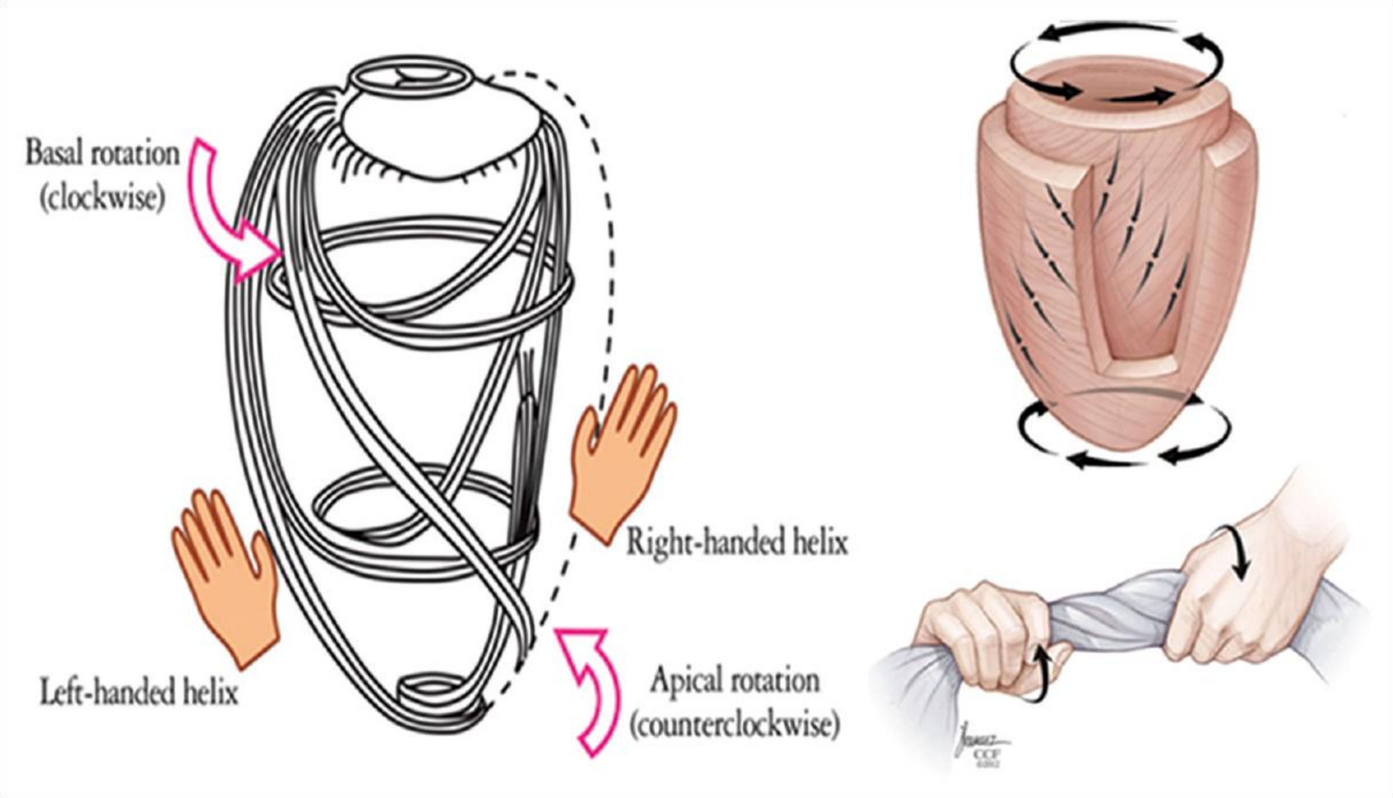


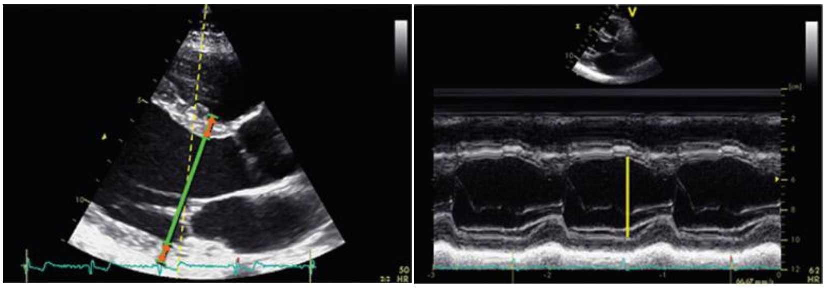
Fig. Myocardial fibres in the **subepicardium** run helically in a left-handed direction, fibres in the **mid layer** run circumferentially, and fibres in the **subendocardium** run helically in a right-handed direction

N.B: TDI reveals that LV short-axis function is generally preserved with age whereas long-axis function does deteriorate with age. It also shows that the endocardium moves faster than the epicardium.

Parameters of LV size, mass & function

	Normal: male (female)	Mild	Moderate: male (female)	Severe
LV wall thickness IVSd / PWd (cm)	≤ 1 (0.9) *(RV free wall <0.5)*		**1.5-2**	>2
LV diastolic diameter (cm)	≤ **5.8 (5.2)** *(RVD1≤2.8, RVD2≤3.3)*		6.4-6.8 (5.7-6.1)	
LV systolic diameter (cm)	≤ 4 (≤ 3.8)			
LV mass (g)	≤ 224 (≤ 162)		259-**292 (187-210)**	
LVEDV index (ml/m^2)	≤74 (≤61)	<97		
LVESV index (ml/m^2)	≤31 (≤24)	<49		
Stroke distance (VTI_{LVOT}) cm	18-22			
SV (ml/beat)	60-100 (> 35 ml/m^2)			
Cardiac output (L/min)	> 4			
FS (%)	28-40 *(RV Fractional area change 32–60)*		20-**15**	
EF (%)	≥ 52 (≥ 54) *(RV EF 45 ± 5)*		45-35	
dP/dt (mmHg/sec.)	≥1000-1200			<800
MPI	≤ 0.47 (<0.4)			
Ś (average of septal and lateral) cm/s	> 7.5 *(another reference >9-10) (>9.5 for RV)*			
GLS (%)	−20			

LV DIMENSIONS



