

Week 4: Valvular Pathology

CVSG2280 Cardiovascular Registry Review by Dr. Alice Matthews

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Registry weight this week. Valvular pathology is the single heaviest topic on both exams. On the **ARDMS RDCS (Adult Echo V24.2)** blueprint it feeds the **Pathology** domain (40% of the exam, the largest single domain) and overlaps the **Measurement and Hemodynamics / Views** domain (25%); smaller pieces touch Anatomy/Physiology (15%), Clinical Care and Safety (11%), and Instrumentation (9%). On the **CCI RCS** blueprint it maps mainly to **Domain D (29%, integration and pathology)** and **Domain B (24%, acquisition and measurement)**, with support from C (25%), E (13%), and A (9%). Roughly one in three registry questions you see will be a valve question.

Note to students: Cutoffs in this guide follow the free ASE guideline landing pages: [ASE 2017 Native Valvular Regurgitation](#) and [EACVI/ASE 2017 Stenosis](#), with management timing from the [2020 ACC/AHA Valvular Heart Disease guideline](#) (Otto et al.). All normal values match the course *Canonical Values* reference so your numbers agree across every week. **What last week's EOM gives you:** Week 3 physics is why Doppler velocities are trustworthy here; the Bernoulli, continuity, and Nyquist ideas you learned then are the engine behind every severity grade below. Your lab may use a specific guideline year; always defer to the protocol your clinical site follows.

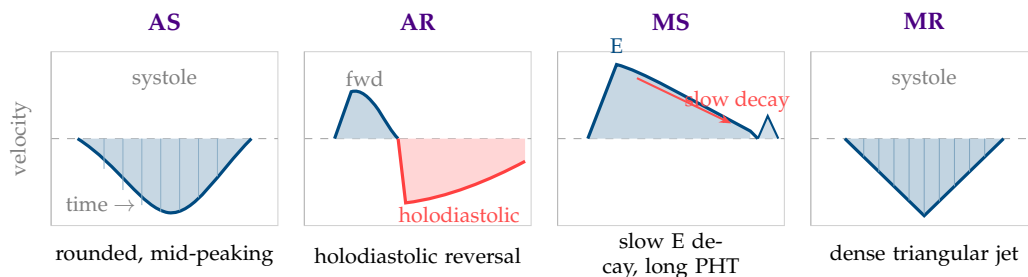
At a Glance: this week in one box

Must-know numbers. Severe AS: peak > 4 m/s, mean gradient > 40 mmHg, AVA < 1.0 cm², DVI < 0.25 . Severe MS: MVA < 1.0 cm², mean gradient > 10 mmHg, PHT > 220 ms. Severe primary MR: VC ≥ 0.7 cm, EROA ≥ 0.40 cm², RVol ≥ 60 mL. Normal valve areas: aortic 2.5 to 4.0, mitral 4.0 to 6.0, tricuspid 7.0 to 9.0, pulmonic 2.5 to 4.0 cm².

Formulas you will reuse. Bernoulli $\Delta P = 4V^2$; continuity AVA = $(CSA_{LVOT} \times VTI_{LVOT}) / VTI_{AV}$; $MVA = 220 / PHT$; PISA EROA = $2\pi r^2 V_{alias} / V_{max}$; $RVSP = 4V_{TR}^2 + RAP$.

Top 3 EOM traps. (1) Grading MR with primary cutoffs when the leaflets are normal and the ventricle is dilated (use the lower *secondary* thresholds). (2) Using $MVA = 220 / PHT$ on a prosthetic mitral valve or in atrial fibrillation. (3) Reporting RVSP as pulmonary artery pressure when pulmonic stenosis is also present.

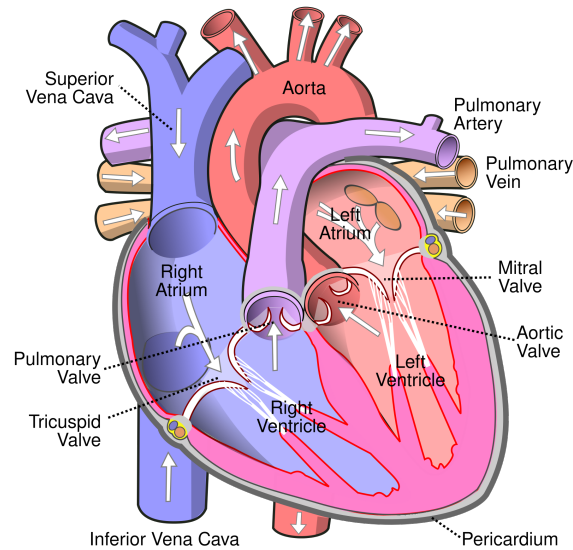
The Four Valves and Registry Fundamentals



Classic spectral Doppler "signatures" of the four valve lesions for pattern recognition: AS (dense rounded mid-peaking systolic CW), AR (descending-aorta holodiastolic flow reversal), MS (tall mitral E wave with slow deceleration / long pressure half-time), MR (dense symmetric triangular high-velocity systolic CW jet).

