

Week 7: Ischemic Heart Disease, Heart Failure & Diastolic Function

CVSG2280 Cardiovascular Registry Review by Dr. Alice Matthews

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Registry weight this week. Ischemic disease, heart failure, and diastolic function sit squarely in the **Pathology** domain, which is the single largest slice of both exams. On the **ARDMS RDCS** (Adult Echo V24.2): Pathology is 40%, Measurement/Views 25%, Anatomy/Physiology 15%, Clinical Care/Safety 11%, Instrumentation 9%. On the **CCI RCS**: Domain D (integration/pathology) 29%, Domain C 25%, Domain B 24%, Domain E 13%, Domain A 9%. This week feeds the Pathology + Measurement core, so expect it to account for a large share (roughly a third to a half) of your scored items.

Note to students: This week follows the [ASE 2025 LV Diastolic Function and HFpEF update](#), the [ASE 2015 Chamber Quantification](#) recommendations, the [ASE strain echocardiography](#) consensus, and the [ASE 2025 Right Heart and Pulmonary Hypertension](#) guidance. **What last week (EOM 6) gives you:** the cardiomyopathy patterns (HCM, DCM, restrictive, amyloid) carry straight into this week's strain and diastolic grading; amyloid's apical sparing is the bridge. All numbers here are quoted from the course canonical values file so they match every other guide. Always defer to the protocol your clinical site follows.

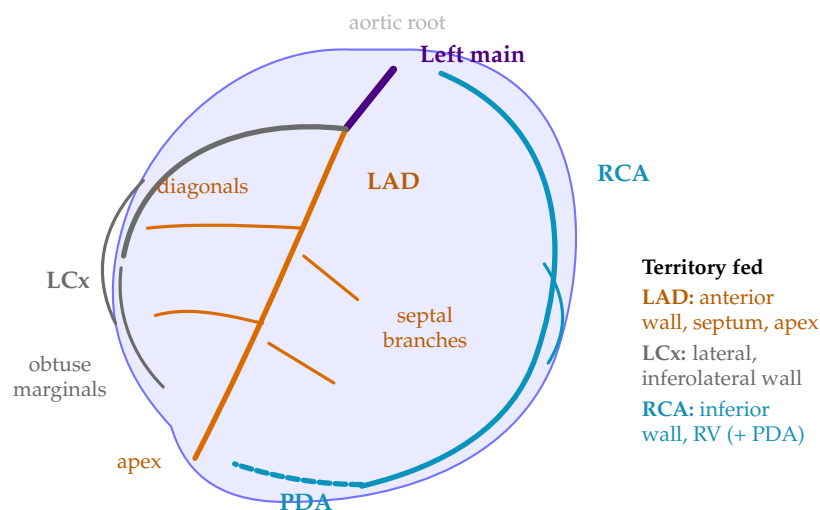
At a Glance: this week in one box

Must-know numbers: EF preserved $\geq 50\%$; FS normal 25 to 45%; dP/dt normal > 1200 mmHg/s (abnormal < 800); E/A normal 0.8 to 2.0; DT normal 160 to 200 ms; septal $e' \geq 7$ cm/s, lateral $e' \geq 10$ cm/s; average $E/e' < 8$ normal, > 14 elevated; LAVi < 28 normal, > 34 abnormal; TR Vmax ≤ 2.8 m/s; GLS normal more negative than -17% (men -17 to -24% , women -18 to -26%).

Formulas you will reuse: $EF = \frac{EDV - ESV}{EDV} \times 100$; $FS = \frac{LVIDd - LVIDs}{LVIDd} \times 100$; $dP/dt = \frac{32}{\Delta t}$; $SV = CSA_{LVOT} \times VTI_{LVOT}$; $CO = SV \times HR$; E/e' ; GLS.

Top 3 EOM traps: (1) calling a normal-looking E/A "normal" when it is really pseudonormal Grade II (use e' and LAVi to unmask it); (2) reading dP/dt against the old > 1000 cutoff instead of > 1200 ; (3) forgetting that amyloid's low GLS *sparcs the apex* (cherry-on-top), so apex strain looks deceptively preserved.

Coronary Anatomy and Wall-Segment Assignment



Schematic coronary tree (anterior view). The left main divides into the LAD (anterior interventricular groove, giving diagonal and septal branches to the anterior wall, septum, and apex) and the LCx (left AV groove, giving obtuse marginals to the lateral wall). The RCA runs the right AV groove and, in a right-dominant heart, gives the PDA (dashed, on the posterior surface) to the inferior wall. Colors match the bullseye: LAD orange, RCA cyan, LCx gray.

