

Week 6: Cardiomyopathies, Pulmonary Hypertension & Arrhythmias

CVSG2280 Cardiovascular Registry Review by Dr. Alice Matthews

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Registry weight this week. Cardiomyopathies, pulmonary hypertension, and arrhythmias sit squarely in the **Pathology** content area. On the **ARDMS RDCS (Adult Echo, V24.2)** the domain split is Anatomy/Physiology 15%, **Pathology 40%**, Clinical Care/Safety 11%, Measurement/Views 25%, Instrumentation 9%; this week feeds the single largest slice, the 40% Pathology block, with spillover into the 25% Measurement/Views block (strain, RV functional indices, PASP). On the **CCI RCS** the domain split is A 9%, B 24%, C 25%, **D 29%**, E 13%; W6 maps mainly to **Domain D (Anatomy, Physiology, Hemodynamics, and Pathology)**, **about 29%**, the heaviest domain on that exam. Expect this material across roughly a third of either registry.

Note to students: Cutoffs in this guide follow the [ASE 2025 LV Diastolic Function and HFpEF update](#), the [ASE 2025 Right Heart and Pulmonary Hypertension](#) and [ASE 2015 Chamber Quantification](#) recommendations, with strain conventions from the ASE/EACVI deformation imaging consensus. **What last week's EOM gives you:** Week 5 procedure prep and clinical safety taught you to set up the RV-focused, subcostal, and PLAX/SAX windows cleanly; this week you point those same windows at diseased muscle. Always defer to the protocol your clinical site follows.

At a Glance: this week in one box

Must-know numbers

- DCM: LVEF $\leq 40\%$ (often $< 35\%$), LVIDd above the chamber-quant normal (men 4.2 to 5.8 cm, women 3.8 to 5.2 cm), EPSS > 6 mm.
- HCM: wall thickness ≥ 15 mm; resting LVOT peak velocity normal < 3.1 m/s, provokable/latent 3.1 to 3.6 m/s, obstructive ≥ 3.6 m/s (≥ 50 mmHg gradient).
- RCM/amyloid: restrictive inflow E/A > 2.0 , DT < 160 ms, average E/e' > 14 ; biatrial enlargement.
- PHTN: mean PAP > 20 mmHg (2025 definition); PASP screening cutoff 35 to 40 mmHg; TR Vmax > 2.8 m/s.
- RV: TAPSE ≥ 1.7 cm, S' ≥ 10 cm/s, FAC $> 35\%$; PAT < 105 ms suggests PHTN.
- TAPSE/PASP coupling: > 0.5 normal, 0.4 to 0.5 borderline, < 0.4 uncoupling, < 0.3 severe uncoupling.

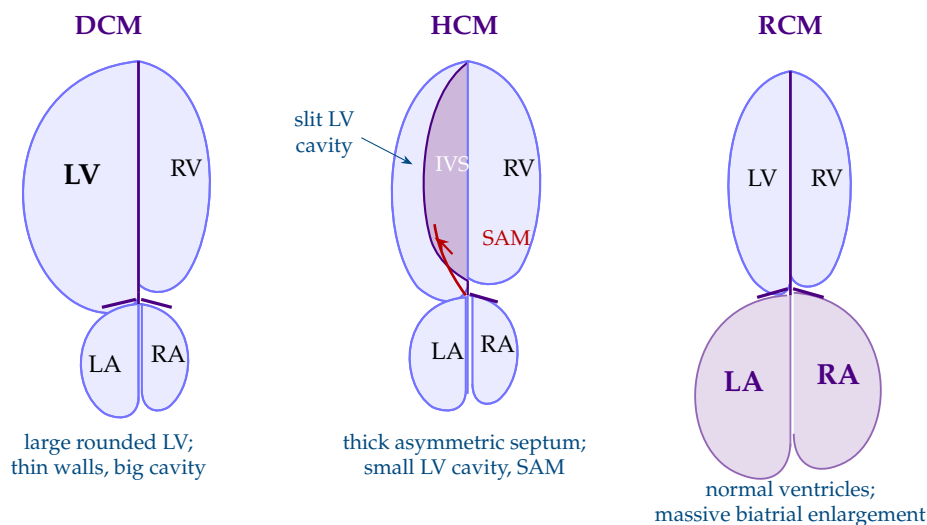
Formulas you will reuse

- $PASP = 4(V_{TR})^2 + RAP$ (TR jet plus IVC-estimated RAP)
- $\Delta P_{LVOT} = 4(V_{LVOT})^2$ (dynamic obstruction in HCM)
- TAPSE/PASP coupling ratio (RV-PA coupling)
- GLS magnitude, normal -18 to -22% (amyloid apical sparing)