The heart has **four valves** that keep blood moving in one direction. Two **atrioventricular (AV)** valves sit between the atria and the ventricles: the **tricuspid** on the right and the **mitral** (bicuspid) on the left. Two **semilunar valves** guard the ventricular outflow: the **pulmonic** valve into the pulmonary artery and the **aortic** valve into the aorta. Every valve disease is either a problem opening (**stenosis**) or a problem closing (**regurgitation**). The sections below

walk through each valve in turn, then summarize processes that target multiple valves at once. The formulas you will use at the machine are collected at the end of the guide.



Coronal cross-section. Blue = deoxygenated (right heart); red = oxygenated (left heart). Arrows trace one full circuit. Image by Wapcaplet via Wikimedia Commons (CC BY-SA 3.0).

Valve disease at a glance. ★ Aortic stenosis dominates adult valvular disease in the United States; mitral regurgitation is second. Right-sided valve lesions are usually functional rather than structural, with trace TR or PR being normal findings on most adult echoes.

Valve	Most common adult pathology	Approximate US prevalence
Aortic	Calcific stenosis	2 to 3% over 65; 3 to 5% over 75
Mitral	Prolapse or regurgitation	MVP $\approx$ 2 to 3%; MR is the 2nd most common VHD
Tricuspid	Functional regurgitation	Trace TR in 80 to 90% of adults; moderate+ in 0.5 to 1%
Pulmonic	Trace regurgitation (physiologic)	Pathologic PV disease is rare in adults

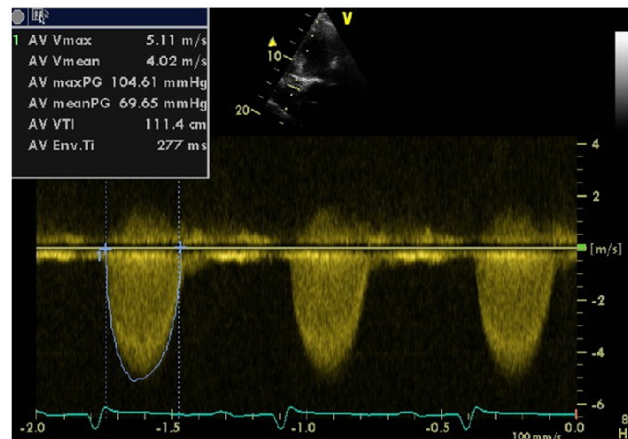
Aortic Valve: Stenosis and Regurgitation

The aortic valve is the three-leaflet semilunar valve between the left ventricle and the aorta; it opens in systole to deliver oxygenated blood to the body. ★ Calcific aortic stenosis is the most common valvular heart disease in the United States, present in roughly 2 to 3% of adults over 65 and 3 to 5% of adults over 75; bicuspid aortic valve, the most common congenital heart abnormality, affects another 1 to 2% of the general population and accelerates the same disease decades earlier.

Aortic stenosis (AS). Thickened, calcified leaflets; restricted systolic opening; high-velocity, dense CW signal that peaks in mid-systole (rounded, not late-peaking); concentric LV hypertrophy. Most common causes are age-related calcification and congenital bicuspid valve. Sample the peak velocity from the apical 5-chamber, suprasternal notch, and right parasternal windows; the true peak often comes from the non-apical windows with a Pedoff (standalone non-imaging CW) probe.

Parameter	Mild	Moderate	Severe
Peak velocity (m/s) ★	< 3.0	3.0 to 4.0	> 4.0
Mean gradient (mmHg) ★	< 20	20 to 40	> 40
AVA (cm ²) ★	> 1.5	1.0 to 1.5	< 1.0
Indexed AVA (cm ² /m ²)	> 0.85	0.60 to 0.85	< 0.60
Dimensionless index	> 0.50	0.25 to 0.50	< 0.25

- ★ **Very severe AS:** some guidelines further subclassify severe AS as $AVA < 0.6 \text{ cm}^2$ with mean gradient $> 60 \text{ mmHg}$. On the registry, severe is severe; in the cath lab, very severe changes management.
- **Low-flow, low-gradient AS:** use dobutamine stress echo. True severe AS shows AVA remaining $< 1.0 \text{ cm}^2$ with the gradient rising past 40 mmHg as flow increases. Pseudo-severe AS shows AVA rising above 1.0 cm^2 as the ventricle contracts more vigorously.



Continuous-wave spectral Doppler across a stenotic aortic valve (apical window) in a 56-year-old adult with severe aortic stenosis. The dense, high-velocity systolic envelope peaks at 5.11 m/s (peak gradient 105 mmHg , mean gradient 70 mmHg), consistent with severe valvular AS. Image by Alammar AK via PMC (Case Reports in Cardiology 2014;2014:213673), CC BY 3.0.