Ischemia is a plumbing problem: each coronary artery feeds a defined region of myocardium, so a wall-motion abnormality in a known segment points you straight at the culprit vessel. The left main divides into the **LAD** (left anterior descending), which wraps the anterior wall, septum, and apex, and the **LCx** (left circumflex), which feeds the lateral wall. The **RCA** (right coronary) supplies the inferior wall, the RV, and (in a right-dominant heart) the posterior descending artery.

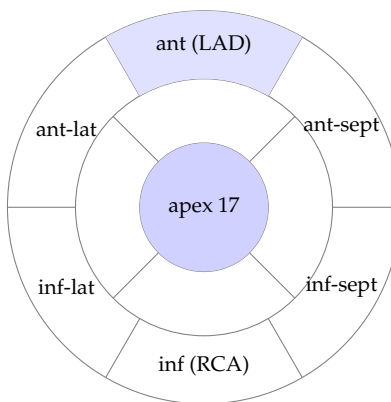
★ The **ASE 17-segment model** divides the LV into 6 basal, 6 mid, 4 apical segments, and 1 apical cap. Memorize which segments belong to which artery; this is the most reliable RWMA-to-vessel question on the registry.

Territory	Artery	17-segment numbers
Anterior wall, septum, apex	LAD	1, 2, 7, 8, 13, 14, 17
Lateral and inferolateral wall	LCx	5, 6, 11, 12, 16
Inferior wall, RV	RCA (or LCx)	4, 10, 15

Dominance note: the inferior and inferolateral basal segments shift between RCA and LCx depending on which vessel gives off the PDA (right-dominant in about 85% of hearts).

Memory hook: “**LAD owns the front and the tip, LCx hugs the side, RCA runs the bottom.**” The apex (segment 17) is almost always LAD, which is why anteroapical aneurysms follow LAD infarcts.

Dominance. The artery that gives off the posterior descending artery (PDA) defines dominance: right-dominant (about 85%), left-dominant (about 8%), or codominant. A dominant RCA means an inferior infarct can also threaten the inferobasal septum and posteromedial papillary muscle, a setup for acute mitral regurgitation.



17-segment bullseye (basal / mid / apical rings + apical cap). Blue = LAD territory.

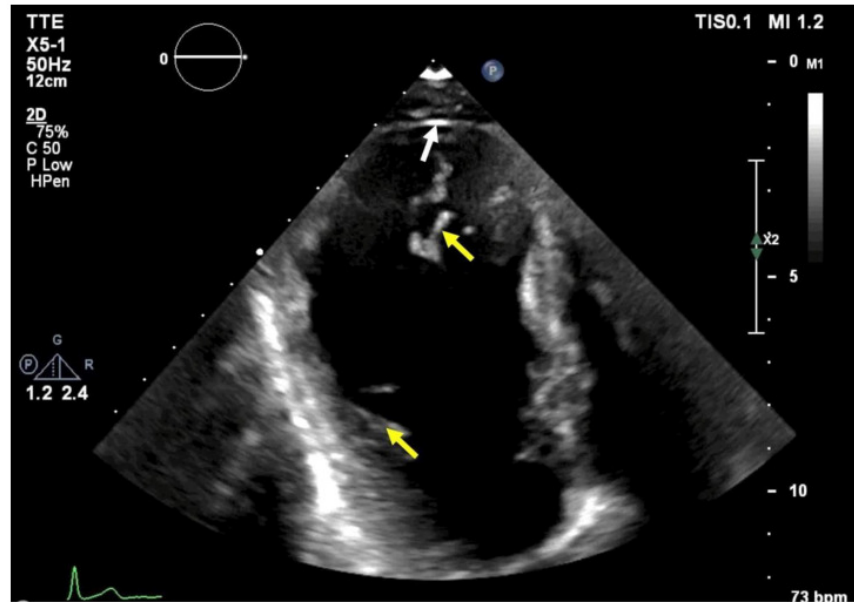
Regional Wall Motion Abnormalities (RWMA)

A wall-motion abnormality means a segment fails to thicken and move inward in systole. You score each of the 17 segments on a 5-point scale, then describe the pattern by territory. Acute ischemia produces a **regional** defect that matches one artery; a global problem (toxic, infiltrative, end-stage) hits everything at once.

★ **Wall-motion grading scale (ASE standard):**

Score	Type	Definition
1	Normal	Systolic thickening and inward motion
2	Hypokinetic	Reduced thickening and inward motion
3	Akinetic	Absent or negligible thickening; no inward motion
4	Dyskinetic	Outward (paradoxical) systolic motion
5	Aneurysmal	Bulges outward with a thinned wall

Memory hook: (motion ladder) “Normal, weak, still, backward, bulging” maps 1 to 5. Score climbs as the muscle gets sicker; only 4 and 5 move the *wrong* way.