Top 3 EOM traps

- Global hypokinesis (DCM) vs. territorial wall motion (ischemia).
- Concentric LVH on 2D that is actually amyloid: the strain bull's-eye, not wall thickness, breaks the tie.
- Reporting RVSP as PASP when pulmonic stenosis coexists (subtract the PV gradient).

Dilated Cardiomyopathy (DCM)



Apical four-chamber silhouettes of the three classic cardiomyopathies (apex up). DCM: dilated, thin-walled left ventricle with a large cavity. HCM: small LV cavity with a thickened asymmetric interventricular septum (IVS) and systolic anterior motion (SAM) of the anterior mitral leaflet toward the septum and LVOT. RCM: normal-sized ventricles with marked biatrial (LA and RA) enlargement.

DCM is a primary disorder of impaired LV contractility: the ventricle dilates, contractility falls, and end-diastolic pressure climbs. Causes split into **ischemic** (prior infarct, so the wall motion follows a coronary territory) and **non-ischemic** (idiopathic, viral, toxic such as alcohol or anthracycline, peripartum, or infiltrative). The classic echo is a big, round, globally weak ventricle.

★ **Global hypokinesis with no territorial pattern** is the signature. All walls move in poorly and uniformly, unlike ischemic disease where one coronary territory is hit hardest.

- **2D/3D:** LV cavity dilation (LVIDd above the chamber-quant normals; men 4.2 to 5.8 cm, women 3.8 to 5.2 cm), reduced EF (< 40%, often < 35%), LA dilation from chronically elevated LV end-diastolic pressure.
- **M-mode:** ★ increased EPSS (mitral E-point to septal separation > 6 mm signals poor LV function), a “B-bump” on the mitral closure line, and reduced aortic root excursion.
- **Doppler:** reduced peak aortic ejection velocity, reduced dP/dt (normal > 1200 mmHg/s; advanced DCM < 600 mmHg/s), secondary (functional) MR from a stretched annulus, and a restrictive inflow pattern ($E/A > 2.0$) as the disease advances.
- **Screen for apical LV thrombus** when EF < 35% with apical akinesis; this is a systemic stroke source.
- **Associated:** atrial fibrillation, secondary pulmonary hypertension, RV dysfunction.

Common Trap

A 58-year-old presents with new heart failure. The echo shows a dilated LV with EF 30%, but the inferior and inferolateral walls are clearly more akinetic than the rest. A classmate calls it dilated cardiomyopathy because the chamber is big and weak. **What is the error?**

DCM is defined by **global, non-territorial** hypokinesis. A regional gradient that follows a coronary distribution (here the right coronary or circumflex territory) points to **ischemic** disease, not idiopathic DCM. The dilation can look identical; the wall-motion pattern is what separates them. Reserve “dilated cardiomyopathy” for diffuse, uniform dysfunction.

Hypertrophic Cardiomyopathy (HCM)

HCM is a primary muscle disorder with asymmetric LV hypertrophy, normal-to-supranormal systolic function, and impaired diastolic function. It is autosomal dominant (about half are familial) and is the leading cause of sudden cardiac death in young athletes. Wall thickness ≥ 15 mm in any segment is the diagnostic threshold.

★ **Systolic anterior motion (SAM)** of the anterior mitral leaflet drags the leaflet toward the septum in systole, narrowing the LVOT and creating both dynamic obstruction and posteriorly directed MR.

