

ESSENTIALS OF CARDIOLOGY

A COMPREHENSIVE GUIDE FOR THEORY EXAMINATIONS

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CHAPTER 1.2

Echocardiography: Concepts and Applications

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Q1. Write a note on basic echo concepts.

1. Physics Fundamentals of Ultrasound

Understanding the physical principles of ultrasound is the foundation for interpreting echo images correctly.

1.1 Sound Wave Properties (Table 1)

TABLE 1: Sound wave properties in echocardiography.

Property	*Description*
Frequency	Clinical echo uses 1–12 MHz. Higher frequency = better resolution but less penetration
Wavelength	Inversely proportional to frequency. Determines axial resolution
Velocity	~1,540 m/s in soft tissue (assumed constant by echo machines)
Impedance	Product of tissue density × sound velocity. Mismatch at boundaries causes reflection

1.2 Axial versus Lateral Resolution

- **Axial resolution:** Shorter pulse duration = better axial resolution (depth direction).
- **Lateral resolution:** Determined by beam width at the focal zone. Narrows at focus, widens beyond.
- **Elevational resolution:** Determined by slice thickness; worst resolution plane in 2D echo.

2. Nyquist Limit and Aliasing

2.1 Definition

- **Nyquist limit:** Maximum Doppler frequency shift that can be accurately measured without aliasing. Equals half the pulse repetition frequency (PRF): Nyquist limit = PRF/2.
- If the true Doppler shift exceeds this limit, the signal wraps around, appearing on the opposite side of the baseline—this artifact is called aliasing.

2.2 Clinical Relevance

- **Pulsed-wave (PW) Doppler:** Most susceptible—samples at discrete intervals (PRF), limiting measurable velocity.
- **Continuous-wave (CW) Doppler:** Does *not* alias—samples continuously and can measure very high velocities (e.g., severe AS jet >4 m/s).
- **Color flow Doppler:** Aliases at lower velocities (typically >70 cm/s), causing a mosaic/mixed color pattern.

2.3 How to Overcome Aliasing

- Increase PRF (reduces depth simultaneously)
- Shift the baseline downward to extend the measurable range on one side
- Use a lower transducer frequency (reduces Doppler shift for a given velocity)

- Switch from PW to CW Doppler for high-velocity jets
- Use the high-PRF mode (allows some range ambiguity to achieve higher velocity measurement).

3. Doppler Modalities

Doppler echocardiography measures velocity of blood flow using the Doppler effect—the shift in sound frequency caused by moving red blood cells.

3.1 Pulsed-wave Doppler

- **Range-specific:** Samples flow at a defined depth (sample volume).
- **Limitation:** Subject to Nyquist aliasing at high velocities (>1.5–2 m/s).
- **Uses:** Mitral inflow (E/A ratio), pulmonary vein flow, LVOT velocity for stroke volume (SV) calculation.

3.2 Continuous-wave Doppler

- **No aliasing;** measures along the entire beam
- **Limitation:** No range specificity (range ambiguity).
- **Uses:** Severe aortic stenosis (AS) (peak gradient via modified Bernoulli), tricuspid regurgitation jet for RVSP.

3.3 Color Flow Doppler

- **Displays velocity/direction as color:** Red = toward transducer, blue = away (BART mnemonic).
- Based on PW principles; aliasing occurs as a mosaic of red, blue, and green at high velocities
- **Uses:** Localize regurgitation jets, septal defects, and stenosis jets.

3.4 Tissue Doppler Imaging

- Measures low-velocity, high-amplitude motion of the myocardium (opposite filter settings to blood flow Doppler)
- *e' velocity at mitral annulus:* Septal ≥**8 cm/s**, lateral ≥**10 cm/s** = normal diastolic function.
- E/e' ratio **>14** suggests elevated left ventricular (LV) filling pressure (diastolic dysfunction grade II-III)

4. The Modified Bernoulli Equation

Used to convert peak Doppler velocity into a pressure gradient across a stenosis or regurgitant orifice: $\Delta P = 4V^2$, where ΔP = pressure gradient (mm Hg) and V = peak velocity (m/s). The proximal velocity is ignored when <1 m/s.

Key applications of the modified Bernoulli equation are summarized in **Table 2**.

TABLE 2: Key applications of the modified Bernoulli equation.

Application	*Criterion*
Aortic stenosis (AS)	Mean gradient >40 mm Hg (peak V > 4 m/s) = severe AS
RVSP/PA pressure	DP (TR) + RAP estimate = RVSP. Normal <35 mm Hg
VSD gradient	Systolic BP − VSD gradient = Right ventricular (RV) pressure
Mitral stenosis (MS)	Mean gradient >10 mm Hg = severe MS (use planimetry + PHT too)

5. Pulse Repetition Frequency and Depth

- PRF is the number of ultrasound pulses emitted per second, directly linked to imaging depth: Depth = c/(2 × PRF)
- **Higher PRF:** Higher Nyquist limit, less aliasing, and shallower depth.
- **Lower PRF:** Greater depth, lower Nyquist limit, and more aliasing risk.
- Frame rate in 2D imaging is also limited by depth: wider sector and deeper imaging = lower frame rate.

6. Diastolic Function Assessment

- Diastolic dysfunction is graded using a combination of parameters as per the 2016 ASE/EACVI guidelines **(Table 3)**.
- Indeterminate pattern when >50% of the four criteria are not met. Clinical correlation needed.

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TABLE 3: Key parameters for diastolic function assessment (2016 ASE/EACVI).

Parameter	*Description/cutoff*
Mitral E/A ratio	E = early diastolic filling; A = atrial kick. Normal: 0.8–2.0
E/e' ratio	Correlates with LV filling pressure. >14 = elevated filling pressure
e' velocity	Septal <7 cm/s or lateral <10 cm/s = impaired relaxation
Left atrial (LA) volume index	>34 mL/m^2 = LA enlargement (chronic pressure elevation)
TR Vmax	>2.8 m/s supports elevated filling pressure

7. Valve Area Calculations

7.1 Continuity Equation (Aortic Stenosis)

Based on conservation of mass—flow proximal must equal flow distal to stenosis:

- **AVA = (LVOT area × VTI1)/VTI2**
- LVOT diameter measured in parasternal long axis (PLAX) in midsystole (0.5–1 cm below aortic valve)
- VTI1 = LVOT VTI (PW Doppler); VTI2 = AV VTI (CW Doppler)
- **AVA <1.0 cm^2 = severe AS;** 1.0–1.5 cm^2 = moderate; >1.5 cm^2 = mild

7.2 Pressure Half-time (Mitral Stenosis)

- *Pressure half-time (PHT):* Time for the peak E-wave pressure gradient to fall by half. **MVA = 220/PHT.**
- **PHT <100 ms = normal; PHT ≥220 ms = severe mitral stenosis (MS)** (MVA ≤1 cm^2)
- **Not reliable** immediately post-valvuloplasty or in significant aortic regurgitation (AR) (changes LV compliance).

7.3 Proximal Isovelocity Surface Area Method (Regurgitation)

Proximal isovelocity surface area (PISA)—measures regurgitant flow using flow convergence: **EROA = 2 × π × r^2 × Va/Vmax**

- **EROA >0.40 cm^2 = severe MR;** >0.20 cm^2 = severe TR
- **Regurgitant volume = EROA × VTI** of regurgitant jet. >60 mL = severe MR

8. Left Ventricular Systolic Function (Table 4)

TABLE 4: LVEF classification (ASE/EACVI).