Transthoracic 2D echocardiogram, apical view, showing an isolated apical left ventricular aneurysm. The white arrow points to the thinned, outpouching aneurysmal apical wall; the yellow arrows mark associated apical trabeculations. The finding is a wall abnormality (apical aneurysm), not an intracardiac thrombus or mass. Image by Channan EM, Pintea Bentea G, Berdaoui B, Damry N, Morissens M, Castro Rodriguez J (F1000Research 2023;12:1486) via PMC (PMC11112464), CC BY 4.0.

Viability versus scar. On dobutamine stress echo, a segment that contracts *better* at low dose but worsens at high dose shows the classic **biphasic** response: viable, ischemic, and worth revascularizing. A segment that never improves at any dose is scar. Sustained improvement that holds at high dose suggests viable, non-ischemic stunned myocardium.

Common Trap

A patient with chest pain has a new akinetic inferior wall (segments 4, 10, 15) and a normal anterior wall. The reading sonographer attributes it to the LAD because "the LAD is the most important artery." What is the error?

The inferior wall is **RCA** (or a dominant LCx) territory, not LAD. The LAD feeds anterior wall, septum, and apex. Match the abnormal *segments* to the artery, not the artery's reputation. An isolated inferior RWMA points to the RCA.

LV Systolic Function Assessment

Systolic function answers one question: how well does the LV empty? Ejection fraction is the headline number, but registries also test fractional shortening, dP/dt , cardiac output, and LV mass. Quote the canonical bands exactly.

★ **Ejection fraction (Simpson biplane)** is the volume-based gold standard: trace the endocardium in the apical 4-chamber and 2-chamber views at end-diastole and end-systole. Preserved EF is $\geq 50\%$ for both sexes (ASE 2015).

Parameter	Normal	Mildly reduced	Moderately reduced	Severely reduced
EF (%)	≥ 50	41 to 49	30 to 40	< 30
FS (%)	25 to 45	18 to 24	10 to 17	< 10
CO (L/min)	4 to 8	n/a	n/a	< 4
CI (L/min/m ²)	2.5 to 4.0	n/a	n/a	< 2.0
dP/dt (mmHg/s)	> 1200	800 to 1200	n/a	< 800

dP/dt is the rate the LV builds pressure during isovolumetric contraction, read off the ascending limb of the **MR continuous-wave jet**. Time how long it takes the jet to rise from 1 m/s to 3 m/s; the pressure change between those two points is fixed at 32 mmHg, so $dP/dt = 32/\Delta t$. A steeper rise (shorter Δt) means a stronger ventricle.

Common Trap

A report lists dP/dt of 1050 mmHg/s and calls LV contractility "normal" because it is above 1000. What is the error?

The canonical normal cutoff is > 1200 mmHg/s, not 1000. A value of 800 to 1200 is *mildly reduced*, and < 800 is severely reduced. The older 1000 figure was retired; 1050 mmHg/s is mildly reduced contractility.

LV mass (Devereux): $LV\ mass = 1.04 \times [(LVIDd + PWd + IVSd)^3 - LVIDd^3] - 13.6$ grams, then indexed to BSA. Normal LV mass index is ≤ 115 g/m² (men) and ≤ 95 g/m² (women). Elevated mass is the structural signature of pressure overload (hypertension, AS) and the substrate for diastolic dysfunction.

LV Diastolic Function Assessment (ASE/EACVI 2016 Algorithm)