Common Trap

A 78-year-old has a calcified aortic valve. Peak velocity sampled only from the apical 5-chamber is 3.6 m/s (moderate). The sonographer reports moderate AS and moves on. **What is the error?**

The apical window often underestimates the true peak. AS velocity must be interrogated from *multiple* windows (suprasternal, right parasternal, and with a Pedoff probe), and the highest reproducible velocity is reported. The non-apical window may reveal a peak $> 4 \text{ m/s}$, reclassifying the patient as severe.

Aortic regurgitation (AR). Diastolic backflow from aorta to LV. Causes: bicuspid valve, endocarditis, annular dilation, rheumatic disease, aortic root pathology. Acute AR (endocarditis, dissection) gives a normal-sized LV with high filling pressure and a short PHT; chronic AR gives a dilated, volume-loaded LV. M-mode shows diastolic flutter of the anterior mitral leaflet with holodiastolic AR.

Parameter	Mild	Moderate	Severe
Vena contracta (cm) ★	< 0.3	0.3 to 0.6	> 0.6
PHT (ms)	> 400	200 to 400	< 200
Jet width / LVOT width (%)	< 25	25 to 64	≥ 65
Jet CSA / LVOT CSA (%)	< 5	5 to 59	≥ 60
Regurg volume (mL/beat)	< 30	30 to 44	> 44
Regurg fraction (%)	< 30	30 to 40	> 40
EROA (cm^2)	< 0.10	0.10 to 0.29	> 0.30

★ **Red flag:** holodiastolic flow reversal in the descending thoracic aorta (PW) confirms severe AR, especially if end-diastolic velocity is $> 20 \text{ cm/s}$.

Pitfalls. Sampling only the apical 5-chamber for peak AV velocity; the true peak often comes from the suprasternal notch or right parasternal window with the Pedoff probe. Measuring LVOT diameter at the wrong level; sample 2 to 3 mm proximal to the valve in zoomed PLAX, mid-systole, inner edge to inner edge.

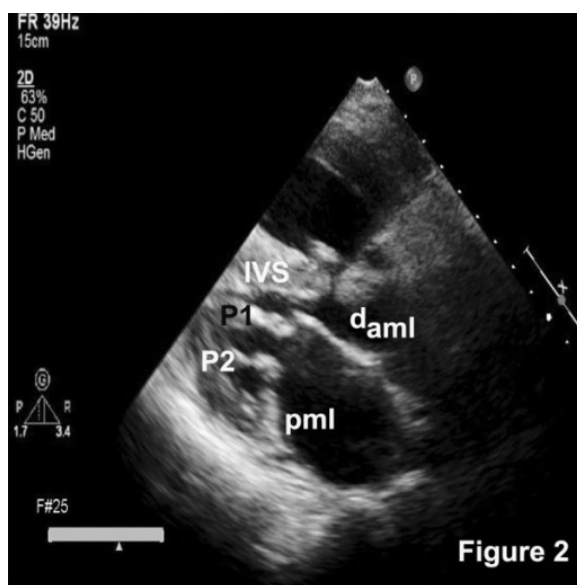
Mitral Valve: Stenosis and Regurgitation

The mitral (bicuspid) valve sits between the left atrium and left ventricle, anchored to the ventricular wall by chordae tendineae and the anterolateral and posteromedial papillary muscles; it opens in diastole to fill the LV. ★ Mitral valve prolapse is the single most common valve abnormality in adults at roughly 2 to 3% of the population, and mitral regurgitation is the second most common valvular disease overall after aortic stenosis; mitral stenosis is now rare in the United States but remains the dominant valvular lesion worldwide because of rheumatic disease.

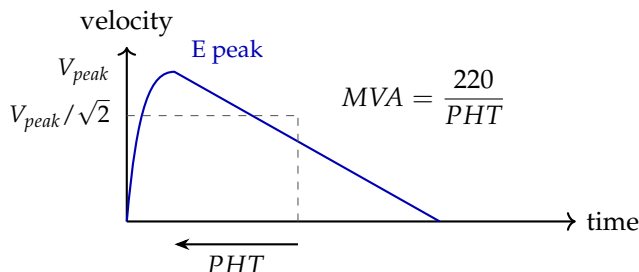
Mitral stenosis (MS). Thickened leaflets with commissural fusion; “hockey stick” doming of the anterior leaflet (rheumatic); dilated left atrium with possible spontaneous echo contrast or appendage thrombus. Causes elevated LA pressure and possible atrial fibrillation. Normal MVA is 4.0 to 6.0 cm².

Parameter	Mild	Moderate	Severe
MVA (cm ²) ★	> 1.5	1.0 to 1.5	< 1.0
Mean gradient (mmHg) ★	< 5	5 to 10	> 10
PHT (ms)	< 150	150 to 220	> 220
Pulmonary artery pressure	Normal	30 to 50 mmHg	> 50 mmHg

- **Planimetry** (tracing the orifice in short axis at mid-diastole) is gold standard when image quality allows. Be careful not to trace the mitral annulus; trace the tips of the open leaflets.
- **Pulmonary venous flow:** systolic blunting or reversal indicates significant MS with elevated LA pressure.