- **Classification:** nonobstructive (about one third) has resting gradient < 50 mmHg and no SAM; obstructive (about two thirds) has resting gradient ≥ 50 mmHg with SAM; provokable/latent has a low resting gradient that climbs with Valsalva, standing, or exercise.
- **2D:** asymmetric septal hypertrophy (anterior septum thicker than posterior wall), a “spade”-shaped short-axis cavity, normal-to-small LV cavity, possible LA enlargement. The **apical variant** (“ace of spades”) confines hypertrophy to the apex with apical thrombus risk.
- **M-mode:** midsystolic notching of the aortic valve (obstruction reduces aortic opening mid-ejection) and premature systolic closure when the SAM leaflet touches the septum.
- **Doppler:** ★ CW from the apical 5-chamber shows a **late-peaking, dagger-shaped** LVOT envelope (peaks mid-to-late systole, unlike aortic stenosis which peaks early). The gradient is **dynamic**: it rises with reduced preload (Valsalva, standing) or increased contractility.
- **Tissue Doppler:** S' is normal or elevated (unlike the reduced S' of DCM or restrictive disease).

★ **Memory hook:** HCM gradient goes **UP** when the heart gets emptier or stronger. Anything that shrinks the cavity (Valsalva, standing from a squat, amyl nitrite historically, dobutamine, hypovolemia) worsens obstruction; anything that fills the cavity (squatting, leg raise, handgrip, volume) relieves it. Run the list preload-down then contractility-up.

Common Trap

A CW envelope through the LVOT in a patient with thick septal walls peaks late in systole and is dagger-shaped. A test-taker labels it severe aortic stenosis because the peak velocity is 4.2 m/s. **What is the error?**

Timing separates the two. Fixed aortic stenosis peaks **early-to-mid** systole with a rounded, symmetric envelope. Dynamic LVOT obstruction in HCM peaks **late** (“dagger” or “scimitar” shape) because the obstruction tightens as the cavity empties. A late-peaking signal with SAM and asymmetric hypertrophy is HCM obstruction, and the gradient is dynamic, not fixed.

Figure pending: HCM with systolic anterior motion of the mitral valve

Restrictive Cardiomyopathy (RCM)

RCM is a stiff, non-dilated ventricle: normal-to-small cavity, severely impaired diastolic filling, near-normal systolic function until late. Causes include amyloidosis (AL or ATTR), hemochromatosis, sarcoidosis, radiation, and idiopathic fibrosis. The hallmark is ★ **biatrial enlargement with a normal-sized ventricle**, because blood backs up into both atria against the stiff ventricles.

- **2D:** normal-to-small LV cavity, normal-to-increased wall thickness (especially amyloid), biatrial dilation, RV wall thickening (normal ≤ 5 mm; restrictive 8 to 10 mm), EF often $> 40\%$ until late.
- **Restrictive inflow:** ★ $E/A > 2.0$, deceleration time < 160 ms (blood crashes into the LA and stops fast), average $E/e' > 14$ (elevated filling pressure), prominent E with small A, and systolic blunting or reversal of pulmonary venous flow.
- **Associated:** moderate pulmonary hypertension, elevated RA pressure (dilated IVC with minimal collapse).

Restrictive vs. constrictive is a recurring exam contrast. Restrictive disease is a **myocardial** problem (stiff muscle, normal pericardium, biatrial dilation, reduced RV function, less septal bounce). Constrictive disease is a **pericardial** problem (thickened pericardium > 3 mm, normal myocardium until late, prominent septal bounce, respiratory variation in inflow, preserved RV function).

Common Trap

A patient has elevated JVP, dyspnea, biatrial enlargement, and a restrictive mitral inflow. A reviewer calls it constrictive pericarditis and recommends pericardiectomy. **What is the error?**

Both restrictive cardiomyopathy and constriction give elevated filling pressures and a small ventricle, but the **pericardium** is the discriminator. Restrictive disease shows a normal pericardium with stiff myocardium, biatrial dilation, and reduced tissue-Doppler e' ; constriction shows a thickened pericardium, a prominent septal bounce, marked respiratory inflow variation, and (paradoxically) **preserved or high** medial e' (annulus paradoxus). Surgery helps constriction, not restriction, so the distinction changes management.

Other Cardiomyopathies

Arrhythmogenic RV cardiomyopathy (ARVC): genetic fibro-fatty replacement of RV myocardium. Echo shows RV dilation (basal diameter > 4.1 cm), RV free-wall akinesis/dyskinesis, and prominent trabeculations; LV involvement is late. Ventricular arrhythmias and SCD in young athletes; cardiac MRI is the gold standard.