Linear LV measurements: LVIDd/s, IVSd, LVPWd should be measured in the PLAX (single long axis view) at the level of the LV minor axis, perpendicular to the LV long axis at or immediately below the level of the MV leaflet tips. The electronic calipers should be positioned on the interface between the myocardial wall and cavity and the interface between the wall and the pericardium (leading edge to leading edge is no longer recommended due to the improved resolution of contemporary echo machines) . Measurements are traditionally made using M-mode (due to its high temporal resolution, which makes it easier to visualize motion of the endocardium), although linear measurements obtained from 2D echo are preferred to avoid oblique sections of the ventricle, which overestimate cavity size and underestimate function.

End-diastolic measurements are often taken at the onset of the QRS complex on the ECG trace. However, the ASE recommends that end-diastole is taken as the frame after MV closure or the frame in which the LV internal dimension is largest.

End-systolic measurements are often taken as the point when the LV internal dimension is at its narrowest but the ASE also suggests using the frame before MV opening as an alternative. The apical 2-chamber view does not show mitral valve opening very well, and so measurements taken in this view should not be based on the timing of mitral valve opening.

Pitfalls in the assessment of LV dimensions include: (1) Failure to take measurements at the correct time points (end-systole or end-diastole) (2) Failure to take measurements perpendicular to the long axis of the LV (3) Failure to identify the endocardium correctly – be particularly careful to avoid mistaking chordae tendineae for the endocardium of the LV posterior wall.