Rheumatic mitral stenosis, parasternal long axis in diastole: the thickened anterior mitral leaflet (labeled d-aml) shows the doming or hockey-stick morphology; pml is the posterior leaflet, IVS the septum. No arrow is drawn; follow the d-aml label. Image by Krishna CS et al. via Open-i (PMC2390577), CC BY 2.0.



Pressure half-time: time for peak inflow velocity to fall to $V_{peak} / \sqrt{2}$ ($\approx 70\%$ of peak). The slower the decay, the tighter the valve.

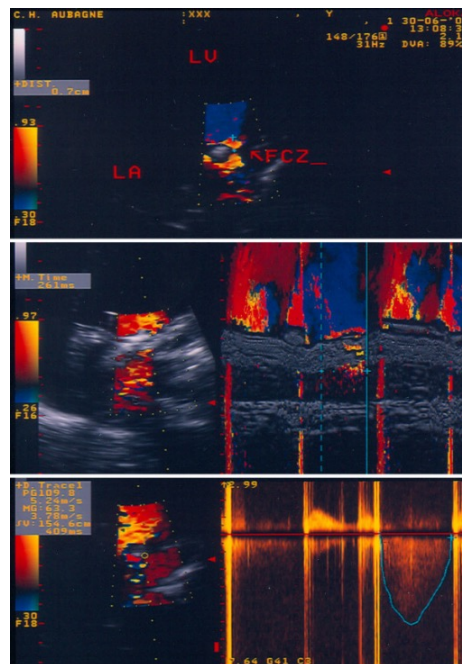
Mitral regurgitation (MR). Backflow from LV to LA during systole. **Primary (organic) MR:** structural leaflet damage (prolapse, flail, endocarditis, rheumatic, annular calcification). **Secondary (functional) MR:** normal leaflets with poor coaptation from LV dilation or annular stretch (tethered coaptation). Both show a dilated LA and LV and a reduced EF in chronic severe disease.

★ **This is the most heavily tested MR concept: the severity cutoffs differ by whether the leaflets are structurally normal.**

Parameter (severe)	Primary MR (leaflets damaged)	Secondary MR (leaflets normal, ventricle dilated)
Vena contracta (cm)	≥ 0.7	≥ 0.4
EROA (cm^2)	≥ 0.40	≥ 0.20
Regurg volume (mL/beat)	≥ 60	≥ 30
Regurg fraction (%)	≥ 50	≥ 50

Note: the secondary-MR severe regurgitant-volume threshold varies between guideline versions and societies (some retain ≥ 30 mL, others use ≥ 45 to 60 mL because of the smaller orifice but higher chronicity); always read which criterion your protocol applies. The lower EROA $\geq 0.20 \text{ cm}^2$ cutoff above is the classic registry answer (traced to ESC 2017 and outcome data), not an ASE figure; the 2020 ACC/AHA VHD guideline grades severe secondary MR at EROA $\geq 0.40 \text{ cm}^2$ with RVol ≥ 60 mL, the same threshold as primary MR. Report which criterion your protocol uses.

- **CW density:** an MR signal approaching antegrade density suggests severe MR.
- ★ **Pulmonary vein systolic flow reversal** is highly specific for severe MR when the jet aims at that vein.
- **Integrated approach:** no single parameter is perfect. Use VC, PISA, CW density, pulmonary venous flow, and structural clues together.



Mitral regurgitation by the PISA (flow-convergence) method: the red FCZ arrow marks the hemispheric color-convergence zone, with the 0.7 cm caliper as the PISA radius (aliasing 30 cm/s). Shown here on a prosthetic mitral regurgitation; the method is identical for native MR. Image by Arques S et al. via Open-i (PMC2644672), CC BY 2.0.

Common Trap

A patient with a dilated cardiomyopathy and structurally normal mitral leaflets has an EROA of 0.25 cm^2 . Using the primary-MR table (severe ≥ 0.40), the tech calls this "moderate." **What is the error?**

This is *secondary* MR (normal leaflets, dilated ventricle), so the lower secondary thresholds apply: EROA ≥ 0.20 cm² is severe. An EROA of 0.25 cm² is **severe secondary MR**, not moderate. The orifice is smaller in secondary MR but carries a worse prognosis, which is why the cutoffs are lower.

Prolapse. One or both leaflets billow > 2 mm above the annular plane into the LA during systole (measure in parasternal long axis). Myxomatous change shows leaflets > 5 mm thick with elongated chords. Complications include flail leaflet, severe MR, and endocarditis risk.

- ★ **Apical 4-chamber false positive:** the mitral annulus is saddle-shaped, so an apical view can make a normal leaflet look like it prolapses. Always confirm prolapse in the parasternal long axis.