Category	*LVEF range*
Normal	EF ≥52% for male, ≥54% for female (biplane Simpson's method preferred)
Mildly reduced	41–52%
Moderately reduced	30–40%
Severely reduced	<30%

8.1 Stroke Volume and Cardiac Output

- **SV = LVOT area × LVOT VTI; Cardiac output (CO) = SV × HR**
- LVOT area = π(d/2)^2, where d = LVOT diameter
- *Normal SV:* 60–100 mL; normal CO: 4–8 L/min.
- VTI >18 cm at LVOT correlates with adequate SV

8.2 Global Longitudinal Strain

It has been discussed in detail in the section on Strain Imaging (Q7).

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9. Common Echo Artifacts and Pitfalls (Table 5)

TABLE 5: Common echo artifacts and pitfalls.

Artifact/pitfall	*Appearance*	*Key cause*	*Correction/clue*
Reverberation	Multiple parallel echoes	Repeated reflections	Change angle/gain
Acoustic shadowing	Signal dropout behind structure	Calcium/prosthetic valve	Alternate window
Aliasing	Color wrap-around	Velocity > Nyquist limit	Increase scale
Dropout artifact	False ASD/VSD/gap	Poor beam alignment	Reimage in other view
Blooming	Overestimated jet size	Excess color gain	Reduce gain
Foreshortened left ventricle (LV)	Small rounded apex	Apical malalignment	True apex view
Mirror image artifact	Duplicate structure	Strong reflector	Different imaging plane

Q2. Write a note on echo assessment of systolic LV function.

1. Introduction

- Echocardiography is the primary noninvasive technique for assessment of LV function in daily cardiology practice.
- Left ventricle is a pressure-generating and flow-generating chamber.
- Systolic performance reflects contractility interacting with preload, afterload, synchrony, and chamber geometry.

2. Echo Windows and Acquisition Essentials

- Reliable systolic function assessment depends on proper acquisition.
- **Basic views:** PLAX, parasternal short-axis (basal, mid, and pical levels), apical four-chamber, apical two-chamber, apical long-axis (three-chamber), and subcostal views.
- **Endocardial border definition is central:** Harmonic imaging, contrast echo, and optimization of gain and depth are essential when borders are not clear.
- In M-mode and linear measurements, the ultrasound beam should be perpendicular to the LV long axis to avoid oblique measurements.
- **Apical foreshortening must be actively avoided**: Underestimates true LV length and therefore underestimates volumes while sometimes overestimating ejection fraction (EF).

3. Components of Systolic Function

- **Global systolic function:** EF, fractional shortening (FS), mitral annular plane systolic excursion (MAPSE), tricuspid annular plane systolic excursion (TAPSE), tissue Doppler S′, dP/dt, SV, 3D EF, and global longitudinal strain (GLS).
- **Regional systolic function:** Wall thickening and endocardial excursion in all coronary territories.
- **Mechanical coordination:** Dyssynchrony, postsystolic shortening, and timing dispersion.
- **Remodeling:** Cavity size, wall thickness, relative wall thickness, LV mass, geometry, and left atrial (LA) consequences.
- **Hemodynamic function:** Forward flow, LVOT VTI, valvulo-arterial interactions, and interpretation under changing loading conditions.

4. Global Left Ventricular Systolic Function

4.1 Qualitative Assessment (Eyeballing)

- Qualitative assessment or "eyeballing" remains the first step
- *Should integrate:* Cavity size, endocardial excursion, inward thickening, mitral annular motion, apex-to-base shortening, and overall vigor of contraction.
- **Hyperdynamic LV:** Suggests increased sympathetic drive, hypovolemia, anemia, sepsis, or significant mitral regurgitation (MR).
- **Mild dysfunction:** Reduced thickening and excursion, usually without major cavity dilatation.

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- **Severe dysfunction:** Large spherical cavity, poor thickening, mitral annular flattening, and little apical movement.
- Visual estimation is quick and often clinically useful but should be confirmed by quantitative methods.

4.2 Linear Methods: Fractional Shortening and Fractional Area Change

Fractional shortening: Measure of LV systolic function based on change in LV internal diameter during the cardiac cycle. Reflects radial (transverse) myocardial contraction.

Formula: FS (%) = [(LVEDD – LVESD)/LVEDD] × 100

- **LVEDD** = LV end-diastolic diameter; **LVESD** = LV end-systolic diameter
- Obtained from M-mode or 2D echo (PLAX/short-axis view); measured at level of mitral valve (MV) leaflet tips or papillary muscles
- **Normal FS: 25–45%.**
- **Reduced FS (<25%):** LV systolic dysfunction.
- **Increased FS (>45%):** Hyperdynamic states (e.g., anemia and sepsis).
- Assumes normal LV geometry
- Not reliable in regional wall motion abnormalities (RWMAs), LV hypertrophy, or LV dilatation with distorted shape
- Only reflects radial function, not longitudinal function

Fractional area change (FAC): Measure of systolic function using change in ventricular cavity area. Commonly used for right ventricle (RV).

Formula: FAC (%) = [(EDA – ESA)/EDA] × 100.

- **EDA** = End-diastolic area; **ESA** = End-systolic area
- **Normal RV FAC: ≥35%** (normal RV systolic function).
- **FAC < 35%:** RV systolic dysfunction.
- Image quality dependent; errors in endocardial border tracing
- Load dependent (preload/afterload changes affect values)
- RV geometry complexity may still limit accuracy

4.3 LVEF Measurement Methods

Ejection fraction (EF): EF (%) = [(LVEDV – LVESV)/LVEDV] × 100

Area-length method:

- LV divided into a stack of small disks from apical four-chamber and apical two-chamber views
- Endocardial borders traced at end-diastole and end-systole
- **Advantages:** More accurate than linear measurements; better reflects global LV systolic function; useful in RWMAs.
- **Limitations:** Depends on good image quality; foreshortening of apical views can cause error; less accurate in arrhythmias and markedly abnormal LV geometry.
- Assumes LV as a prolate ellipse. **Formula: LV volume = (5/6) × area × length.**
- Area from PSAX view; length from apex to mitral annulus in apical two-chamber view
- **Limitation:** Geometric assumption dependent; less accurate in distorted LV.

Strain imaging:

- **Formula:** Strain (%) = [(LES – LED)/LED] × 100.
- **GLS normal:** −18 to −22% (approximately); more negative = better function.
- **Longitudinal strain:** Most reproducible; derived from subendocardial fibers.
- **Radial strain:** Myocardial thickening (positive values).
- **Circumferential strain:** Shortening along LV circumference; generated by mid-myocardial fibers.
- **Key concept:** Most cardiac diseases affect subendocardium first; longitudinal strain is affected earliest.
- **Apical sparing in amyloidosis:** Cardinal pattern distinguishing amyloidosis from other causes of LVH.
- **Advantages:** Detects subclinical LV dysfunction; useful in chemotherapy cardiotoxicity, HFpEF, early ischemia; less load dependent than EF.

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- **Limitations:** Dependent on image quality; vendor variability; requires dedicated software.
- Full-volume dataset acquired; software calculates LVEDV, LVESV, and EF.
- **Advantages:** Less operator dependent, more reproducible, and no geometric assumptions (especially 3D).
- **Limitations:** Needs good image quality; stitch artifacts in arrhythmias; limited availability.

4.4 Cardiac Output by Echocardiography

- **SV = VTI × CSA; CO = SV × HR; CI = CO/BSA**
- **Step 1:** Measure LVOT diameter from PLAX view at level of aortic annulus.
- **Step 2:** Calculate CSA = $\pi(D/2)^2$.
- **Step 3:** Measure LVOT VTI using PW Doppler (apical 5C/3C view).
- **Step 4:** Calculate SV, CO, and CI.
- **SV:** ~60–100 mL; **CO:** ~4–8 L/min; **CI:** ~2.5–4.0 L/min/m^2.
- Error in LVOT diameter causes major error in SV (squared relationship)
- Assumes circular LVOT; Doppler alignment errors; arrhythmias affect accuracy.