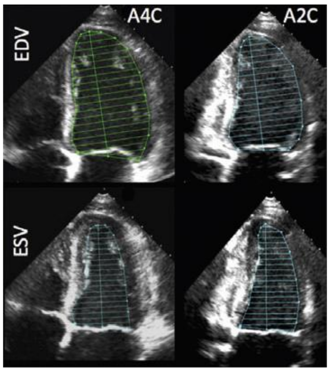
LV sphericity index- is the ratio of long to minor axis. With progressive dilation, the minor axis increases disproportionally, and the ratio of long to minor axis decreases. Typically, sphericity index < 1.5 implies pathologic remodeling. This leads to apical and lateral displacement of the papillary muscles and reduction of the length of the mitral apparatus and resulting in functional MR. Sphericity index is a predictor of functional (exercise) capacity

LV VOLUMES

LV volumes can be calculated using linear measurements, volumetric measurements (± contrast) and 3D echo:

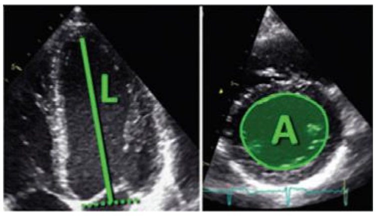
1. Linear measurements: the cubed (Teichholz) and Quinones methods for calculating LV volumes from LV linear dimensions are no longer recommended for clinical use as they rely on the assumption of a fixed geometric LV shape such as a prolate (spheroid) ellipsoid, which does not take into account the variations in regional wall motion and is highly prone to inaccuracies, particularly in IHD.

2. Volumetric measurements (modified Simpson's method and area length method)- are usually based on tracings of the interface between the compacted myocardium and the LV cavity.



Modified Simpson's rule method- is the best (and most commonly used) way of calculating LV volumes. It is also known as the biplane method of discs, as it essentially splits the LV into discs (usually 20) of differing sizes from base to apex and uses the summation of the discs for determination of the LV volume. The apical 4C & 2C are used. Echo machines automate much of the process and require the operator to:

- Obtain the best view of the LV, in the apical 4C view, paying particular attention to clear endocardial border definition and avoidance of foreshortening (results in volume underestimation) and artefacts. Any visible papillary muscles and trabeculae should be ignored (the widely reported underestimation of LV volume by echocardiography, compared to CMR, is, in part, due to failure to, or the difficulty of, excluding trabeculae from the cavity tracing). Acquiring LV views at a reduced depth to focus on the LV cavity will reduce the likelihood of foreshortening and minimize errors in endocardial border tracings.
- Freeze a loop and find the end-diastolic image.
- Now trace the endocardial border from the mitral valve annulus all the way down to the apex and then back up to the annulus on the opposite side. At the mitral valve level, the contour is closed by connecting the two opposite sections of the mitral ring with a straight line.
- Scroll to the end-systolic frame and trace the endocardial border
- Repeat the whole process in the apical 2C view; this will take better account of the elliptical cross-section of the LV and any regional wall motion abnormalities.

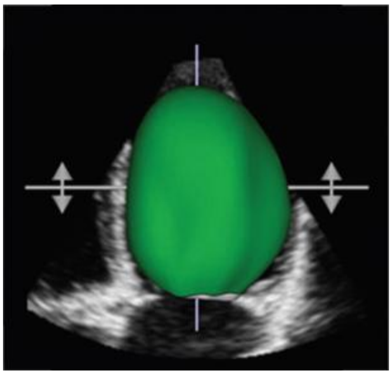


Area–length method- an alternative method to calculate LV volumes when apical endocardial definition precludes accurate tracing. In this method the LV is assumed to be bullet shaped (which does not always hold true). can be useful for estimating LV volumes when the endocardium cannot be seen clearly enough to allow accurate tracing. However, it does make major assumptions (and simplifications) about the shape of the LV:

1. In the PSAX, mid-LV level, freeze a loop and find the end-diastolic frame. Perform planimetry by tracing the endocardial border to calculate the cross-sectional area of the LV cavity at this level in cm^2. Ignore the presence of the papillary muscles as you trace the endocardium.
2. In the apical 4C, in the end-diastolic frame, measure the length of the LV long axis from the apex to the mid-point of the mitral valve.
3. The LVEDV, in mL, is given by: LVEDV = (5 x area x length) ÷ 6
4. Scroll to the end-systolic frame and repeat steps 1 to 3 to obtain the LVESV.