Common Trap

On the apical 4-chamber view, the anterior mitral leaflet appears to bow toward the left atrium in systole. The tech diagnoses mitral valve prolapse. **What is the error?**

The saddle shape of the mitral annulus makes normal leaflets appear to cross the annular plane in the apical 4-chamber view, a classic false positive. Prolapse must be confirmed in the **parasternal long axis**, where billowing > 2 mm above the annular plane is the true diagnostic criterion.

Pitfalls. Measuring the PISA radius to the wrong aliasing shell; shift the Nyquist baseline toward the jet, then measure to the first crisp aliasing line at peak flow. Using PHT for MVA in atrial fibrillation or with concurrent AR; average several cycles and prefer planimetry when AR shortens the decay slope.

Tricuspid Valve: Regurgitation and Stenosis

The tricuspid valve is the three-leaflet AV valve between the right atrium and right ventricle, anchored by chordae to three papillary muscles (anterior, posterior, and septal). ★ Trace tricuspid regurgitation is normal and seen in 80 to 90% of healthy adult echoes; clinically significant (moderate or greater) TR is present in roughly 0.5 to 1% of the general population and rises sharply with age. Most TR is **functional** (secondary to RV or annular dilation, pulmonary hypertension, or atrial fibrillation) rather than caused by a structural leaflet problem.

Regurgitation. Functional causes (RV dilation, pulmonary hypertension, annular stretch) are most common. Organic causes: rheumatic disease, Ebstein anomaly, carcinoid syndrome, endocarditis (IV drug use). Findings: dilated RV and RA, flat septum, and paradoxical septal motion.

Parameter	Mild	Moderate	Severe
Vena contracta (cm)	No validated cutoff	< 0.7	≥ 0.7
CW density / contour	Faint, parabolic	Dense, variable	Dense, triangular
Hepatic vein systolic flow	S $>$ D	Blunted S	Systolic reversal

Note on TR vena contracta: a single mild cutoff is not validated, so VC < 0.7 cm spans the mild-to-moderate range; VC ≥ 0.7 cm is severe. Grade with the integrated picture (CW density, hepatic vein flow, RV/RA size), not VC alone.

- ★ **RVSP estimation** is the most important quantitative task: use the TR jet velocity with Bernoulli plus estimated RAP.
- **PISA for TR is less validated** than for MR or AR. Use it as an adjunct, not a standalone severity measure.

Stenosis. Rare; almost always rheumatic and accompanied by mitral disease. Thickened leaflets with restricted diastolic opening and doming. A mean gradient > 5 mmHg and valve area < 1.5 cm² are consistent with significant stenosis. Normal TV area is 7.0 to 9.0 cm².

Pitfalls. Underestimating TR velocity from a poorly aligned signal; sample apical 4-chamber, parasternal RV inflow, and subcostal, and use agitated saline contrast if the envelope is weak. Equating RVSP with PASP when PS coexists; subtract the pulmonic peak gradient before reporting PASP.

Pulmonic Valve: Stenosis and Regurgitation

The pulmonic valve is the three-leaflet semilunar valve between the right ventricle and the main pulmonary artery.

★ Trace pulmonary regurgitation is physiologic and visible in roughly three-quarters of normal adult echoes; pathologic PR is uncommon outside of repaired tetralogy of Fallot. Pulmonary stenosis is almost always congenital and accounts for about 8 to 10% of all congenital heart defects, making the pulmonic valve the least frequently diseased valve in routine adult practice.

Stenosis. Usually congenital (isolated valvular PS or tetralogy of Fallot); acquired causes are rare. Thickened, doming leaflets; post-stenotic dilation of the main pulmonary artery; RV hypertrophy.

Parameter	Mild	Moderate	Severe
Peak velocity (m/s)	< 3.0	3.0 to 4.0	> 4.0
Peak gradient (mmHg)	< 36	36 to 64	> 64

Regurgitation. Often physiologic; pathologic causes include repaired tetralogy, endocarditis, carcinoid, or severe pulmonary hypertension. Diastolic flow reversal in the main pulmonary artery (color or PW). Jet width and duration correlate with severity.

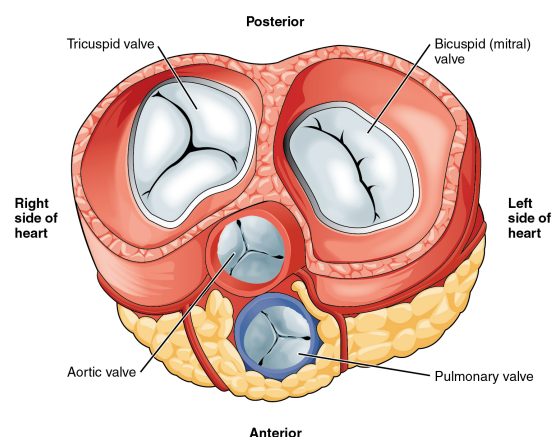
Parameter	Mild	Moderate	Severe
Jet width / annulus (%)	< 25	25 to 46	> 46
PHT (ms)	> 100	80 to 100	< 80

- Note: some guidelines use < 100 ms as the severe cutoff for PR PHT. Consistency within your lab matters more than any single number.