4.5 dP/dt (Rate of Rise of LV Pressure)

- Measures the rate of rise of LV pressure during isovolumic contraction; reflects LV contractility
- **Method:** Derived from CW Doppler of MR jet. Measure time for velocity to increase from 1 m/s to 3 m/s.
- **dP/dt = 32/time (second)**
- **Normal dP/dt: >1,200 mm Hg/second;** Reduced (<1,000 mm Hg/second) = LV systolic dysfunction.
- **Advantage:** Relatively load independent; useful in MR patients.
- **Limitation:** Requires adequate MR jet; not usable if no MR present.

4.6 Tei Index (Myocardial Performance Index) (Fig. 1)

- Combined index of systolic + diastolic function. **Formula: Myocardial performance index (MPI) = (IVCT + IVRT)/ET**
- **IVCT** = Isovolumic contraction time; **IVRT** = Isovolumic relaxation time; **ET** = Ejection time
- **Normal MPI:** ~0.39 ± 0.05

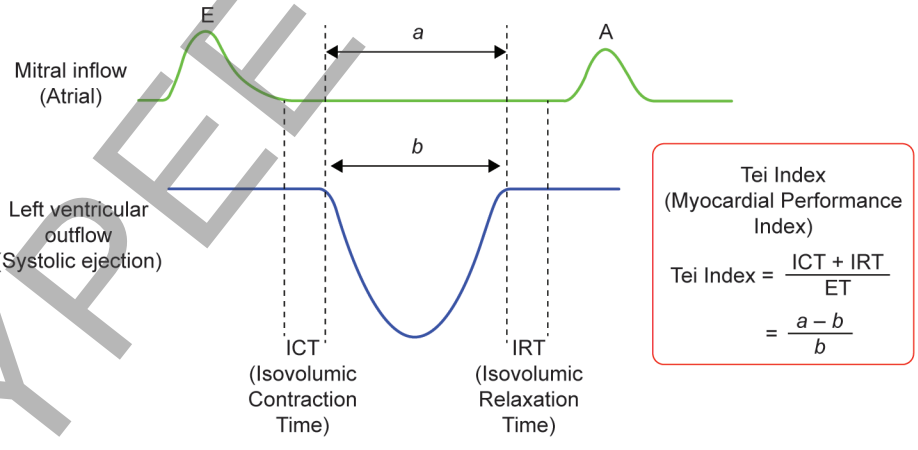

Fig. 1: Schematic representation of the Tei index [myocardial performance index (MPI)]. MPI = (IVCT + IVRT)/ET. (ET: ejection time; IVCT: isovolumic contraction time; IVRT: isovolumic relaxation time)
Note: A higher MPI indicates worse ventricular function. Normal MPI ~0.39 ± 0.05 (by PW Doppler).

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- **Higher MPI = Worse ventricular function:** IVCT and IVRT increase; ET decreases
- **Advantages:** Combines systolic + diastolic function; less dependent on ventricular geometry; can be applied to RV.
- **Limitations:** Affected by heart rate and rhythm; less reliable in atrial fibrillation (AF), significant valvular disease; load dependent.

4.7 E-point Septal Separation

- Distance between anterior MV leaflet (E-point) and interventricular septum; measured in PLAX view using M-mode
- **Normal E-point septal separation (EPSS): <5–7 mm.**
- **>7 mm:** Suggests LV systolic dysfunction; **>10 mm:** severe LV systolic dysfunction.
- **Physiological basis:** In normal LV, strong contraction moves anterior MV leaflet close to septum; in dysfunction, dilated LV + reduced SV prevents this.
- **Not reliable in:** MS, severe MR, AR, LV outflow obstruction, abnormal septal motion (LBBB and RV pacing).
- Cannot replace Simpson biplane EF.

4.8 S′ (S prime)—Tissue Doppler Imaging

- Peak systolic velocity of myocardium at mitral annulus (LV function) or tricuspid annulus (RV function); measured using tissue Doppler imaging (TDI) in apical four-chamber view.
- **Normal LV S′ (lateral): ≥10 cm/s; Normal RV S′: ≥9.5–10 cm/s.**
- **Reduced S′:** Systolic dysfunction; reflects longitudinal myocardial contraction.
- **Early detection:** Reduces before EF falls (subendocardial fibers affected first).
- **Limitation:** Angle dependent; affected by tethering, translational motion, and load.

4.9 Mitral Annular Plane Systolic Excursion

- Longitudinal displacement of mitral annulus toward apex during systole. Measured via M-mode in apical four-chamber with cursor through lateral mitral annulus.
- **Normal: ≥10–12 mm;** reduced MAPSE = LV systolic dysfunction.
- **Limitation:** Angle dependent; not reliable in mitral annular calcification or prosthetic valves.

4.10 Tricuspid Annular Plane Systolic Excursion

- Longitudinal displacement of tricuspid annulus toward apex during systole. Measured via M-mode in apical four-chamber with cursor through lateral tricuspid annulus
- **Normal: ≥17 mm;** <17 mm = RV systolic dysfunction.
- **Limitation:** Angle dependent; load dependent; assesses only longitudinal RV function; may be misleading in severe tricuspid regurgitation.

Q3. Write a note on dobutamine stress echocardiography.

1. Definition and Overview

- Dobutamine stress echocardiography (DSE) is a noninvasive pharmacological stress test that uses incremental intravenous doses of dobutamine—a synthetic catecholamine with predominant β1-adrenergic agonist activity—to simulate the physiological demands of exercise on the myocardium.
- Real-time echocardiographic imaging is performed simultaneously to evaluate wall motion, cardiac function, and myocardial viability.
- The test exploits the principle that ischemic or hibernating myocardium will demonstrate characteristic changes in wall motion and thickening in response to increasing inotropic and chronotropic demand.

1.1 Mechanism of Action—Dobutamine

- Predominantly stimulates β1-adrenoceptors; increases heart rate, contractility (positive inotrope), and myocardial oxygen demand
- **Low-dose effect (5–10 µg/kg/min):** Primarily inotropic; augments contractility; recruits hibernating myocardium (biphasic response).

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- **High-dose effect (20–40 µg/kg/min):** Increases heart rate, further raises oxygen demand; provokes ischemia in territories supplied by significantly stenosed coronaries.
- **Atropine (0.25–1 mg IV):** Added if target heart rate is not achieved with dobutamine alone.

2. Indications

2.1 Diagnosis of Coronary Artery Disease

- Detection of obstructive coronary artery disease (CAD) in symptomatic patients with intermediate pretest probability
- Patients with inconclusive or uninterpretable ECG (LBBB, paced rhythm, LVH with ST changes, and preexcitation)
- Patients with equivocal exercise stress test results
- Evaluation of chest pain syndromes where anatomy is uncertain.

2.2 Assessment of Myocardial Viability

Dobutamine stress echocardiography distinguishes three states of dysfunctional myocardium:

- **Stunned myocardium:** Dysfunctional but viable; intact cellular integrity.
- **Hibernating myocardium:** Chronically hypoperfused viable myocardium; function improves at low-dose dobutamine (5–10 µg/kg/min); hallmark is the biphasic response.
- **Scarred/infarcted myocardium:** Nonviable; no response to dobutamine at any dose.

