

Valvular Disease Study Guide

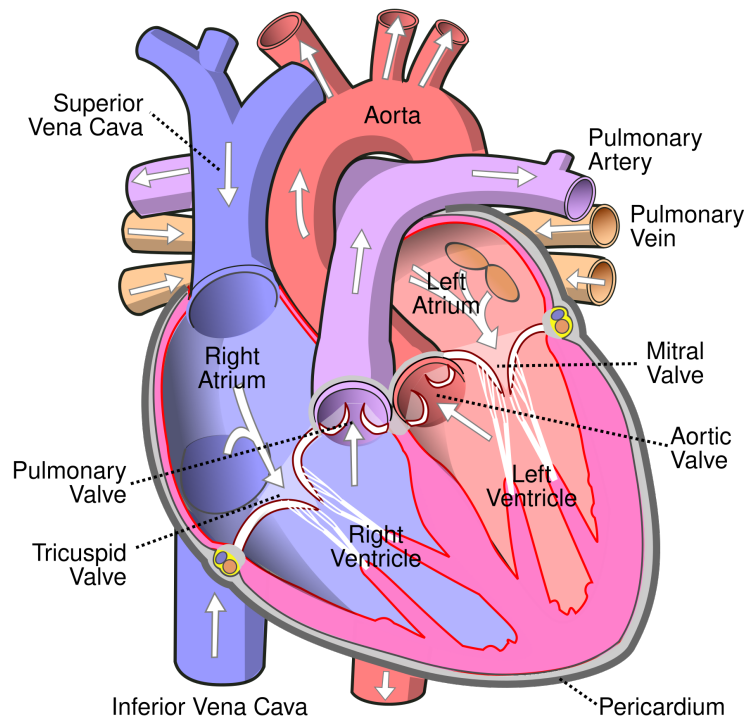
Cardiac Ultrasound Reference by Dr. Alice Matthews

amatthews@concorde.edu · Concorde Career College Portland

Note to students: Cutoffs in this guide are drawn from the [ASE 2017 regurgitation](#) and [EACVI/ASE 2017 stenosis](#) recommendations and the [2020 ACC/AHA VHD guideline](#) (Otto et al.). Your lab may use a specific guideline year; always defer to the protocol your clinical site follows.

The Four Valves of the Heart

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

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.

Stenosis: Thickened, calcified leaflets; restricted systolic opening; high-velocity, dagger-shaped cw signal; concentric LV hypertrophy. Most common causes are age-related calcification and congenital bicuspid valve.

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 into AVA < 0.6 cm² with mean gradient > 60 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 cm² with gradient rise > 40 mmHg as flow increases. Pseudo-severe AS shows AVA rising above 1.0 cm² as the ventricle contracts more vigorously.

Regurgitation: Diastolic backflow from aorta to LV. Causes: bicuspid valve, endocarditis, annular dilation, rheumatic disease, aortic root pathology. Diastolic flutter of anterior mitral leaflet on M-mode (with holodiastolic AR); LV dilation (chronic).

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 46	> 46
Jet CSA / LVOT CSA (%)	< 5	5 to 20	> 20
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 velocity > 20 cm/s.

Pitfalls. Sampling only the apical 5-chamber for peak AV velocity; 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.

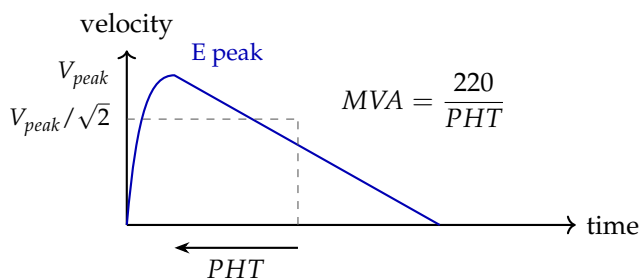
Mitral Valve

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.

Stenosis: Thickened leaflets with commissural fusion; “hockey stick” doming of anterior leaflet (rheumatic); dilated left atrium with possible spontaneous echo contrast or appendage thrombus. Causes elevated LA pressure and possible atrial fibrillation.

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 pressure	Normal	30 to 50 mmHg	> 50 mmHg

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



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.

Regurgitation: Backflow from LV to LA during systole. Organic causes: prolapse, flail, endocarditis, rheumatic damage, annular calcification. Functional MR: normal leaflets with poor coaptation from LV dilation/annular stretch. Dilated LA and LV; reduced EF in chronic severe disease.

This is the most common registry question on MR severity: the cutoffs differ by whether the valve leaflets are structurally normal.

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

- **CW density:** 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 + structural clues together.

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. An apical view can make a normal leaflet look like it prolapses. Always confirm in the parasternal long axis before calling prolapse.