Pitfalls. Calling trace PR pathologic; physiologic PR is visible in roughly three-quarters of normal echoes. Off-axis sampling underestimates PV velocity; confirm beam alignment in subcostal short axis or parasternal short axis at the base.

Diseases Affecting Multiple Valves

A handful of processes target several valves at once. Recognize the pattern first, then evaluate each valve individually with the criteria above.



Superior view of the cardiac fibrous skeleton. All four valves sit in a single plane, which is why diffuse processes such as rheumatic disease, endocarditis, and carcinoid can involve more than one valve at the same time. Image by OpenStax College via Wikimedia Commons (CC BY 3.0).

Process	Valves typically affected	Key sonographic clues
Rheumatic heart disease	MV > AV > TV (rarely PV)	Commissural fusion; “hockey stick” doming; thickened tips with mobile bodies
Infective endocarditis	Native: MV or AV; IV drug use: TV	Oscillating vegetations; new regurg; perforation; perivalvular abscess on prosthetics
Carcinoid heart disease	TV + PV (right-sided only)	Pearly thickened, retracted, fixed leaflets; severe TR with PS in the same patient
Marfan / connective tissue	AV root and MV	Aortic root > 4 cm with AR; bileaflet MV prolapse
Radiation heart disease	AV, MV, often pericardium	Annular and basal leaflet thickening with tip sparing; pericardial thickening
Drug-induced (fen-phen, ergots)	MV, AV, TV	Restrictive thickening with chordal shortening (mimics carcinoid on the left side)
Degenerative / senile calcific	AV (calcific AS); MV annular calcification	Heavy calcium on leaflet bodies; commissures spared (versus rheumatic)
Functional / secondary	MV, TV	Structurally normal leaflets; dilated ventricle or annulus; tethered coaptation
Bicuspid aortic valve	AV (early AS or AR) plus aortopathy	Two leaflets; eccentric closure line; ascending aortic dilation

- ★ **Bicuspid AV** is the single most common congenital heart abnormality (1 to 2% of the population) and the leading cause of AS or AR before age 65.
- ★ **Carcinoid affects right-side valves only** because the vasoactive metabolites are cleared by the lungs. **Memory hook:** think “carcinoid = right side only” (TV and PV); if a left-side carcinoid lesion appears, look for a right-to-left shunt (PFO) or pulmonary metastases.
- **Rheumatic mitral stenosis** is now uncommon in the United States but remains the leading cause of MS worldwide. **Memory hook:** rheumatic order of attack is “Mitral first, Aortic second” (MV > AV > TV > PV).

Common Trap

A patient has thickened, retracted tricuspid and pulmonic leaflets with severe TR and pulmonic stenosis, plus flushing and diarrhea. The tech also reports a thickened mitral valve from the same carcinoid process. **What is the error?**

Classic carcinoid heart disease is **right-sided only** because the lungs inactivate the vasoactive serotonin metabolites before they reach the left heart. Left-sided carcinoid lesions should prompt a search for a **right-to-left shunt (PFO)** or **pulmonary metastases**; do not attribute a left-sided lesion to carcinoid without one of those explanations.

Prosthetic Valve Assessment

Prosthetic valves are either **mechanical** (bileaflet, tilting disk, or older ball-in-cage; durable but require lifelong anticoagulation) or **bioprosthetic** (stented or stentless tissue; no long-term anticoagulation but degenerate over 10

to 20 years). ★ Every prosthesis is mildly stenotic by design, so “normal” Doppler numbers run higher than for a native valve; compare against valve-specific reference ranges and, ideally, the patient’s own baseline study.

The single most useful screening number is the dimensionless index (DVI). For an aortic prosthesis a falling DVI signals obstruction; for any prosthesis a new or rising gradient compared with baseline is the red flag.

Quick screening thresholds (any prosthesis):

Finding	Suggests normal function	Suggests obstruction
Aortic prosthesis peak velocity	< 3 m/s	> 4 m/s
Aortic prosthesis mean gradient	< 20 mmHg	> 35 mmHg
Aortic DVI	> 0.35	< 0.25
Mitral prosthesis mean gradient	< 5 mmHg	> 10 mmHg
Mitral prosthesis PHT	< 130 ms	> 200 ms

Note: an aortic prosthesis mean gradient of 20 to 35 mmHg is indeterminate, not normal; per the ASE prosthetic valve guideline it is possible obstruction and requires the DVI, the acceleration time, and comparison with the patient’s baseline study before you call it normal or obstructed.

★ **Size-specific normal Doppler ranges (aortic prosthesis).** A “high” gradient on a small prosthesis can still be normal; always check the size.