Clinical utility: Predicts likelihood of functional recovery after revascularization (PCI or CABG). Segments showing the biphasic response have the highest predictive value for recovery postrevascularization.

2.3 Preoperative Risk Stratification

- Prior to major noncardiac surgery (vascular, orthopedic, and abdominal) in intermediate-to-high-risk patients
- Especially useful when functional capacity is poor or unknown (<4 METs).

2.4 Evaluation of Known Coronary Artery Disease

- Assessment of hemodynamic significance of intermediate coronary stenoses identified on angiography
- Postrevascularization assessment of procedural success and residual ischemia

2.5 Valvular Heart Disease

- **Low-flow low-gradient aortic stenosis (LFLG-AS):** Differentiates true severe AS (small increase in area, significant rise in gradient) from pseudosevere AS (normalization of valve area, minimal gradient change) using low-dose DSE.
- **Contractile reserve:** Improvement in LVEF ≥20% predicts better surgical outcomes.
- Evaluation of MR severity under dynamic conditions.

2.6 Other Indications

- **HOCM:** Assessment of dynamic LVOTO.
- Cardiac transplant recipients for allograft vasculopathy (who cannot exercise normally)
- Assessment after acute myocardial infarction—identifying infarct-related artery territory ischemia and viability.

3. Protocol and Test Conduct

3.1 Standard Protocol (Table 6)

TABLE 6: Dobutamine stress echocardiography protocol stages.

Stage	*Dobutamine dose/action*
Stage 1 (low dose)	5 µg/kg/min for 3 minutes
Stage 2	10 µg/kg/min for 3 minutes
Stage 3	20 µg/kg/min for 3 minutes
Stage 4	30 µg/kg/min for 3 minutes
Stage 5 (peak dose)	40 µg/kg/min for 3 minutes
Atropine (if needed)	0.25 mg IV boluses up to 1–2 mg total if target HR not achieved

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Target heart rate = 85% of maximum predicted heart rate (MPHR); MPHR = 220 - Age (in years).

For viability assessment: only low-dose stages (5–10 mcg/kg/min) are used; high doses are not needed.

3.2 Imaging

- *Standard 2D echocardiographic views:* PLAX, parasternal short-axis, apical four-chamber, and apical two-chamber.
- Images acquired at baseline, each dose stage, and recovery (5–10 minutes postinfusion)
- Side-by-side comparison of wall motion using quad-screen display
- TDI and myocardial strain may be added for quantification.

3.3 End Points for Test Termination

- Achievement of target heart rate
- New or worsening wall motion abnormalities in ≥2 segments
- Sustained ventricular tachycardia or other serious arrhythmias
- Severe hypertension (SBP >220 mm Hg or DBP >120 mm Hg)
- Significant hypotension (SBP drop >40 mm Hg)
- *Severe symptoms:* Angina, dyspnea, and presyncope.
- ST-segment changes (≥2 mm horizontal or downsloping depression).

4. Interpretation of Results

4.1 Wall Motion Scoring

Each of the **17 myocardial segments** (American Heart Association model) is assessed using the Wall Motion Score Index (WMSI) **(Table 7)**.

TABLE 7: Wall motion score index (WMSI)—AHA 17-segment model.

Score	*Definition*
1	Normokinesia—normal inward motion and thickening
2	Hypokinesia—present, but reduced inward motion
3	Akinesia—absent inward motion, no thickening
4	Dyskinesia—paradoxical outward motion during systole
5	Aneurysmal—diastolic deformation with dyskinesis

WMSI = Sum of individual segment scores/Number of segments scored. Normal WMSI = 1.0; higher values indicate more extensive dysfunction.

4.2 Positive Test for Ischemia

- New wall motion abnormality (hypokinesia, akinesia, and dyskinesia) in a previously normal segment at stress
- Worsening of a preexisting abnormality to a higher score at stress
- Failure of LV cavity to diminish appropriately (ischemic dilatation)
- Global LV dysfunction (EF decrease) at peak stress without alternative explanation.

4.3 Biphasic Response—Viability

Hallmark of hibernating myocardium:

- **Baseline:** Resting wall motion abnormality (hypo/akinesia).
- **Low dose (5–10 µg/kg/min):** Improvement or normalization of wall motion.
- **High dose (≥20 µg/kg/min):** Worsening or return of abnormality due to ischemia.
- Sustained improvement (without worsening at high dose) suggests stunned myocardium or small-vessel disease.

4.4 Diagnostic Performance and Response (Table 8)

- *Sensitivity for CAD:* ~80–85%.
- *Specificity for CAD:* ~85–90%.
- *Sensitivity for viability:* ~80–85% (biphasic response).

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TABLE 8: DSE response patterns and interpretation.

DSE response	*Wall motion change*	*Viability*	*Clinical meaning*
Normal myocardium	Normal → hyperdynamic	Viable	Normal perfusion/function
Sustained improvement	Improves at low dose; remains improved	Viable	Myocardial stunning
Biphasic response	Improves at low dose → worsens at high dose	Viable + ischemic	Hibernating myocardium (best predictor of recovery)
Worsening only	Deteriorates with stress	Ischemia	Viable but flow-limiting CAD
No change	Persistent akinesia/hypokinesia	Nonviable	Scar/fibrosis
Dyskinesia, no improvement	No contractile reserve	Nonviable	Transmural scar
Improvement in ≥5 segments	Significant viable myocardium	Viable	Benefit from revascularization likely

(CAD: coronary artery disease; DSE: dobutamine stress echocardiography)

5. Contraindications

5.1 Absolute Contraindications

- Acute myocardial infarction (within 2–7 days) or unstable angina (not stabilized)
- Severe systemic hypertension—SBP >180 mm Hg or DBP >110 mm Hg at rest
- Significant cardiac arrhythmias—uncontrolled rapid AF, sustained VT, advanced AV block (second/third degree) without a pacemaker.
- Known severe LVOTO (HOCM with significant gradient) when used at high doses
- Aortic dissection (acute) or uncontrolled heart failure decompensation
- Hypersensitivity or allergy to dobutamine; severe uncontrolled hyperthyroidism

5.2 Relative Contraindications

- Moderate AS (mean gradient 25–40 mm Hg)
- Frequent ectopics or nonsustained VT on baseline ECG
- Moderate systemic hypertension (BP 160–180/100–110 mm Hg)
- **Note:** DSE preferred over vasodilator stress (adenosine/dipyridamole) in asthma/COPD–vasodilators cause bronchospasm.
- Recent stroke (<2 months) or TIA; known intracardiac thrombus; pregnancy (theoretical risk)

6. Complications and Safety

6.1 Common Side Effects

- Palpitations, anxiety, tremor (sympathomimetic effects—transient)
- Chest discomfort; hypertension or hypotension

6.2 Serious Complications (Rare)

- **Sustained VT or VF:** <0.05%; **MI:** <0.01%; **Death:** <0.02%
- Severe hypotension or hypertensive crisis; eosinophilic myocarditis (rare)

6.3 Reversal Agent

Short-acting β-blocker: Metoprolol (IV) or esmolol. Dobutamine half-life ~2 minutes, so adverse effects resolve rapidly on stopping infusion.

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Q4. Write a note on diastolic stress test echocardiography.

1. Definition

Diastolic stress echocardiography is an exercise-based echocardiographic test designed to unmask abnormal LV diastolic reserve by demonstrating exercise-induced elevation of LA pressure or pulmonary capillary wedge pressure using Doppler surrogates such as mitral E/e' and tricuspid regurgitation velocity.