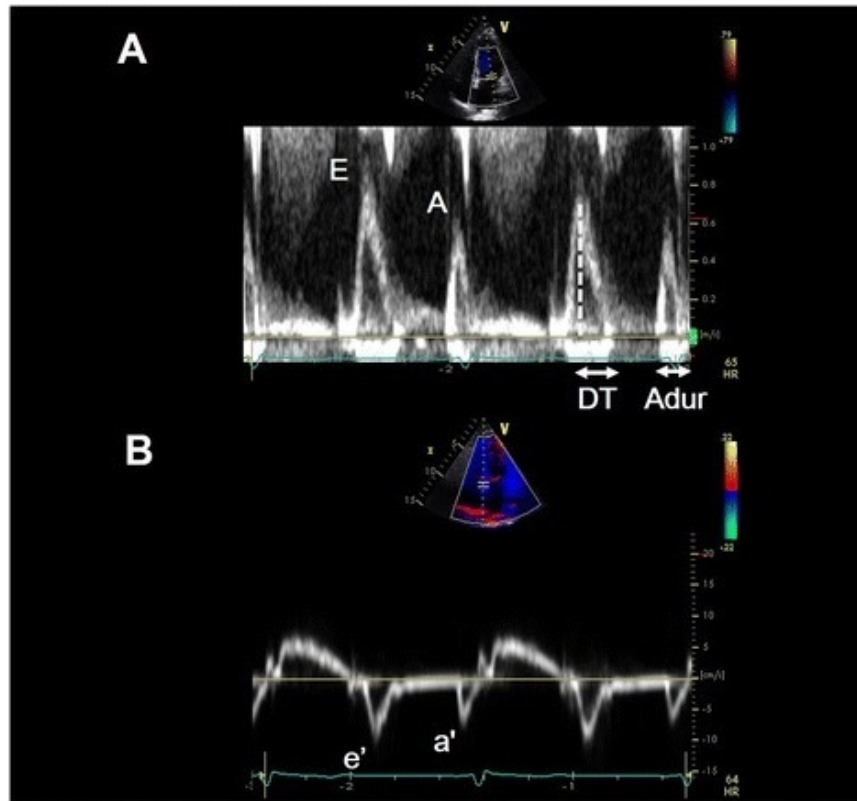
Diastole is how the ventricle relaxes and fills. When the LV stiffens, filling pressures rise, and the four-pillar workup is built to detect that rise even when EF is preserved (HFpEF). The four pillars are **average E/e'**, **septal and lateral e'**, **LA volume index**, and **peak TR velocity**.

Memory hook: (four diastolic pillars) "Every Echo Lab Tests" = E/e', e' (annular), LAVi, TR velocity. If two of four are abnormal, filling pressure is elevated.

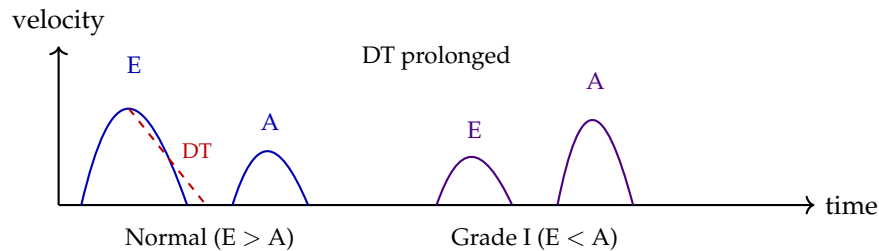
Pillar	Normal	Abnormal (2016)	Note
Average E/e'	< 8	> 14	8 to 14 borderline
Septal e' (cm/s)	≥ 7	< 7	2016 cutoff; prognostic < 6
Lateral e' (cm/s)	≥ 10	< 10	2016 cutoff; prognostic < 7
LAVi (mL/m ²)	< 28	> 34	28 to 34 indeterminate
TR Vmax (m/s)	≤ 2.8	> 2.8	suggests elevated PA pressure

These pillar cutoffs are the **ASE/EACVI 2016** values, which remain the registry-exam standard. The 2025 update lowers the e' thresholds (see the ASE 2025 update box below).

★ **Mitral inflow components:** E wave 60 to 100 cm/s (early passive filling), A wave 40 to 70 cm/s (atrial kick). E/A normal is 0.8 to 2.0; deceleration time normal is 160 to 200 ms; IVRT normal is 60 to 90 ms.