Valve type and size	Normal peak velocity (m/s)	Normal mean gradient (mmHg)
17 mm mechanical	3.6 to 4.2	9 to 11
19 mm mechanical	3.1 to 3.9	7 to 9
21 mm mechanical	2.7 to 3.4	5 to 7
23 mm mechanical	2.3 to 2.9	4 to 6
19 mm bioprosthetic	2.2 to 2.6	3 to 5
21 mm bioprosthetic	1.9 to 2.4	2 to 4
23 mm bioprosthetic	1.6 to 2.1	1 to 3

Size-specific normal Doppler ranges (mitral prosthesis).

Valve type and size	Normal peak velocity (m/s)	Normal mean gradient (mmHg)
25 mm mechanical	2.2 to 2.8	4 to 6
27 mm mechanical	1.9 to 2.5	3 to 5
29 mm mechanical	1.7 to 2.3	2 to 4
27 mm bioprosthetic	1.6 to 2.0	2 to 4
29 mm bioprosthetic	1.4 to 1.8	1 to 3

- ★ **Do not use $MVA = 220/PHT$ on a prosthetic mitral valve;** the constant was derived for native rheumatic valves and is invalid here.
- Normal “washing” jets:** mechanical valves have small, symmetric, built-in regurgitant jets by design. A new, eccentric, or paravalvular jet is pathologic.
- Acoustic shadowing and reverberation** from the prosthesis hide the structure behind it; image a mechanical mitral valve from the TEE/left-atrial side and an aortic prosthesis from multiple windows.

Common Trap

A 21 mm mechanical aortic prosthesis shows a peak velocity of 3.2 m/s and a mean gradient of 6 mmHg. The tech compares this to native cutoffs (peak > 3 is moderate AS) and reports prosthetic stenosis. **What is the error?**

Prosthetic valves are inherently stenotic, so native cutoffs do not apply. For a 21 mm mechanical aortic valve, a peak of 2.7 to 3.4 m/s and a mean gradient of 5 to 7 mmHg are **normal**. Use size-specific reference ranges, the DVI, and the patient's baseline study before calling prosthetic obstruction.

Pitfalls. Comparing a prosthetic gradient to native-valve cutoffs and over-calling stenosis; always use prosthesis-specific ranges or the patient's baseline. Missing paravalvular regurgitation because shadowing hides it; sweep around the sewing ring and use color off-axis.

Endocarditis and Valve Infection

Infective endocarditis is infection of the valve leaflets or endocardium, classically producing **vegetations** (irregular, mobile, low-reflective masses attached to the upstream/low-pressure side of a leaflet that move independently of the leaflet). It causes new regurgitation, leaflet perforation, and, on prosthetics, perivalvular abscess and dehiscence. Native left-sided valves (MV, AV) are most commonly involved; ★ **right-sided (tricuspid) endocarditis is associated with intravenous drug use.**

Feature	Typical endocarditis finding
Vegetation	Oscillating, low-reflective mass on the upstream leaflet side
Motion	Independent, chaotic motion separate from the leaflet
Result	New or worsening regurgitation; perforation; flail
Prosthetic clue	Perivalvular abscess, rocking valve, new paravalvular leak
TEE role	Far more sensitive than TTE for small vegetations and abscess

- **Duke criteria** combine echo findings (vegetation, abscess, new dehiscence, new regurgitation) with microbiologic and clinical data to make the diagnosis; the sonographer documents the echo evidence.
- ★ **TEE is the test of choice** when endocarditis is suspected and TTE is negative or the patient has a prosthesis.

Pitfalls. Mistaking a normal variant (Lambl's excrescence, nodule of Arantius, suture material) for a vegetation; correlate with the clinical picture and motion. Calling a negative TTE a rule-out for endocarditis; recommend TEE when suspicion is high.

Essential Formulas

Simplified Bernoulli: $\Delta P = 4V^2$ \ Plain English: a faster jet means a bigger pressure difference. **Why it matters:** converts any CW velocity into a gradient (AS, MS, TR). Use the modified form $\Delta P = 4(V_2^2 - V_1^2)$ when the proximal velocity $V_1 \geq 1.5$ m/s.

Continuity (AVA): $AVA = \frac{CSA_{LVOT} \times VTI_{LVOT}}{VTI_{AV}}$ \ Plain English: what flows into the LVOT must come out the valve, so a smaller orifice needs a faster jet. **Why it matters:** this is the backbone of AS grading and is gradient-independent. $CSA_{LVOT} = \pi(d/2)^2$.

Dimensionless index (DVI): $DVI = \frac{VTI_{LVOT}}{VTI_{AV}}$ \ Plain English: a velocity ratio that needs no diameter measurement. **Why it matters:** the most error-resistant AS screen; severe AS when < 0.25 , and a falling DVI flags prosthetic obstruction.

Pressure half-time (MVA): $MVA = \frac{220}{PHT}$ \ Plain English: the slower the mitral inflow decays, the tighter the valve. **Why it matters:** fast bedside MVA. Do *not* use it on a prosthetic mitral valve, in atrial fibrillation, or with significant AR. Normal MVA is 4.0 to 6.0 cm².