- Most useful when symptoms are exertional but resting echocardiography is nondiagnostic.
- Central to the modern work-up of suspected HFpEF where patients have normal or borderline filling pressures at rest yet develop abnormal filling pressures during exercise.

2. Pathophysiological Basis

- Exercise increases venous return, heart rate, blood pressure, and afterload, and shortens diastolic filling time
- **Normal ventricle:** Compensates with rapid relaxation, preserved suction, and proportional augmentation of transmitral flow and annular relaxation velocity; filling pressure does not rise excessively.
- **Diseased ventricle:** Cannot compensate; LA pressure rises disproportionately.
- HFpEF affects mainly older adults with hypertension, obesity, diabetes, atrial disease, or renal dysfunction; resting echo may miss early or intermittent abnormalities.

3. Indications

- **Primary indication:** Unexplained exertional dyspnea with suspected HFpEF when resting EF is preserved and standard resting data are normal, borderline, or inconclusive.
- The 2025 ASE update indicates the test in patients with dyspnea and **grade 1 diastolic dysfunction at rest** and in patients with normal resting LA pressure but clinical suspicion of HFpEF
- **Clinical clues raising pretest probability:** Older age, female sex, obesity, hypertension, diabetes, CKD, AF history, exercise intolerance, and borderline natriuretic peptide elevation.
- **Resting echo clues:** E/e' values suspicious but not frankly abnormal, mild LA enlargement, reduced LA reservoir strain (LASr), or subtly reduced GLS despite preserved EF.
- **Other situations:** Hypertrophic cardiomyopathy (HCM), athletes with dyspnea (physiologic vs. disease), microvascular disease, selected valvular or infiltrative disorders.

4. When Not to Rely on It?

- Noninvasive interpretation is difficult in: significant mitral annular calcification, prior MV repair or prosthesis, moderate or severe MR, constrictive pericarditis, marked regional dysfunction, LBBB, important AR, or absent/poor TR signal.
- In such settings, invasive exercise hemodynamics may be more appropriate.

5. Exercise Modality

- **Exercise (not dobutamine) is the preferred stressor** for diastolic assessment—dobutamine increases lusitropy, potentially masking the abnormality.
- **Supine bicycle exercise:** Generally considered optimal as it permits Doppler acquisition during exercise at staged workloads.
- **Treadmill exercise:** More common but postexercise acquisition may miss the brief diagnostic window as diastolic variables can normalize rapidly after stopping.

6. Technical Protocol

- Record baseline resting images, blood pressure, rhythm, transmitral inflow, septal and lateral e', TR velocity, and resting ventricular and atrial structure
- Use bicycle exercise with graded workloads and acquire apical Doppler windows during exercise, not only afterward
- Target data collection before marked tachycardia causes E and A fusion (problematic at HR above ~110 bpm)

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- Record symptoms, workload, heart rate, blood pressure, and whether the clinical complaint is reproduced.
- Acquire exercise E/e' and exercise TR velocity at the stage most relevant to symptoms.

7. Parameters to Record

- Mitral inflow E and A velocities at rest and during exercise
- Septal and lateral e' velocities with calculation of average E/e'
- Peak TR velocity during exercise to estimate exercise pulmonary pressure response
- Resting LA volume index (LAVI) and baseline global assessment of LV structure and EF
- Blood pressure, rhythm, heart rate, symptom reproduction, and workload achieved
- *Newer/adjunctive markers:* LA strain, GLS, untwisting indices, PASP/CO slope, lung ultrasound B-lines—not yet required in every laboratory protocol.

8. Interpretation

Positive diastolic stress test: Exercise produces abnormal E/e' and a concomitant rise in TR velocity in a patient whose symptoms and clinical phenotype fit HFpEF or exertional diastolic dysfunction.

- **E/e' >14–15 together with exercise TR velocity >3.2 m/s = positive**
- **Negative test:** Suggests preserved diastolic reserve; does not by itself prove symptoms are noncardiac.
- **False-negative studies:** Data acquired too late, insufficient workload, E and A fusion too early, TR jet not measurable, or pericardial/valvular/pulmonary vascular limitation.

Q5. Write a note on contrast echocardiography.

1. Definition and Overview

- **Contrast echocardiography:** Cardiac ultrasound using gas-filled microbubbles [ultrasound contrast agents (UCAs)] administered intravenously to enhance acoustic reflectivity of blood.
- Markedly improves delineation of cardiac structures, endocardial borders, and myocardial perfusion.
- Microbubbles are pure intravascular tracers—remain within the bloodstream (do not cross into the interstitium) and are excreted via the lungs after phagocytosis or destroyed by the ultrasound beam.

2. Ultrasound Contrast Agents

2.1 Composition

- **Microscopic gas-filled spheres (1–10 µm diameter)** stabilized by a shell of phospholipid, albumin, or polymer
- **Gas core:** High-molecular-weight, low-solubility gas (octafluoropropane—C_3F_8, or sulfur hexafluoride—SF_6) that resists dissolution in blood.

2.2 Commercially Available Agents (Table 9)

TABLE 9: Commercially available ultrasound contrast agents (UCAs).

Agent (Trade name)	*Shell/gas*
SonoVue (Lumason)	Phospholipid/SF_6
Definity (MRX-115)	Phospholipid/octafluoropropane
Optison	Albumin/octafluoropropane
Echovist/Levovist	Galactose shell/air (first generation—largely obsolete)

2.3 Mechanism of Action

- When exposed to ultrasound energy, microbubbles undergo nonlinear oscillation (resonance), generating harmonic signals distinct from surrounding tissue
- Modern contrast-specific imaging modes (e.g., pulse-inversion harmonic imaging and power modulation) selectively detect these harmonic signals, improving signal-to-noise ratio.

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3. Clinical Indications and Uses

3.1 Left Ventricular Opacification—Primary Indication

- **Indication:** Two or more endocardial segments not visualized on resting TTE.
- Improves accuracy of LVEF assessment; enhances detection of RWMA
- Particularly valuable in obese patients, COPD, postsurgical patients, and ICU patients
- Reduces interobserver variability in LVEF measurement.

3.2 Stress Echocardiography

- Significantly improves diagnostic yield of stress echo in patients with suboptimal image quality
- Allows reliable detection of stress-induced RWMA; reduces nondiagnostic study rates from ~30% to <5%
- Combined with myocardial perfusion imaging (MPI) for dual assessment of function and perfusion.

3.3 Myocardial Perfusion Imaging

- At low mechanical index (MI <0.3), microbubbles remain intact within myocardial capillaries. Ischemic or infarcted zones appear as **perfusion defects**.
- Detection of CAD; assessment of myocardial viability (hibernating vs. stunned vs. infarcted)
- **Microvascular obstruction (MVO):** "No-reflow" phenomenon postprimary PCI.

3.4 Assessment of Cardiac Masses/Thrombi

- **LV thrombus:** Appears as a nonenhancing, avascular filling defect; contrast significantly increases sensitivity for LV apical thrombus (sensitivity 61% → >90%).
- **Cardiac tumors:** Myxomas show heterogeneous enhancement; malignant tumors show heterogeneous vascularity; thrombus shows no enhancement.

3.5 Congenital Heart Disease

- **Agitated saline contrast (bubble study):** Detects right-to-left shunts—PFO, ASD, and pulmonary arteriovenous malformations (PAVMs).
- **Intracardiac shunts:** Microbubbles appear in left heart within 3–5 cardiac cycles; later appearance (>5 cycles) suggests pulmonary AVM.

3.6 Ventricular Structural Assessment

- **HCM:** Accurate detection of apical variant in particular.
- **Noncompaction cardiomyopathy (LVNC):** Demonstrates deep trabeculations and sinusoids filled with contrast.
- **Ventricular aneurysm versus pseudoaneurysm:** Pseudoaneurysm has a narrow neck with contrast entry.