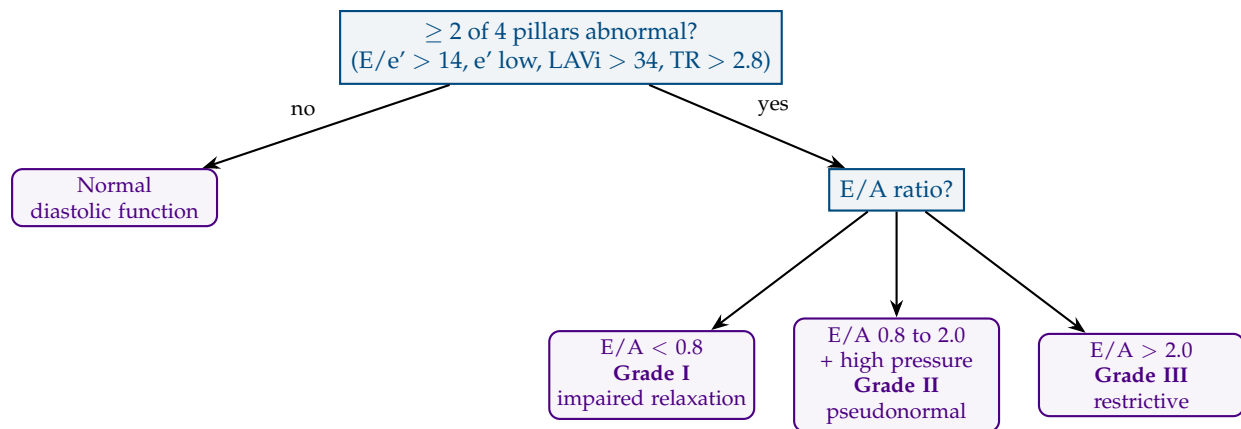


Mitral inflow pulsed-wave Doppler (panel A) showing the early (E) and late (A) diastolic filling waves; the dashed line follows the descending limb of the E wave and the DT arrow at the baseline marks the deceleration time, the interval from the E peak down to baseline along that down-slope (Adur marks A-wave duration). Panel B is the companion tissue Doppler tracing of the mitral annulus (e prime, a prime). Image by Jae-Hwan Lee and Jae-Hyeong Park via Clinical Hypertension (PMC4750785), CC BY 4.0.



Mitral inflow PW envelopes. Normal: tall E, shorter A (E/A 0.8 to 2.0). Impaired relaxation reverses the ratio (E/A < 0.8) with a prolonged deceleration time.

The grading flowchart. The single most tested diastolic concept is that an apparently “normal” E/A can be **pseudonormal** (Grade II): the inflow looks fine because rising LA pressure pushes the E wave back up, but the annular e' is low and the LA is enlarged. That is why e' and LAVi exist: to unmask pseudonormalization.



Simplified ASE/EACVI 2016 decision path (registry standard). Pillars first; if elevated, the E/A ratio sorts the grade.

★ **Full grading table (ASE/EACVI 2016, registry standard):**

Grade	E/A	DT (ms)	Lateral e' (cm/s)	E/e'	LAVi (mL/m ²)	PV pattern
Normal	0.8 to 2.0	160 to 200	> 10	< 8	< 28	S > D
I (impaired)	< 0.8	> 200	< 10	8 to 14	normal or high	S > D
II (pseudonormal)	0.8 to 2.0	160 to 200	< 10	> 14	> 34	S blunted/reversed
III (restrictive)	> 2.0	< 160	< 10	> 14	> 34	S reversed

The e' column lists the lateral cutoff for brevity; per the four-pillar definition, annular e' is abnormal when septal e' < 7 OR lateral e' < 10 cm/s (2016 cutoffs).

Memory hook: (grades I to III) "Stiff, sneaky, stuck." Grade I is just stiff (slow relaxation), Grade II is sneaky (looks normal but is not), Grade III is stuck (high pressure, fixed restrictive filling).

ASE 2025 update (labeled; the 2016 cutoffs above remain the registry-exam standard). The 2025 LV Diastolic Function and HFpEF update lowered the tissue-Doppler thresholds and added an atrial-strain variable. Abnormal: lateral e' ≤ 7 cm/s (was < 10), septal e' ≤ 6 cm/s (was < 7), average e' ≤ 6.5 cm/s (new averaged variable), and LA reservoir strain ≤ 18% (new variable). E/e' becomes site-specific (average ≥ 14, septal ≥ 15, lateral ≥ 13). Use the 2016 numbers as the registry answer; quote these only when a question explicitly references the 2025 guideline.

Common Trap

Mitral inflow shows E/A of 1.2 and a deceleration time of 180 ms, both squarely "normal." The tech signs out normal diastolic function. What is the error?

This can be **Grade II pseudonormal**. A normal-looking E/A with a low lateral e' (< 10), an E/e' > 14, and an enlarged LA (LAVi > 34) is elevated filling pressure masquerading as normal. Never grade diastole from inflow alone; always check the annular e' and LA size.

Pulmonary vein flow. Normal is systolic-dominant (S > D) with a small atrial reversal wave. As LA pressure climbs, the S wave blunts and then reverses, and the atrial reversal (Ar) lengthens. A pulmonary-vein Ar duration that exceeds the mitral A duration is a specific sign of elevated LV end-diastolic pressure.

PH special case. In moderate or severe pulmonary hypertension, use the *lateral* E/e' rather than the average: lateral E/e' > 13 indicates elevated LV filling pressure, < 8 is normal. The 2025 PH threshold is mean PAP > 20 mmHg; PASP > 35 to 40 mmHg by echo remains the practical screening cutoff.

Strain Imaging (Global Longitudinal Strain and GLS)

Strain measures how much the myocardium deforms (shortens) during systole; it catches dysfunction before EF drops. Because longitudinal fibers shorten toward the apex, GLS is reported as a **negative** percentage, and “worse” means closer to zero (less negative).