Contrast agents should be used when needed to improve endocardial delineation. Contrast-enhanced images may provide larger volumes than unenhanced images that are closer to those obtained with CMR. Care should be taken to avoid acoustic shadowing, which may occur in LV basal segments in the presence of high concentrations of contrast.

Automatic LV contour assessment is a relatively new method to assess LV volumes and highly affected by the quality of image and the consequent computer processing usually improved by operator's editing.



3-D echo- gets around many of the problems inherent in assessing LV volumes using 2-D echo. 2D echo relies heavily on geometric assumptions and manual tracking of the endocardial border in assessing ventricular function. Using 3D echo, the volume of the LV is calculated from the entire endocardial surface without the need for assumptions about its shape. Because the issue of foreshortening is less relevant in 3D data sets, 3D image acquisition should focus primarily on including the entire left ventricle within the pyramidal data set. To ensure reasonably accurate identification of end-systole, the temporal resolution of 3D imaging should be maximized without compromising spatial resolution. Tracking of the endocardium is done by semi-automatic border detection algorithms. The assessment of LV volumes, mass and function using 3D echo has been well validated and shown to be more accurate (and reproducible) than 2D techniques and has a close approximation with CMR.

Recommendation

- LV size should be routinely assessed on 2D echo by calculating volumes using the biplane method of disks summation technique.
- In laboratories with experience in 3D echo, 3D measurement and reporting of LV volumes is recommended when feasible depending on image quality.
- When reporting LV linear dimensions, the recommended method is 2D-guided measurements (rather than M-mode measurements).
- LV size and volume measurements should be reported indexed to BSA.

LVH & LV MASS

The echo assessment of LVH includes:

1. Description of the overall appearance of the LV (concentric versus asymmetrical hypertrophy). LVH has been categorized into two distinct basic types: concentric hypertrophy, occurring in response to a sustained pressure overload (hypertension and AS) in which wall thickness increases without chamber enlargement, and eccentric hypertrophy, in response to a chronic volume overload (AR and MR) in which chamber volume enlarges without a relative increase in its wall thickness. Asymmetric and apical hypertrophy are 2 typical types of hypertrophy in patients with hypertrophic cardiomyopathy.
2. Measurement of LV dimensions
3. Calculation of LV mass
4. Assessment of LV function (systolic and diastolic)
5. A search for underlying causes, such as aortic stenosis or aortic coarctation.

LV mass increases with LVH, which can be a result of a primary cardiomyopathy (e.g. hypertrophic or infiltrative cardiomyopathy) or secondary to increased LV afterload (e.g. hypertension, aortic stenosis).

LV mass using linear measurements: the so-called cubed (Teichholz) formula is fast and widely used but Small measurement errors can become greatly magnified because of the cubing of LV dimensions and it should not be used in cases with distorted LV geometry (e.g. isolated areas of hypertrophy).

LV mass using volumetric measurements: modified Simpsons' rule method, area-length method and truncated ellipsoid formula.

3D echo LV mass estimation: direct measurement without geometrical assumptions about cavity shape and hypertrophy distribution. In abnormally shaped ventricles or in patients with asymmetric or localized hypertrophy, 3D echo is recommended.

LV SYSTOLIC FUNCTION

LV systolic function can be affected by preload (e.g. IV fluid overload) as well as afterload (aortic resistance). Most clinically used parameters of ventricular function including stroke volume and ejection fraction are afterload dependent. Several methods have been proposed for correcting for afterload or creating afterload-independent indices of LV performance, including calculating ventricular wall stress and creation of pressure volume loops. Clearly, the better the endocardial border definition, the more accurate the measurement.