3.7 Miscellaneous

- Prosthetic valve function and paravalvular regurgitation assessment
- **Alcohol septal ablation in HCM:** Contrast delineates target septal perforator territory.
- *Critical care/ICU:* Rapid bedside assessment of LVEF when CT is not feasible.

4. Contraindications

4.1 Absolute Contraindications

- **Known hypersensitivity/allergy:** Prior serious adverse reaction to any UCA or its components.
- **Severe pulmonary hypertension:** PA systolic pressure >90 mm Hg (risk of acute worsening)—now often listed as relative per ESC/ASE.
- **Intracoronary use:** Not approved in most countries; rapid bolus can cause ECG changes and hemodynamic instability.

4.2 Relative Contraindications

- **Acute coronary syndrome:** Microbubbles may worsen ischemia via coronary embolization.

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- **Intracardiac or pulmonary right-to-left shunt:** Microbubbles bypass pulmonary capillary filter; now listed as relative contraindication—assess risk versus benefit.
- Respiratory failure, worsening heart failure, ventricular arrhythmias (high-MI ultrasound + UCA can rarely trigger VA)
- *Pregnancy and lactation:* Safety not established; hepatic/renal impairment may require dose adjustment.

Q6. Write a note on three-dimensional echocardiography (3DE).

Introduction

- **Three-dimensional echocardiography:** Advanced cardiac ultrasound technique that acquires volumetric datasets to provide spatial, anatomical, and functional information beyond standard 2D imaging.
- Overcomes the fundamental limitation of 2D echo—the need to geometrically model complex cardiac structures from multiple 2D planes

1. Technical Principles

Matrix Array Transducer

- **~2,000–3,000 piezoelectric elements** arranged in a 2D grid; allows electronic beam steering in both azimuthal and elevation planes without mechanical movement
- **Frequency range:** 2–4 MHz (adults); higher for pediatric/TEE probes.
- Full-volume datasets typically a 60° × 60° or 90° × 90° pyramid

ECG Gating

- **Multi-beat gating (typically 4–7 beats):** Stitches subvolumes together at end-expiration, increasing spatial and temporal resolution.
- **Stitching artifacts:** Occur if arrhythmia or breathing disrupts alignment between beats.
- **Single-beat acquisition:** Avoids stitching but sacrifices temporal resolution.

3D Transesophageal Echocardiography

- **Higher frequency probe (5–7 MHz):** Superior resolution.
- **Preferred for:** MV anatomy, LA appendage, interatrial septum, and structural heart disease (SHD) procedures.
- **"Surgeon's view" of MV:** En face from the left atrium looking toward the ventricle—gold standard for MV surgical planning.

2. Types of 3D Acquisition Modes (Table 10)

TABLE 10: 3D echocardiography acquisition modes.

Mode	*Volume size*	*Frame rate*	*Key use*
Live 3D (narrow angle)	Small pyramid	High	Valve morphology, procedural guidance
Live 3D zoom	Cropped volume	Moderate	Valve en face views, structural procedures
Full volume (four-beat gated)	Full LV/RV	Moderate	Volumetry, mass, EF
Full volume (single beat)	Full LV/RV	Lower	Atrial fibrillation, poor breath-holders
3D color Doppler	Volume + flow	Low	Regurgitation quantification

(LV: left ventricle; RV: right ventricle)

3. Clinical Indications

3.1 Left Ventricular Assessment

- **3D LV volumetry:** Most accurate noninvasive method for EDV, ESV, and EF—without geometric assumptions.
- **Reference standard:** MRI (3DE correlates better with MRI than 2D biplane Simpson's, which underestimates LV volumes by ~10–20%).

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- **Recommended for:** Serial monitoring of LV function in chemotherapy-related cardiotoxicity, valvular heart disease follow-up, and heart failure.
- 3DE-derived LV mass is more reproducible than 2D M-mode or area-length methods; key in HCM and hypertensive heart disease assessment
- 3D speckle tracking assesses regional time-to-minimum-volume, providing a dyssynchrony index; useful in CRT candidate selection and response assessment

3.2 Mitral Valve

The MV is arguably the structure most transformed by 3D echo. Precise anatomical delineation of the MV complex is critical for surgical and catheter-based interventions.

- 3D planimetry of MVA is highly accurate, especially when leaflet tips are irregular (postcommissurotomy, calcified MS)
- Eliminates angulation error inherent in 2D planimetry; identifies commissural fusion, subvalvular disease, and calcification extent
- **3D transesophageal echocardiography (3D TEE):** "Surgeon's view" allows en face visualization—essential for leaflet prolapse localization (Carpentier classification).
- **3D vena contracta area (VCA):** >0.4 cm^2 indicates severe MR (more accurate than 2D PISA which assumes hemispheric shape—invalid for elliptical orifices).
- **MitraClip/TEER guidance:** Identifies leaflet segments, coaptation gap.
- Precise segmental identification (A1-A3, P1-P3, and commissures) for surgical repair planning; differentiates billowing from prolapse and identifies chordal rupture

3.3 Aortic Valve

- **AVA by 3D planimetry:** Accurate in bicuspid aortic valve (BAV) where LVOT is elliptical, not circular (continuity equation underestimates AVA in BAV).
- **TAVR planning:** 3D TEE guides device sizing and deployment; assesses annular dimensions (perimeter-derived diameter > area-derived diameter preferred).
- **AR severity:** 3D VCA >0.6 cm^2 = severe AR.

3.4 Right Ventricular Assessment

- **Most accurate noninvasive RV volumetry:** RV has complex crescent-shaped geometry making 2D volumetry inaccurate.
- **RVEF < 44% by 3DE:** Indicates RV dysfunction.
- **Key in:** Pulmonary hypertension, ARVC, congenital heart disease, and right-sided valve disease.

3.5 Structural Heart Disease Interventions

Real-time 3D TEE is indispensable for procedural guidance:

- **ASD/PFO closure:** Sizing of defect, guiding catheter and device position, confirming device placement, and ruling out residual shunt.
- **LAA occlusion (Watchman/Amulet):** Optimal landing zone sizing, confirming device seal.
- **TAVR:** Annulus measurement, valve positioning, and paravalvular leak assessment.
- **MitraClip/TEER:** Guiding transseptal puncture height, clip grasping, and real-time monitoring.
- **Septal myectomy/alcohol septal ablation in HCM:** Identifying LVOT obstruction mechanism.

3.6 Congenital Heart Disease

- Complex spatial relationships of intracardiac structures visualized without geometric assumptions
- *VSD:* Location, size, number, relation to great vessels; AV canal defects; transposition of great arteries; single ventricle morphology.
- Accurate functional assessment of morphologically RV acting as systemic ventricle.

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3.7 Left Atrial Appendage and Thrombus

- **3D TEE:** Superior characterization of LAA anatomy (chicken wing, cactus, windsock, and cauliflower morphology).
- *Thrombus detection:* 3D provides multiplane confirmation to distinguish thrombus from pectinate muscles/artifact.

3.8 Infective Endocarditis

- *Delineation of vegetations:* Size, mobility, attachment, and involvement of multiple structures.
- **Perivalvular complications:** Abscesses, fistulae, pseudoaneurysms—3D TEE significantly more sensitive than 2D.