★ **Normal GLS:** men -17 to -24% , women -18 to -26% . Borderline is -16 to -17% ; clearly abnormal is worse than -16% (closer to zero). Because the sign trips people up, think in magnitude: a healthy heart shortens a lot (big negative number); a sick heart shortens little (small negative number).

Memory hook: (strain sign) “More minus is more muscle.” A normal LV is around -20% ; an amyloid or HFpEF heart drifts toward -10% to -15% .

Diagnosis	GLS threshold	RV strain	Notes
Normal LV	men -17 to -24% , women -18 to -26%	more negative than -20%	n/a
Reduced-EF cardiomyopathy HFpEF	worse than -16% $> -15\%$ (worse than expected)	often $> -15\%$ often abnormal	predicts adverse outcomes early marker of dysfunction
Amyloidosis (AL/TTR)	overall $\leq -16\%$; apical sparing ≥ 2	often $> -15\%$	“cherry-on-top” pattern
Acute MI (regional)	reduced GLS in territory, normal elsewhere	may be normal	viable if improves on stress

Pattern recognition. A territorial strain drop matches the infarct artery (LAD = anteroapical, RCA = inferobasal, LCx = lateral). **Takotsubo** (“apical ballooning”) shows apical dyskinesia with *normal* coronaries. **Amyloid** shows the cherry-on-top bullseye: dark (low strain) base and mid-walls with a bright (preserved) apex.

Common Trap

An amyloid bullseye plot shows preserved apical strain. The student concludes the apex is healthy and rules out infiltrative disease. What is the error?

Relative apical sparing is the hallmark of cardiac amyloidosis, not evidence against it. The base and mid-walls are severely impaired while the apex is relatively spared, producing the “cherry-on-top” map. An apical sparing ratio ≥ 2 supports amyloid.

RV and LA strain. RV free-wall strain normal is about -29% (abnormal worse than -20%); RV GLS including septum is about -17% normal (abnormal worse than -15%) and is prognostic in PH, HF, acute MI, and amyloid. LA reservoir strain (peak LAS) is normally about 39% ; the ASE 2025 diastolic update treats a peak LAS $\leq 18\%$ as the abnormal cutoff (a new diastolic variable), and a reduced reservoir strain also tracks with worse NYHA class and predicts AF recurrence after ablation.

Cardiac Hemodynamics Block (Carry-Forward Formulas)

These formulas were introduced in Weeks 1 and 4 and reappear here because ischemia and HF questions fold them into output and severity calculations. They are collected in the Essential Formulas section below; the registry expects you to apply them without a prompt.

Tool	What it answers	Key cutoff
Continuity (AVA)	aortic valve area	severe AS $< 1.0 \text{ cm}^2$
Dimensionless index	AS severity without diameter	severe < 0.25
Pressure half-time	mitral valve area	$MVA = 220/PHT$; not in AF/AR

Tool	What it answers	Key cutoff
PISA (EROA, RVol)	regurgitation severity	shift Nyquist toward jet
Modified Bernoulli	pressure gradient from velocity	use when $V_1 \geq 1.5$ m/s

Professional Ethics, Scope of Practice, and HIPAA

Three frameworks govern your conduct: the **CCI Code of Ethics** (cardiac sonographer standard), the **SDMS Code of Ethics** (revised 2024; effective 09/24/2024), and **HIPAA** (the 1996 federal privacy statute). The registry tests the principles and, more often, the application.

CCI Code of Ethics (core tenets). Maintain the highest standard of professional conduct; hold appropriate credentials (ARDMS RDCS or CCI RCS); follow imaging protocols; **practice within your scope**; protect patient privacy (HIPAA); refuse unethical requests; and **report unethical conduct to institutional leadership**.

SDMS Code of Ethics (revised 2024; three principles).

- **Memory hook: "W-C-I": Well-being, Competence, Integrity.**
- **Principle I (Promote patient well-being):** explain the procedure; respect autonomy and the right to refuse; provide non-judgmental care; protect privacy, dignity, and comfort (draping); maintain HIPAA confidentiality; promote safety.
- **Principle II (Promote competent practice):** obtain education and credentials; uphold protocols; **practice within scope and acknowledge personal and legal limits**; pursue continuing education; perform only physician-ordered, medically indicated studies; avoid impairing substances.
- **Principle III (Promote professional integrity and public trust):** communicate truthfully; avoid conflicts of interest; accurately represent credentials; bill ethically; and **report deviations from the Code to institutional leadership**.