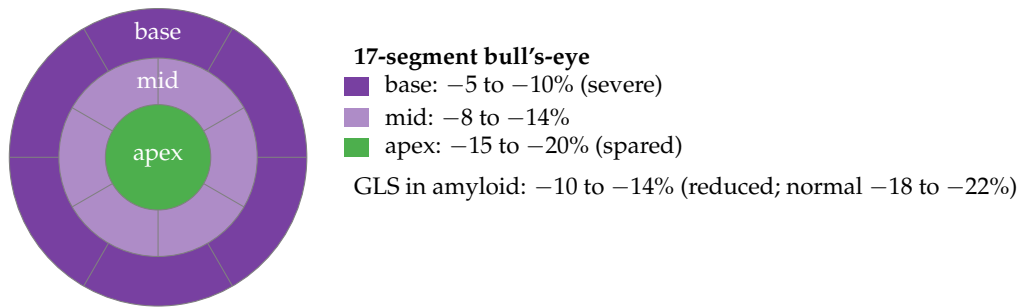
LV non-compaction (LVNC): excessive trabeculae with deep recesses; a noncompacted-to-compacted ratio > 2.3 at end-systole supports the diagnosis; risks are heart failure, arrhythmia, and thrombus.

Takotsubo (stress) cardiomyopathy: transient ★ **apical ballooning** (akinetic apex, hyperdynamic base) triggered by emotional or physical stress, with **normal coronaries**. Usually recovers in weeks, but the acute phase carries shock and arrhythmia risk.

Infiltrative and Inflammatory Cardiomyopathies

Amyloidosis (AL or ATTR) deposits misfolded protein in the myocardium. AL is light-chain (acquired, poor prognosis); ATTR is transthyretin, either hereditary (ATTRm) or wild-type (ATTRwt, an emerging cause of HFpEF in elderly men). Echo: LV wall thickening (often 12 to 15+ mm) that **mimics** hypertrophy, a granular “sparkling” myocardium, biatrial enlargement, biventricular involvement, and a restrictive inflow.

★ **The single most specific echo finding is the "cherry on top" (apical-sparing) strain pattern:** basal and mid segments are severely reduced while the apex stays relatively preserved. On the bull's-eye this looks like a red base with a green apical cap. It is rare in any other disease, so it breaks the tie between amyloid and look-alike LVH.



Apical-sparing ("cherry on top") strain map. Dark = severely reduced longitudinal strain at the base and mid; green = relatively preserved apex. Highly specific for cardiac amyloid.

Sarcoidosis: granulomatous infiltration favoring the basal inferoseptum; regional wall-motion abnormality and thin-walled aneurysm (VT and SCD risk); cardiac MRI confirms. **Hemochromatosis:** iron overload giving a dilated, dysfunctional LV with a restrictive pattern. **Fabry disease:** X-linked lysosomal storage with severe concentric LVH and a short anterior mitral leaflet. **Myocarditis:** acute (often viral) inflammation with reduced EF and a frequent pericardial effusion; may evolve into DCM or recover.

Figure pending: Cardiac amyloid: concentric LVH, granular sparkling

Hypertensive Heart Disease

Chronic systemic hypertension produces **concentric LVH** (thick walls, normal-to-small cavity), **diastolic dysfunction** that progresses with worsening control, LA enlargement, aortic sclerosis, mitral annular calcification, and atrial fibrillation. The early Doppler signature is ★ impaired relaxation: $E/A < 0.8$ with a prolonged deceleration time, which can later pseudonormalize and finally become restrictive.

Pulmonary Hypertension (PHTN)

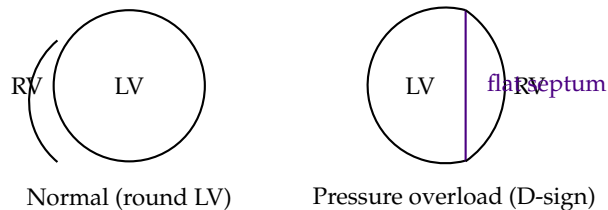
PHTN is grouped into five WHO categories: (1) pulmonary arterial hypertension (idiopathic, connective tissue, congenital), (2) left heart disease (HFpEF, mitral or aortic disease), (3) lung disease (COPD, interstitial fibrosis), (4) chronic thromboembolic (CTEPH), and (5) miscellaneous or multifactorial.