Echo assessment of LV systolic function

1. Fractional shortening (FS%)- is a measure of the percentage change in LV dimensions between diastole and systole. Fractional shortening % = [(LVEDD –LVESD) ÷ LVEDD] × 100. A normal FS% lies in the range 25–43%

2. Ejection fraction (EF%)- is the most widely quoted measure of LV systolic performance, and expresses the percentage of volume of blood pumped out of the LV with each heartbeat. EF= (LVEDV-LVSV)÷LVEDVx100. **i.e.,** EF = SV ÷ LVEDV x100. A normal EF is ≥ 55 %. LV volumes can be calculated using linear measurements, volumetric measurements and 3D echo (see above). The EF is affected later than other parameters (diastolic function, longitudinal deformation) in a cardiomyopathy process.

Hints:

- MR overestimates LV EF since LV volumes are ejected into the LA in addition to the aorta, hence EF will increase.
- Accurate measurement of EF is crucial in the monitoring of patients with breast cancer receiving Trastuzumab (Herceptin), a drug that can affect LV function. It is recommended that treatment should not be offered to those with an EF ≤ 55 %. Patients on treatment should have a cardiac functional assessment every 3 months and if the EF drops by 10% ejection points or more from baseline and to below 50% then treatment should be suspended. The BSE stresses the importance of measuring EF accurately in these patients, using the modified Simpson's rule method (or 3-D echo) and with the availability of LV contrast as appropriate, and that echo departments should have recent audit data to demonstrate that they can reproducibly measure EF to the requirements of these guidelines.

The European Society of Cardiology criteria for 'normal or mildly abnormal LV function' are:

- LVEF >50%
- LVEDV index <97 mL/m^2
- LVESV <49 mL/m^2

3. LV Stroke Volume (SV), CO & CI- when velocity is integrated over time (VTI), the units that result from this operation are a measure of distance (cm), hence the term stroke distance, which is the linear distance that the blood travels during one flow period. When VTI and the corresponding cross-sectional area (cm^2) are measured at the same point, their product equals stroke volume (cm^3 or ml), which is the volume of blood ejected by the heart with each contraction (assuming no valvular regurgitation or cardiac shunt). In this way SV can be calculated by echo across any structure where one can measure cross sectional area and velocity of blood flow. This could be the aorta, the pulmonic artery, or across any of the valves. The easiest and least variable place to measure SV is at the LVOT. The LVOT diameter changes very little through systole and diastole and is assumed to be constant and closely approximating a circle in shape, however this introduces some error as it is in fact elliptical in many patients.

Stroke volume (SV) = stroke area x stroke distance
SV = CSA_{LVOT} x VTI_{LVOT} … ($CSA = \pi.r^2$)
SV = LVOT
Stroke volume index (SVi) = SV/BSA
CO = SV x HR/1000
CI = CO/BSA
SV & EF can be used to estimate LVEDV: EF = SV ÷ LVEDV x100
N.B: Stroke distance (VTI_{LVOT}) < 18cm is an adverse prognostic marker in DCM

4. Rate of ventricular pressure rise (dP/dt)- can be estimated by using time interval between 1 and 3m/sec on MR velocity CW spectrum during IVCT i.e. in early phase of systole before AV opens when there is no significant change in LA pressure. This is a relatively load-independent measure of ventricular contractility. It is rapid with normal LV systolic function. dP/dt starts to fall if systolic function is impaired. The measurement of dP/dt requires the presence of MR:

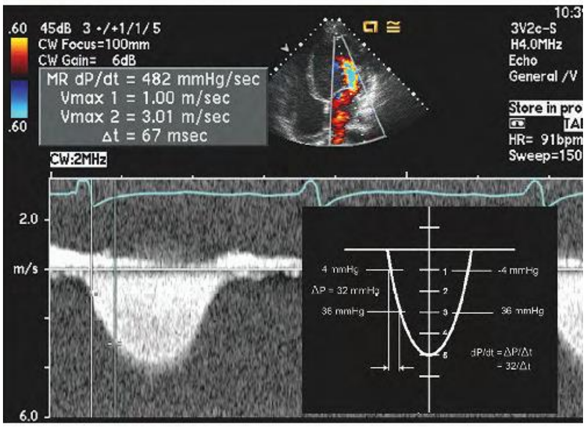
a) In the apical 4-chamber view, use CW Doppler to obtain a spectral trace of MR, ensuring careful alignment of the ultra-sound beam with the regurgitant jet. Set the sweep speed as high as possible to 'spread out' the trace and make it easier to mark the relevant timepoints (usually 100 mm/sec).