Scope of practice. ★ The range of activities a sonographer is qualified, trained, and legally authorized to perform. You *are* qualified to acquire 2D, M-mode, spectral and tissue Doppler, color, contrast, stress echo, and (with training) TEE and strain. You are **not** authorized to interpret images, diagnose disease, modify medications, or perform interventional procedures. **Scope creep** is the trap: telling a patient "you have an enlarged heart," advising on anticoagulation, or performing a procedure outside your training.

Common Trap

During a study the patient asks, "Do I have a hole in my heart?" The sonographer, confident in the bubble study, says yes. **What is the error?**

Diagnosis and interpretation are the reading physician's scope, not the sonographer's. The correct response is to acquire and document the findings, reassure the patient that the physician will review and discuss results, and defer the interpretation. Answering crosses into scope creep regardless of how clear the image looks.

HIPAA essentials. The Health Insurance Portability and Accountability Act of 1996 protects patient privacy and the security of health information. **Protected Health Information (PHI)** is any data that can identify a patient in a healthcare context: name, MRN, phone, email, date of birth, images, notes, diagnoses, medication lists. De-identified data (identifiers removed) is not PHI. Use the **minimum necessary** information, secure and log off workstations, never share passwords, never email PHI to personal accounts, and report breaches to your compliance officer. ★ Breach reporting follows a three-tier rule: **affected individuals are always notified** (regardless of size); a breach affecting ≥ 500 individuals is also reported to **HHS and the media** without unreasonable delay (within 60 days); a breach affecting < 500 individuals is logged and reported to HHS annually. Common breaches: an open chart left on screen, discussing a patient in an elevator or cafeteria, or sharing identifiers with a student without consent.

Scope of Practice vs. Mandatory Reporting Cheat Sheet

Situation	Sonographer action	Scope decision
New aortic stenosis seen on a routine study	Report the measurement to the attending; do not diagnose	Within scope; interpretation is the physician's role
Patient asks "do I have a hole in my heart?"	Defer to the physician's report; do not answer	Outside scope; diagnosis requires a physician
Patient is unsafe (fall risk, altered mental status)	Notify clinical staff or nursing immediately	Mandatory reporting; patient safety
Suspected abuse or neglect indicators	Escalate to the supervisor or mandatory-reporter coordinator	Mandatory reporting; facility policy prevails
Colleague scanning incorrectly	Discuss privately; escalate if a safety concern	Professional responsibility; quality improvement
Patient discloses substance abuse or suicidal thoughts	Inform the clinical team immediately; do not minimize	Mandatory reporting; facility policy critical

Essential Formulas

Ejection fraction: $EF = \frac{EDV - ESV}{EDV} \times 100\%$ Plain English: the fraction of end-diastolic volume the LV pumps out per beat. Why it matters: it is the headline systolic number; preserved is $\geq 50\%$, and the EF band drives nearly every HF management decision.

Fractional shortening: $FS = \frac{LVIDd - LVIDs}{LVIDd} \times 100\%$ Plain English: how much the LV cavity narrows in one M-mode line. Why it matters: a quick linear systolic proxy when a full Simpson trace is not feasible; normal is 25 to 45%.

dP/dt (contractility): $dP/dt = \frac{32}{\Delta t}$ Plain English: how fast the LV builds pressure, timed from 1 to 3 m/s on the MR jet (the 32 is the gradient difference between those two points: $4 \cdot 3^2 - 4 \cdot 1^2 = 36 - 4 = 32$ mmHg). Why it matters: a load-light index of contractility; normal > 1200 mmHg/s, severely reduced < 800 .

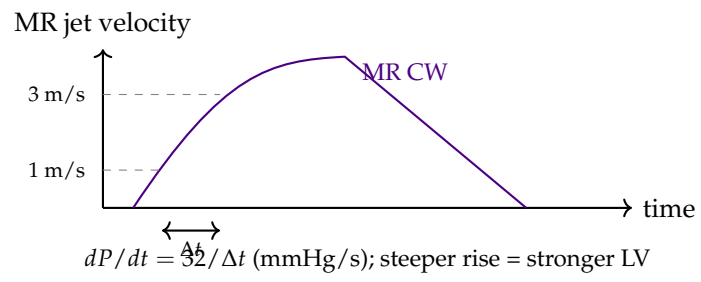
Stroke volume and output: $SV = CSA_{LVOT} \times VTI_{LVOT}$, $CO = SV \times HR$, $CI = \frac{CO}{BSA}$ Plain English: volume per beat, per minute, and normalized to body size. Why it matters: low output ($CO < 4$, $CI < 2.0$) defines the hemodynamic severity of heart failure.