★ **The 2025 definition lowered the threshold to mean PAP > 20 mmHg** (previously > 25 mmHg). By echo, the practical screening cutoff is PASP > 35 to 40 mmHg, and TR Vmax > 2.8 m/s is the entry flag.

Memory hook: PHTN echo workup, right to left across the heart: TR jet to estimate PASP, then RV size and function (TAPSE, S', FAC), then septal position (D-sign), then RA size, then the PA itself (diameter, PAT, midsystolic notch), then IVC for RAP. Walk the blood backward from the lungs.

- **PASP estimation:** from the TR jet using $4(V_{TR})^2 + RAP$, with RAP read off IVC size and collapse.
- **TR jet and PA flow clues:** short acceleration time, **midsystolic notching** of the PV envelope (a rapid pressure rise slams the valve mid-systole), and a dense triangular CW envelope.
- **RV assessment:** dilation (basal diameter > 4.1 cm), **septal flattening** (the D-sign), reduced TAPSE (< 1.7 cm), reduced S' (< 10 cm/s), reduced FAC (< 35%), and RV free-wall strain less negative than -17%.
- **McConnell's sign:** ★ RV free-wall hypokinesis with a **normally moving apex**; classic for acute pulmonary embolism (acute RV pressure overload).
- **RA and PA:** RA area > 18 cm² is enlarged; PA diameter > 25 mm; ★ PAT < 105 ms suggests PHTN.

Memory hook: Septal motion tells you the overload type: the septum flattens in **systole** for **pressure** overload (PHTN, PS) and in **diastole** for **volume** overload (ASD, severe TR). "Pressure pushes in systole, volume fills in diastole."



Parasternal short axis. A round LV is normal; a flattened interventricular septum makes the LV look like a "D" when RV pressure (or volume) overload pushes the septum leftward.

NEW (2025 ASE): RV-PA coupling is assessed with the ★ **TAPSE/PASP ratio**. As PASP rises and TAPSE falls, the ratio drops: > 0.5 mm/mmHg is normal (the RV keeps up), < 0.4 is uncoupling, and < 0.3 is severe uncoupling tied to higher mortality.

Cor Pulmonale (Pulmonary Heart Disease)

Acute cor pulmonale (PE, RV infarct) gives abrupt RV dilation with a normal LV, an elevated TR gradient, and McConnell's sign. **Chronic** cor pulmonale (COPD, interstitial fibrosis, chronic PE) gives RV **hypertrophy** (thick RV free wall, trabeculation) with gradual dilation, a progressively rising TR gradient, and a compressed LV.

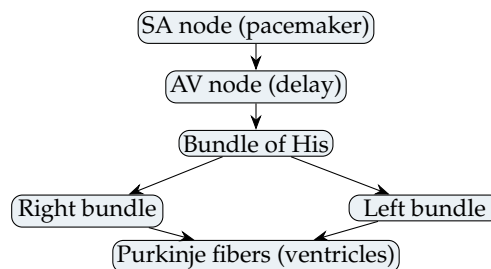
Arrhythmia Assessment on Echo

Atrial fibrillation (AF): ★ LA enlargement (diameter > 4.0 cm or LAVi > 34 mL/m²), reduced LA reservoir strain, loss of the atrial kick (so stroke volume falls), and beat-to-beat variation that forces you to **average multiple cycles**. On spectral inflow the discrete E/A is replaced by chaotic fibrillatory waves. AF plus left atrial appendage stasis raises stroke risk, so **TEE rules out LAA thrombus before cardioversion** (LAA emptying velocity < 20 cm/s flags high risk).

Premature ventricular contractions (PVCs)/ectopy: early beats that distort beat-to-beat measurements; average over several cycles and watch for ectopy-driven wall-motion changes.

Pacemakers, ICD leads, and CRT: a pacing lead is a bright linear echo in the RA or RV (it may be seen crossing the tricuspid valve); dual-chamber systems have RA and RV leads; **CRT** adds an LV lead through the coronary sinus to the lateral wall to correct dyssynchrony. An ICD has a thick echogenic RV coil. Watch for perforation, lead-associated thrombus, or fracture.