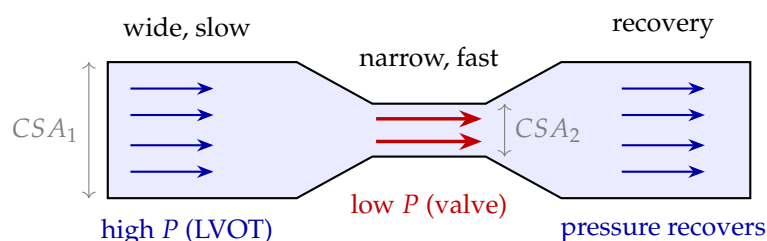
PISA (regurgitation): $EROA = \frac{2\pi r^2 \times V_{alias}}{V_{max}}$ and $RVol = EROA \times VTI_{regurg}$ \ Plain English: flow accelerates into hemispheric shells before a leak; measure one shell to back-calculate the orifice. **Why it matters:** quantifies MR/AR severity; shift the Nyquist baseline toward the jet and measure r to the first crisp aliasing shell (V_{alias} usually 30 to 40 cm/s).

RVSP via TR: $RVSP = 4(V_{TR})^2 + RAP$ \ Plain English: the speed of the TR jet plus the right atrial pressure equals RV systolic pressure. **Why it matters:** the chief quantitative task in TR and the main echo screen for pulmonary hypertension. Estimate RAP from IVC size and collapse.

Common Trap

Pulmonic stenosis and tricuspid regurgitation coexist. The tech computes $RVSP = 4V_{TR}^2 + RAP = 70$ mmHg and reports pulmonary artery systolic pressure of 70 mmHg. **What is the error?**

With PS present, $RVSP \neq PASP$. The stenotic pulmonic valve drops pressure between the RV and the pulmonary artery. Subtract the peak **pulmonic** gradient ($4V_{PS}^2$) from the RVSP to estimate true PASP; otherwise pulmonary pressure is badly overestimated.



Continuity: $CSA_1 \cdot V_1 = CSA_2 \cdot V_2$ • Bernoulli: $\Delta P = 4V^2$ across the narrowing.

Doppler View Cheat Sheet

Valve	Stenosis	Regurgitation
Aortic	CW: apical 5-chamber or suprasternal	CW: apical 5-chamber

Valve	Stenosis	Regurgitation
Mitral	PW at leaflet tips; PHT from inflow	PISA: apical 4-chamber; CW across valve
Tricuspid	PW at leaflet tips; mean gradient	CW: parasternal RV inflow or apical 4-chamber
Pulmonic	CW: parasternal short axis or subcostal	CW: parasternal short axis

Glossary

- **VC** — vena contracta; narrowest part of a regurgitant jet
- **EROA** — effective regurgitant orifice area (cm²)
- **PISA** — proximal isovelocity surface area method
- **CW / PW** — continuous-wave / pulsed-wave Doppler
- **VTI** — velocity-time integral (Doppler envelope area, cm)
- **PHT** — pressure half-time; time for V to fall to $V/\sqrt{2}$
- **LVOT** — left ventricular outflow tract
- **LVESD** — LV end-systolic diameter
- **RAP** — right atrial pressure (from IVC size + collapse)
- **RVSP** — right ventricular systolic pressure
- **MAC** — mitral annular calcification
- **DVI** — dimensionless index ($\text{LVOT VTI} \div \text{AV VTI}$; severe AS < 0.25)
- **Holodiastolic** — present throughout diastole
- **Commissural fusion** — fused leaflet edges; hallmark of rheumatic disease
- **Coaptation** — overlap of leaflets when closed; poor coaptation causes functional regurg
- **Planimetry** — direct tracing of a valve orifice in short axis
- **Vegetation** — mobile infective mass on the upstream leaflet side
- **Eccentric closure** — off-axis closure line (e.g., bicuspid AV)
- **Nyquist** — max unambiguous velocity ($\text{PRF}/2$); above this, signal aliases
- **Pedoff probe** — standalone CW transducer for high-velocity AS jets

Registry Connection

Where this week lands on the registry. ARDMS RDCS (Adult Echo) subdomain: **Pathology** (valvular stenosis and regurgitation, prosthetics, endocarditis), with heavy overlap into **Measurement and Hemodynamics** (continuity, PISA, PHT, RVSP). CCI RCS: primarily **Domain D** (integration of pathology) and **Domain B** (acquisition and measurement). **Item formats to expect:** severity-grading questions that hand you peak velocity, mean gradient, AVA, and DVI together and ask for one answer; "which window/which formula" matching; primary-versus-secondary MR cutoff distinctions; calculate-MVA-then-decide-when-not-to-use-PHT items; estimate RVSP from a TR envelope; and image identification (hockey-stick MS, dense high-velocity AS jet, PISA convergence, vegetation). **Carry forward into Week 5:** the same Doppler quantification skills and the integrated-severity habit move into procedural and critical-findings echo, where new or worsening regurgitation becomes an urgent communication.

Study smart. Scan with confidence.

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