4. Artifacts in 3D Echocardiography

4.1 Stitching Artifacts

- **Cause:** Misalignment of subvolumes during multibeat ECG-gated full-volume acquisition due to cardiac arrhythmia, respiratory motion, or probe movement.
- **Recognition:** Characteristic straight-line discontinuity; moving structures appear split or duplicated.
- **Solutions:** Ask patient to hold breath; use single-beat acquisition in arrhythmia; confirm with 2D or live 3D narrow-angle views.

4.2 Shadowing and Dropout

- **Cause:** Dense structure (calcification, prosthetic valve) completely attenuates ultrasound beam.
- **Clinical consequence:** Can mimic perivalvular abscess, ASD, or periprosthetic leak; can hide vegetations or thrombus.
- **Solutions:** TEE for posterior structures; multiple acoustic windows to triangulate shadowed areas.

4.3 Reverberations and Side-lobe Artifacts

- **Reverberations:** Multiple reflections between highly reflective surfaces create "ghost" structures; characteristic: fixed relationship to probe.
- **Side/grating-lobe artifacts:** Parasitic beams reflect off bright structures, mapping them to incorrect locations; appear as echogenic "smearing" near highly reflective objects.
- **Solutions:** Reduce gain; harmonic imaging; change acoustic window.

4.4 Near-field Clutter

- Dense echoes in near field (close to transducer) obscure structures immediately beneath; can hide apical thrombus or mimic pericardial effusion.
- **Solutions:** Lower-frequency transducer; move transducer; use contrast agents.

4.5 Temporal Resolution Artifacts

- **Cause:** Insufficient frame rate (<20 volumes/second) causes temporal blurring; relevant in 3D color Doppler (lowest frame rate mode).
- **Solution:** Increase frame rate by narrowing acquisition sector, reducing depth, using single-beat acquisition.

4.6 Spatial Resolution Artifacts

- 3DE has lower spatial resolution than 2D, particularly in the elevation plane; thin structures may appear thickened or overprominent.
- **Solution:** Use 3D zooms for targeted high-resolution acquisition of specific structures.

5. Advantages of 3D Echo

- No geometric assumptions—accurate volumes regardless of LV/RV shape
- En face views—unique perspective for valves (mitral, aortic, and tricuspid)
- Real-time procedure guidance for structural interventions
- Reproducible volumetry with less observer variability than 2D
- **3D PISA and VCA:** More physiological quantification of regurgitation.
- Integration with speckle tracking for 3D strain and dyssynchrony analysis.

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Q7. Describe the principles, components, and measurement techniques of myocardial strain imaging. What are the clinical applications of GLS?

1. Basic Concepts of Strain

- **Strain (epsilon, e):** Fractional change in myocardial length relative to its original length.
- **Formula:** Strain (%) = [(L – L0)/L0] × 100%, where L0 = baseline length, L = deformed length.
- **Sign convention:** Negative = shortening or compression; Positive = elongation or thickening
- **Strain rate:** Rate of deformation (strain per unit time, s-1); sensitive to rapid transient changes

2. Components of Myocardial Strain (Table 11)

TABLE 11: Components of myocardial strain.

Strain component	*Plane of measurement*	*Echo view*	*Normal direction*	*Clinical utility*
Longitudinal strain (LS)	Long axis (base to apex)	Apical 4C, 2C, 3C	Negative (shortening)	Most validated; reflects subendocardial function; Global longitudinal strain (GLS) most used
Circumferential strain (CS)	Short axis (circumferential)	Parasternal SAX (midcavity)	Negative (shortening)	Reflects midwall function; complements LS
Radial strain (RS)	Radial (wall thickness)	Parasternal SAX	Positive (thickening)	Most variable; least reproducible; limited clinical use
Area strain	Combined LS + CS	SAX + apical views	Negative	Comprehensive but less standardized
3D strain	All directions (3D)	3D acquisition	Negative/positive	Avoids through—plane motion; lower frame rate

3. Measurement Techniques

3.1 Tissue Doppler Imaging-based Strain

- **Principle:** Derives strain from myocardial velocity gradient between two points; strain rate calculated first, strain obtained by integration.
- **Advantages:** High temporal resolution (>100 fps); real-time measurement.
- **Limitations:** Angle-dependent (measures only along beam direction); high signal noise; technically demanding; largely superseded by two-dimensional speckle-tracking echocardiography (2D-STE).

3.2 Two-dimensional Speckle-tracking Echocardiography—Gold Standard

- **Principle:** Natural acoustic markers (speckles) in the myocardium are tracked frame-to-frame throughout the cardiac cycle using pattern-matching algorithms.
- **Angle-independent:** Unlike TDI, not limited by beam orientation.
- **Frame rate requirement:** 50–90 fps for optimal tracking; higher frame rates reduce accuracy.
- **Limitation:** Vendor-specific algorithms; poor inter-vendor agreement; values not interchangeable across platforms.
- **EACVI/ASE standardization:** GLS is the most reproducible parameter across vendors (2015 consensus, 2022 updates).

3.3 Three-dimensional Speckle-tracking Echocardiography

- Tracks speckles in 3D space; eliminates through-plane motion error
- **Limitations:** Lower temporal and spatial resolution; requires good acoustic window; less validated than 2D-STE.
- 3D area strain (combining LS and CS)—emerging but not routinely used.

4. Global Longitudinal Strain—Most Clinically Validated Parameter

- **Measurement:** Average of all segmental longitudinal strain values across three apical views (four-chamber, two-chamber, three-chamber/long-axis)

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- **Normal value:** −20% or more negative (range: −18 to −22% depending on vendor); less negative = worse function.
- **Abnormal cutoff:** GLS > −16% (less negative than −16%)—EACVI/ASE 2015 consensus.
- **Sensitivity:** Detects subclinical LV dysfunction before EF declines; reflects subendocardial fibers (most vulnerable to ischemia and fibrosis).
- **Bull's eye map:** 17-segment color-coded display; basal/mid segments as outer rings, apical as inner rings; allows visual identification of regional strain abnormalities.

5. Clinical Applications of Global Longitudinal Strain

5.1 Detection of Subclinical Left Ventricular Dysfunction

- **EF-GLS mismatch:** Preserved EF with reduced GLS (>−16%) = subclinical dysfunction; seen in early cardiomyopathy, hypertension, and diabetes.
- Particularly useful where visual wall motion assessment is unreliable (e.g., concentric LVH with preserved EF).

5.2 Heart Failure

- **HFrEF:** GLS correlates with severity; stronger predictor of mortality than EF in some cohorts.
- **HFpEF:** GLS commonly reduced despite preserved EF; GLS < −14% in HFpEF = worse prognosis; discriminates HFpEF from healthy controls.
- Reduced GLS supports elevated filling pressures when diastolic parameters are indeterminate.

5.3 Chemotherapy-related Cardiac Dysfunction Monitoring

- **Guideline recommendation:** 2022 ESC Cardio-Oncology Guidelines—GLS is a key tool for early cardiotoxicity detection.
- **Early marker:** Relative reduction in GLS >15% from baseline = early subclinical cardiotoxicity (even if EF preserved).
- **Precedes EF decline:** GLS changes occur weeks to months before EF drops below 50%.
- **Monitoring schedule:** Baseline GLS → after 3–4 cycles → end of therapy → 3 months → 12 months post-treatment.
- **Actionable threshold:** Relative reduction >15% in GLS → consider cardioprotective therapy (ACE inhibitor + β-blocker), intensify monitoring.
- **Agents:** Anthracyclines, trastuzumab, tyrosine kinase inhibitors, immune checkpoint inhibitors.