Memory hook: ★ **Loss of atrial kick costs filling.** Atrial systole contributes roughly the last 20% of LV filling; in AF that contribution disappears, so stroke volume and cardiac output drop even when EF looks unchanged. "No kick, less fill."



Conduction pathway. AF disorganizes the atrial step above the AV node; pacing leads substitute downstream electrical timing.

Essential Formulas

Pulmonary artery systolic pressure (TR jet):
$$PASP = 4 (V_{TR})^2 + RAP$$

Plain English: square the peak TR velocity, multiply by four for the RV-to-RA gradient, then add the right atrial pressure read from the IVC. Why it matters: this is the workhorse screen for pulmonary hypertension (PASP $>$

35 to 40 mmHg is abnormal). Caveat: if pulmonic stenosis coexists this gives RVSP, so subtract the PV gradient $4(V_{PV})^2$ to recover true PASP.

RAP from the IVC (2025 ASE):

IVC diameter	Collapse with sniff	RAP estimate
< 2.1 cm	> 50%	3 mmHg
< 2.1 cm	≤ 50%	8 mmHg
> 2.1 cm	> 50%	8 mmHg
> 2.1 cm	≤ 50%	15 mmHg

LVOT gradient (HCM): $\Delta P_{LVOT} = 4(V_{LVOT})^2$

Plain English: simplified Bernoulli applied to the dynamic LVOT jet from the apical 5-chamber. Why it matters: a resting peak velocity ≥ 3.6 m/s (≥ 50 mmHg) defines obstructive HCM and the gradient rises with provocation; the envelope peaks late and is dagger-shaped, which distinguishes it from fixed aortic stenosis.

RV-PA coupling: $\text{Coupling} = \frac{TAPSE \text{ (mm)}}{PASP \text{ (mmHg)}}$

Plain English: how far the tricuspid annulus moves per unit of pulmonary pressure it works against. Why it matters: > 0.5 mm/mmHg is a well-adapted RV, < 0.3 mm/mmHg is severe uncoupling and predicts poor outcomes; it captures RV failure earlier than TAPSE alone.

Global longitudinal strain (GLS): $GLS (\%) = \frac{L_{end-systole} - L_{end-diastole}}{L_{end-diastole}} \times 100$

Plain English: the percent shortening of the myocardium along its long axis, tracked by speckle imaging and reported as a negative number (more negative is better). Why it matters: normal is -18 to -22% ; GLS falls before EF in early disease, and the **apical-sparing** pattern is highly specific for amyloid.

Fractional area change (RV systolic function): $FAC (\%) = \frac{A_{diastole} - A_{systole}}{A_{diastole}} \times 100$

Plain English: the percent the RV cavity area shrinks from diastole to systole in the RV-focused apical 4-chamber. Why it matters: $FAC > 35\%$ is normal; it is a quick 2D read of RV systolic function when strain is unavailable.

W6 Doppler View Cheat Sheet

Target	What you measure	Best view
RV size and function	TAPSE, TDI S', FAC	RV-focused apical 4-chamber; TDI at TV annulus
RV free wall	Strain, wall thickness	RV-focused apical; subcostal
RA size and pressure	RA area, IVC diameter and collapse	Apical 4-chamber; subcostal
PA diameter and PAT	PA diameter, acceleration time	Parasternal short axis at aortic-root level
TR velocity (PASP)	CW peak jet velocity	Apical 4-chamber; parasternal RV inflow
Septal flattening (D-sign)	Systolic vs. diastolic flattening	Parasternal short axis; apical 4-chamber
LV strain / GLS	Regional strain, bull's-eye	Apical 4-, 2-, and 3-chamber (speckle tracking)
Amyloid apical sparing	Apical preserved, base reduced	Apical 4-, 2-, 3-chamber strain
AF burden / LA function	Fibrillatory waves, LA strain	Apical 4-chamber (Doppler and 2D)

Target	What you measure	Best view
LVOT gradient (HCM)	Late-peaking dagger envelope	Apical 5-chamber CW
Pacemaker / ICD lead	Linear echogenicity in RA/RV	Apical 4-chamber; subcostal

Severity quick reference.