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

The tricuspid valve is the three-leaflet atrioventricular 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 echos; 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: Most often functional from RV dilation or pulmonary hypertension (annular stretch). Organic causes: rheumatic disease, Ebstein anomaly, carcinoid syndrome, endocarditis (IV drug use). Dilated RV and RA; flat septum; paradoxical septal motion may be present.

Parameter	Mild	Moderate	Severe
Vena contracta (cm)	Not defined	< 0.7	> 0.7
CW density / contour	Faint, parabolic	Dense, variable	Dense, triangular
Hepatic vein systolic flow	S > D	Blunted S	Systolic reversal

- **RVSP estimation** is the most important quantitative task: use 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. Mean gradient > 5 mmHg and valve area < 1.5 cm² are consistent with significant stenosis.

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

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 echos; 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 main pulmonary artery; RV hypertrophy.

Parameter	Mild	Moderate	Severe
Peak velocity (m/s)	< 3	3 to 4	> 4
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). Duration and width of the regurgitant jet correlate with severity.

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

Essential Formulas

Simplified Bernoulli: $\Delta P = 4V^2$ (peak gradient from cw jet velocity; use modified form $\Delta P = 4(V_2^2 - V_1^2)$ when $V_{proximal} \geq 1.5$ m/s)

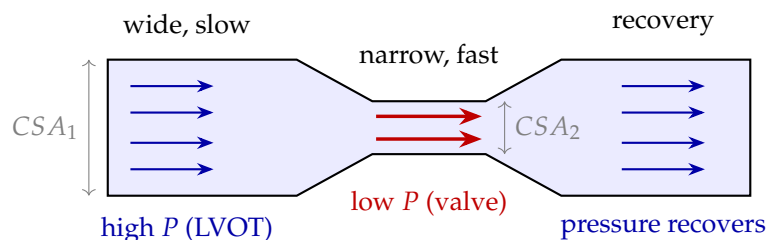
Continuity AVA: $AVA = \frac{CSA_{LVOT} \times VTI_{LVOT}}{VTI_{AV}}$ (conservation of mass; compare LVOT inflow to aortic valve outflow; shortcut ratio of peak velocities works if spectral shapes are similar)

Pressure Half-Time MVA: $MVA = \frac{220}{PHT}$ (do not use for prosthetic mitral valves; normal MVA is 4.0 to 6.0 cm²)

PISA (EROA): $EROA = \frac{2\pi r^2 \times V_{alias}}{V_{max}}$ and $RV = EROA \times VTI_{regurg}$ (measure PISA radius from leaflet tips to first aliasing shell; V_{alias} is the Nyquist limit, usually 30 to 40 cm/s)

RVSP via TR: $RVSP = 4(V_{TR})^2 + RAP$ (use cw of apical 4-chamber or parasternal RV inflow; estimate RAP from IVC size and collapsibility)

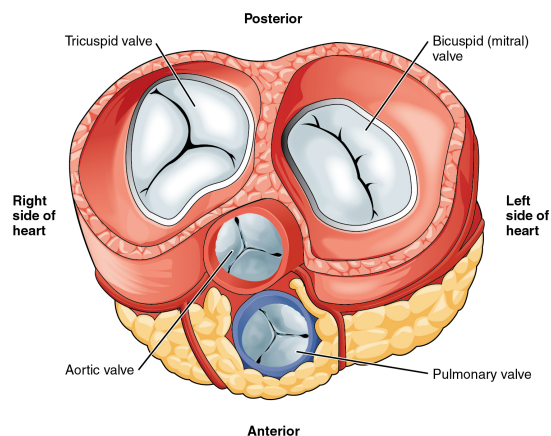
Important caveat: If pulmonic stenosis is present, do not equate RVSP with pulmonary artery systolic pressure. Measure the peak pulmonic velocity and subtract that gradient from RVSP to estimate true PASP.



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

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, pergolide)	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. If left-side carcinoid lesions appear, 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.

Doppler View Cheat Sheet

Valve	Stenosis	Regurgitation
Aortic	CW: apical 5-chamber or suprasternal	CW: apical 5-chamber
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 (cm^2)
- **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)
- **MAC** — mitral annular calcification
- **DI** — dimensionless index ($\text{LVOT VTI} \div \text{AV VTI}$; severe AS < 0.25)
- **Nyquist** — max unambiguous velocity ($\text{PRF}/2$); above this, signal aliases

Study smart. Scan with confidence.

[Dr. Alice Matthews](#) · deepneuro.ai/alice

Dr. Alice Matthews · amatthews@concorde.edu · alice@deepneuro.ai · Concorde Career College Portland