E/e' (filling pressure): $E/e' = \frac{\text{mitral E}}{e'}$ Plain English: the inflow speed divided by how fast the annulus recoils. Why it matters: a noninvasive surrogate for LV filling pressure; average > 14 is elevated and is a core diastolic pillar.

Global longitudinal strain: $GLS = \frac{L_2 - L_1}{L_1} \times 100\%$ where L_1 is the end-diastolic (baseline) myocardial length and L_2 is the end-systolic (shortened) length. Plain English: the percent the myocardium shortens end to end (reported negative). Why it matters: detects subclinical dysfunction before EF falls and reveals signature patterns (amyloid apical sparing, territorial infarct).

LV mass (Devereux): $LVM = 1.04[(LVIDd + PWd + IVSd)^3 - LVIDd^3] - 13.6$ Plain English: cube the outer dimension, subtract the cube of the cavity, scale by muscle density. Why it matters: indexed mass > 115 (men) or > 95 (women) g/m² confirms hypertrophy, the substrate for diastolic dysfunction.

Modified Bernoulli: $\Delta P = 4(V_2^2 - V_1^2)$ Plain English: turns a Doppler velocity into a pressure gradient; drop V_1 when it is < 1.5 m/s to get $\Delta P = 4V_2^2$. Why it matters: the backbone of every gradient, RVSP, and dP/dt timing in this guide.



Topic Cheat Sheet: Best View and Modality

Task	Best view	Modality	Normal / cutoff
EF (Simpson biplane)	A4C + A2C	2D trace	preserved $\geq 50\%$
Fractional shortening	PLAX	M-mode	25 to 45%
dP/dt	A4C (MR jet)	CW Doppler	$> 1200 \text{ mmHg/s}$
Mitral E and A	A4C, MV tips	PW Doppler	E 60 to 100; A 40 to 70 cm/s
Deceleration time	A4C, MV tips	PW Doppler	160 to 200 ms
Septal / lateral e'	A4C, annulus	TDI	$\geq 7 / \geq 10 \text{ cm/s}$
LA volume index	A4C + A2C	area-length	$< 28 \text{ mL/m}^2$
TR peak velocity	A4C, RV inflow	CW Doppler	$\leq 2.8 \text{ m/s}$
GLS	A4C, A2C, A3C	speckle tracking	men -17 to -24 , women -18 to -26%
RWMA scoring	PLAX, PSAX, A4C, A2C	2D	all segments score 1

Glossary

- **EF** — ejection fraction; percent of LV emptied per beat
- **FS** — fractional shortening; M-mode systolic proxy
- **LVOT** — left ventricular outflow tract; continuity reference
- **VTI** — velocity-time integral; Doppler envelope area (cm)
- **dP/dt** — rate of LV pressure rise; contractility estimate
- **GLS** — global longitudinal strain; percent fiber shortening
- **E/A** — early-to-atrial mitral inflow velocity ratio
- **DT** — deceleration time; E-wave downslope duration (ms)
- **IVRT** — isovolumetric relaxation time (ms)
- **LAVi** — left atrial volume index (mL/m^2)
- **e'** — tissue-Doppler annular early diastolic velocity
- **E/e'** — surrogate for LV filling pressure
- **RWMA** — regional wall motion abnormality; ischemia marker
- **LAD / LCx / RCA** — the three main coronary arteries
- **PDA** — posterior descending artery; defines dominance
- **Pseudonormal** — Grade II diastolic pattern masking high pressure
- **Restrictive** — Grade III; high pressure, fixed filling
- **Apical sparing** — preserved apical strain in amyloidosis
- **Takotsubo** — apical ballooning with normal coronaries
- **HFpEF** — heart failure with preserved ejection fraction
- **Viability** — biphasic stress response; muscle worth revascularizing
- **LV mass index** — LV mass / BSA (g/m^2); hypertrophy marker

Registry Connection

ARDMS RDCS subdomain: Pathology (ischemic disease, heart failure, diastolic dysfunction) plus Measurement/Views (EF, strain, Doppler indices). **CCI RCS:** Domain D (integration of pathology with hemodynamics) and Domain C (measurement). **Item formats to expect:** match an RWMA segment set to its coronary artery; read a diastolic grade from a four-pillar data set (and catch pseudonormal); pick the correct EF/FS/dP/dt band from canonical cutoffs; recognize the amyloid "cherry-on-top" strain map; calculate SV, CO, and E/e' on demand.

Carry forward into Week 8: the TR-velocity and RVSP work you previewed here becomes the full pulmonary-pressure and great-vessel workup (aorta, pericardium, tamponade) next week; the right-heart strain and PH thresholds carry straight over.

Study smart. Scan with confidence.

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