b) Using the trace, mark the points where the regurgitant jet velocity reaches 1 m/s and also 3 m/s. Measure the time interval (dt) between these two points in seconds

c) dP/dt is therefore calculated, in mmHg/s, by dividing "the change in pressure gradients (dP) between the two velocities" by the measured time interval (dt).

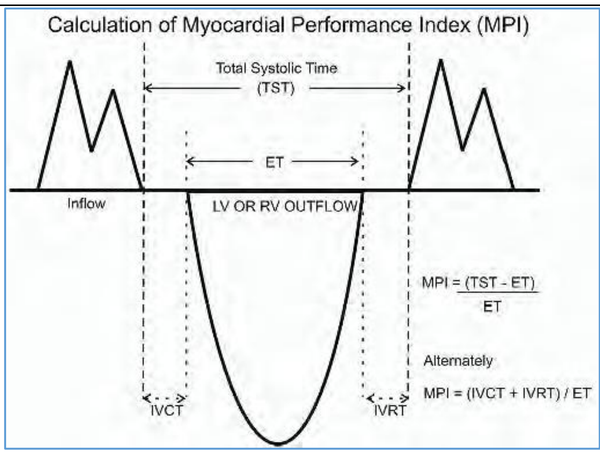
Most machines will provide reference lines at 1 and 3 m/sec and will calculate and display dP/dt automatically.
dP/dt should not be used if there is acute MR, or if there is significant AS or hypertension.
A normal LV will have a dP/dt > 1200 mmHg/s, whereas a severely impaired LV usually has a dP/dt < 800 mmHg/s

Calculating the left ventricular *dP/dt* from the CW Doppler MR spectral signal:
Left: A continuous wave spectral Doppler image recorded in a patient with severe LV systolic dysfunction in which the online measurement of *dP/dt* is noted to be 482 mm Hg/sec.
Right: The methodology for this determination, which includes recording CW Doppler imaging of MR at a high sweep speed (150 mm/sec in this example) and defining points for which the mitral regurgitation velocity has reached 1 and 3 m/sec, is depicted. This represents a 32 mm Hg/sec pressure increase in the LV into a low compliance left atrium, thus making this a relatively load-independent measure of contractility. The time between the two points required to reach 1 and 3 m/sec (Δt) is then divided into the pressure difference (32 mm Hg) for calculation of *dP/dt*.



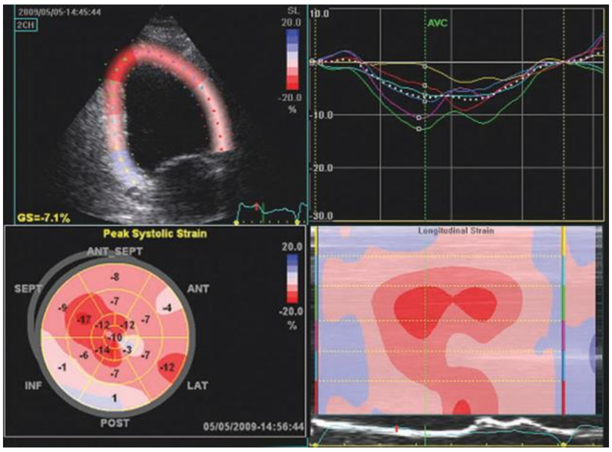
5. Myocardial perfusion index (MPI) or Tei index- is a PW Doppler derived index comparing the total isovolumic times with the ejection time. In both isovolumic times (IVRT & IVCT) both AV and MV are closed and there is no movement of blood.
MPI = (IVCT + IVRT) ÷ ET. i.e. it represents the proportion of the time in which there is no movement of blood to the ejection time. This index combines features of both systolic and diastolic function. Systolic dysfunction ↑ pre-ejection (IVCT) and ↓ ejection time (ET). Both systolic and diastolic dysfunction ↑ IVRT. A normal Tei index is < 0.4 (<0.3 for RV) with progressively greater values implying progressively worse ventricular function.