Parameter	Normal	Abnormal threshold
LVEF (preserved overall)	$\geq 50\%$	$\leq 40\%$ reduced; 41 to 49% mildly reduced
HCM wall thickness	n/a	≥ 15 mm
HCM resting LVOT velocity	< 3.1 m/s	3.1 to 3.6 m/s provokable/latent; ≥ 3.6 m/s obstructive
Restrictive inflow E/A	0.8 to 2.0	> 2.0
Deceleration time	160 to 200 ms	< 160 ms (restrictive)
Average E/e'	< 8	> 14
TR Vmax	≤ 2.8 m/s	> 2.8 m/s
Mean PAP (2025)	≤ 20 mmHg	> 20 mmHg
PASP (echo screen)	< 35 mmHg	35 to 40 mmHg and above
TAPSE	≥ 1.7 cm	< 1.7 cm
RV S'	≥ 10 cm/s	< 10 cm/s
RV FAC	$> 35\%$	$< 35\%$
PA acceleration time	≥ 105 ms	< 105 ms
TAPSE/PASP coupling	> 0.5 mm/mmHg	0.4 to 0.5 borderline; < 0.4 uncoupling; < 0.3 severe
GLS (magnitude)	-18 to -22%	less negative than -18%

Glossary

- **DCM** — dilated cardiomyopathy; big, globally weak LV
- **HCM** — hypertrophic cardiomyopathy; asymmetric LVH, dynamic LVOT obstruction
- **RCM** — restrictive cardiomyopathy; stiff non-dilated LV, biatrial dilation
- **SAM** — systolic anterior motion of the mitral leaflet (obstructive HCM)
- **EPSS** — E-point to septal separation; > 6 mm means poor LV function
- **LVOT** — left ventricular outflow tract
- **dP/dt** — rate of LV pressure rise; normal > 1200 mmHg/s
- **Apical ballooning** — akinetic apex with hyperdynamic base (Takotsubo)
- **McConnell's sign** — RV free-wall hypokinesis with a spared apex (acute PE)
- **D-sign** — flattened septum giving a D-shaped LV in short axis
- **TAPSE** — tricuspid annular plane systolic excursion; normal ≥ 1.7 cm
- **TDI S'** — systolic tissue-Doppler velocity of the TV annulus; ≥ 10 cm/s
- **FAC** — RV fractional area change; normal $> 35\%$
- **PAT** — pulmonary artery acceleration time; < 105 ms suggests PHTN
- **RV-PA coupling** — TAPSE/PASP ratio; < 0.3 severely uncoupled
- **Infiltrative CM** — myocardium loaded with abnormal substance (amyloid, iron, granuloma)
- **Amyloidosis** — misfolded protein deposition; apical-sparing strain
- **ATTR** — transthyretin amyloid; ATTRm hereditary or ATTRwt wild-type
- **Myocarditis** — acute myocardial inflammation; may evolve to DCM
- **GLS** — global longitudinal strain; normal -18 to -22%
- **Speckle tracking** — frame-to-frame tracking of acoustic markers
- **Bull's-eye plot** — 17-segment color map of regional strain
- **AF** — atrial fibrillation; LA enlargement, lost atrial kick
- **LAA** — left atrial appendage; thrombus source in AF
- **CRT** — biventricular pacing for dyssynchrony
- **RAP** — right atrial pressure (from IVC size and collapse)
- **PASP** — pulmonary artery systolic pressure

Registry Connection

This week feeds the **ARDMS RDCS Pathology** subdomain (cardiomyopathies, pulmonary hypertension, RV pathology) with measurement crossover into Measurement/Views (strain, TAPSE, FAC, PASP), and **CCI RCS Domain D**. Expect items that ask you to: read a strain bull's-eye and name the disease (apical sparing = amyloid); separate global (DCM) from territorial (ischemic) wall motion; tell a late-peaking dynamic LVOT envelope (HCM) from early-peaking aortic stenosis; estimate PASP from a TR-jet image plus an IVC clip; and grade diastolic dysfunction and PHTN severity from a value table. **Carry forward into Week 7:** the diastolic-function flowchart (E/A, DT, E/e', LAVi, TR Vmax) and the GLS habit move straight into ischemic disease and formal diastolic grading.

Study smart. Scan with confidence.

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