5.4 Valvular Heart Disease

- **AS:** GLS < −14% in asymptomatic severe AS = LV afterload mismatch and subclinical dysfunction; may prompt earlier AVR consideration.
- **AR:** GLS detects LV dysfunction before EF falls; GLS reduction despite preserved EF = early decompensation.
- **MR:** GLS < −18 to −19% in primary MR = subclinical LV dysfunction; helps timing of surgery in asymptomatic severe MR; 2021 ESC valve guidelines acknowledge GLS role.

5.5 Cardiac Amyloidosis—Apical Sparing Pattern (Pathognomonic)

- **Pattern:** Reduced basal and midwall LS with relatively preserved apical LS on bull's eye map ("cherry on top" appearance).
- **Mechanism:** Amyloid deposits preferentially infiltrate basal and mid segments; apical segments relatively spared (thinner walls, less infiltration).
- **Relative apical sparing ratio (RASR):** = Apical strain/(basal + mid strain); RASR >1.0 suggests amyloidosis.
- **Diagnostic value:** Differentiates cardiac amyloidosis from HCM, hypertensive heart disease; sensitivity ~80%, specificity ~73%.
- **Both AL and ATTR:** Apical sparing seen in both; ATTR typically has more marked LVH and lower voltage on ECG.

5.6 Hypertrophic Cardiomyopathy

- **GLS reduced** even with preserved or supranormal EF

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- **Regional pattern:** Most marked strain reduction in hypertrophied basal segments; apical LS may be compensatory increased (opposite to amyloid).
- **Differentiating HCM from athlete's heart:** GLS typically normal or supranormal in athlete's heart (–22 to –25%); reduced in HCM.
- Useful in gene-positive, phenotype-negative HCM relatives (subclinical dysfunction).

5.7 Ischemic Heart Disease

- *Regional strain abnormalities:* Reduced LS in ischemic territories; improves detection of wall motion abnormalities versus visual assessment.
- **Post-MI:** GLS predicts LV remodeling and adverse outcomes after STEMI.
- **Stress echo:** Worsening LS during stress = ischemia; higher sensitivity than visual wall motion scoring.
- **Viability:** Dobutamine strain reserve (improvement in LS with low-dose dobutamine) predicts myocardial viability and functional recovery.

5.8 Myocarditis

- Global and regional LS reduction correlates with extent of inflammation/fibrosis
- Strain abnormalities may persist despite EF normalization (subclinical residual dysfunction)
- Useful for monitoring recovery and guiding return-to-sport decisions.

5.9 Preoperative Cardiac Risk Assessment

- **GLS < −16%:** Associated with increased perioperative cardiovascular events in noncardiac surgery.
- Additive to clinical risk scores when EF is preserved.

Q8. Describe right ventricular strain and left atrial strain. What are their clinical applications?

1. Right Ventricular Strain

1.1 Measurement

- **RV free wall longitudinal strain (RVFWLS):** Measured from three RV free wall segments (basal, mid, and apical) in apical four-chamber view; most validated RV strain parameter.
- **Four-chamber RV GLS:** Includes septal contribution in addition to free wall; reflects combined septum + free wall; less specific for RV intrinsic dysfunction.
- **Normal RVFWLS:** −20 to –25% (more negative = better RV function).
- **Abnormal cutoff:** RVFWLS > –17% (less negative) = RV dysfunction.
- **Limitations:** Foreshortening of RV free wall in apical view; vendor variability; poor acoustic window at lateral wall.

1.2 Clinical Applications of Right Ventricular Strain

- **Pulmonary arterial hypertension (PAH):** RVFWLS is a strong independent predictor of mortality; RVFWLS > –17% = high risk; serial measurement monitors treatment response; 2022 ESC PAH guidelines recognize RVFWLS as risk stratification tool.
- **RV infarction:** Regional RV free wall LS reduction identifies extent of ischemia.
- **Postcardiac surgery:** RVFWLS detects RV dysfunction after CABG, valve surgery; predicts postoperative outcomes.
- **Congenital heart disease:** RV strain assesses systemic RV function (TGA, ccTGA); monitors subpulmonary RV in repaired TOF.
- **Pulmonary embolism:** Acute RV strain reduction in massive/submassive PE; correlates with RV dilatation and troponin elevation.
- **Tricuspid regurgitation:** RV strain detects early RV dysfunction before dilatation or overt failure.
- **Cardio-oncology:** Subclinical RV dysfunction in cancer patients on chemotherapy.

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2. Left Atrial Strain

2.1 Measurement and Phases (Table 12)

TABLE 12: Phases of left atrial (LA) strain.

Phase	*Cardiac event*	*Physiological correlate*	*Clinical relevance*
Reservoir (LASr)	Ventricular systole; LA fills from pulmonary veins while mitral valve closed	LA compliance, reservoir function; reflects LV filling pressure	Most validated; >23%; reduced in diastolic dysfunction, HFpEF, atrial fibrillation (AF) risk
Conduit (LAScd)	Early diastole; passive LA emptying into left ventricle (LV) after mitral valve opens	Passive LV filling; reflects LV relaxation and compliance	Less validated; reduced in impaired relaxation
Booster/ contractile (LASct)	Late diastole (atrial kick); active LA contraction	Active LA contribution to LV filling (atrial kick)	Absent in AF; reduced in LA dysfunction; reflects intrinsic LA myocardial function

2.2 Normal Values

- **LASr:** >23–38% (varies by vendor; lower limit approximately 23%).
- LAScd and LASct are less standardized; LASr most commonly reported and validated.

2.3 Clinical Applications of Left Atrial Strain

- **Diastolic dysfunction and elevated filling pressures:** LASr <18% suggests elevated LV filling pressures; additive to E/e' ratio in indeterminate diastology.
- **HFpEF:** LASr often reduced even when conventional diastolic parameters are borderline; discriminates HFpEF from healthy controls; LASr correlates with exercise-induced dyspnea.
- **AF:** Reduced LASr predicts AF recurrence after cardioversion and catheter ablation; predicts new-onset AF in hypertension, DM, and HF.
- **MS:** LASr correlates with hemodynamic severity; reduced LASr identifies LA dysfunction and fibrosis.
- **Cryptogenic stroke:** Reduced LASr identifies LA myopathy and cardioembolic risk even without documented AF ("atrial cardiopathy"); may guide anticoagulation decisions.
- **MR:** LA strain reflects LA adaptation to chronic volume overload; helps risk stratify asymptomatic patients.
- **Cardio-oncology:** LA strain detects atrial dysfunction in patients on chemotherapy before overt AF develops.
- **Hypertension and diabetes:** LASr reduced in preclinical stages; subclinical LA remodeling preceding diastolic dysfunction.

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Tanmay R Boob completed his DM in Interventional Cardiology from the UN Mehta Institute of Cardiology and Research Centre, Ahmedabad, Gujarat, India, securing a gold medal from Gujarat University. He completed his MBBS and MD in Internal Medicine from Seth GS Medical College and King Edward Memorial (KEM) Hospital, Mumbai, Maharashtra. He has multiple achievements to his credit, including the Shrenik Shah Medal, the Nattubhai Harijivan Patel Medal, and the MVPM Scholar Award. He currently serves as the Director of Dr Ganesh Boob Memorial Multi-Speciality Hospital and as a Consultant Interventional Cardiologist, Department of Cardiology at Rainbow Institute of Medical Sciences (RIMS) Hospital, Amravati, Maharashtra. His clinical work spans coronary, peripheral, structural, and pediatric intervention; device therapy; and advanced cardiac critical care. He has contributed to peer-reviewed publications in cardiology. Teaching has remained close to his interests throughout his training, and this book grows out of that commitment—an effort to make "Essentials of Cardiology" clear, accessible, and examination-ready for trainees.



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