Schematic outlining calculation of the myocardial performance index (MPI). The myocardial performance index is the ratio of the sum of the isovolumic contraction and relaxation times (IVCT, IVRT) to ejection time (ET). It can be calculated by subtracting ET from total systolic time (TST) as noted in the two alternate formulas.
N.B: IVCT is short in normal systolic function with a rapid rise to peak pressure; both are altered with impaired function.



6. Annular systolic velocity- is a marker of longitudinal LV function (and global LV function in a uniformly contracting ventricle). Normal average of septal and lateral Ś > 7.5 cm/s (in other references >9-10)

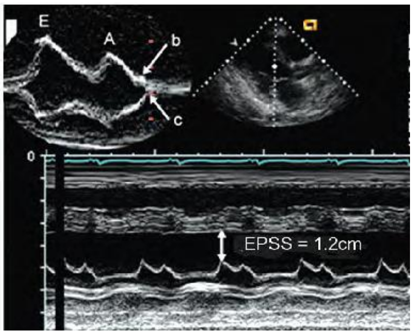
7. Global longitudinal strain (GLS)



- The most commonly used strain-based measure of LV global systolic function
- Obtained often with speckle tracking, less frequently with tissue Doppler imaging (TDI)
- GLS is the relative length change of the LV myocardium between end-diastole and end-systole
- GLS measurements obtained in the three standard apical views should be averaged
- GLS calculation can be obtained using endocardial, mid-wall, or average deformation
- Most of data come from mid-wall GLS, which is reproducible and robust
- In a healthy person, a peak GLS around −20% can be expected, and the lower the absolute value of strain is below this value, the more likely it is to be abnormal
- GLS decreases with age and is slightly higher in women
- GLS can be obtained from 2D or 3D

8. M-mode parameters of LV dysfunction

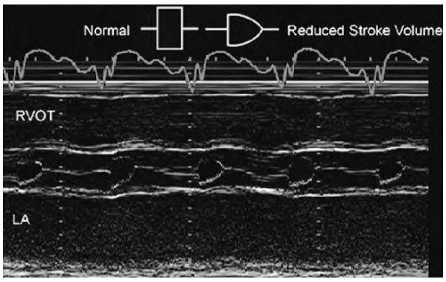
- **Mitral annular plane systolic excursion (MAPSE)** has been proposed as a useful echocardiographic parameter for the assessment of LV longitudinal function and correlates with global systolic function of the LV (similar to annular systolic velocity). MAPSE could be measured from four sites of the atrioventricular plane corresponding to the septal, lateral, anterior, and posterior walls using the apical 4C & 2C by M-mode echo. The average normal value for the four annular regions (septal, anterior, lateral, and posterior) ranged between 12-15 mm and a value of <8 mm was associated with a depressed LVEF and a mean value ≥10 mm was linked with preserved EF.



- **Increased MV E-point septal separation (EPSS)** (normal 6-7mm) due to increased LVEDD &/or decreased the magnitude of opening of the mitral valve (due to decreased stroke volume). EPSS is also increased with AR due to limited MV opening.

- **Interrupted (delayed) closure of MV with a B bump**, indicating transient reopening of the MV due to atrial contraction against noncompliant LV due to ↑LVDP).

- **Gradual curved (rounded) closure of the AV at end-systole** due to decreasing forward flow at the end of systole due to ↓LV SV



9. Other echo findings in systolic LV dysfunction:

- **Spontaneous echo contrast:** Severe LV impairment causes sluggish blood flow, which is thought to be related to the appearance of spontaneous echo contrast.
- **RWMA:** ischaemic LV dysfunction is the commonest cause although also encountered in cardiomyopathies, particularly sarcoidosis.