

Dynamic Left Ventricular Outflow Tract Obstruction (DLVOTO)

Key bedside message: Consider DLVOTO in a patient with hypotension or shock, a hyperdynamic “great-looking” LV, and worsening or limited response to escalating catecholamines.

Definition

Dynamic LVOT obstruction is a non-fixed, systolic narrowing of the left ventricular outflow tract. It typically occurs when a structurally susceptible, small, hyperdynamic LV becomes underfilled and/or exposed to increased contractility or reduced afterload.

Mechanism and pathophysiology

The usual mechanism is systolic anterior motion (SAM) of the mitral valve. Reduced preload, reduced afterload, tachycardia, and catecholamine-driven hypercontractility narrow the LV cavity and accelerate systolic flow through the LVOT.

- **Drag forces:** high-velocity systolic flow pushes the anterior mitral leaflet toward the septum.
- **Venturi effect:** once the leaflet enters the high-velocity stream, local pressure falls and pulls it further into the LVOT.
- **Mitral regurgitation:** anterior leaflet displacement prevents normal leaflet coaptation, often causing posteriorly directed MR and elevated left atrial pressure.

DLVOTO versus fixed intraventricular obstruction (FIVO)

Feature	DLVOTO	FIVO
Level of obstruction	Subaortic / LVOT	Mid-cavitary
SAM and MR	Usually present	Absent or trivial
Most useful clue	Late-peaking, dagger-shaped Doppler profile with LVOT aliasing	Doppler localizes obstruction to the mid LV cavity
Management nuance	Treat preload, afterload, heart rate, contractility, and MR/left atrial pressure physiology	Management overlaps, but SAM-related MR is not the dominant issue

Clinical impact

- **Hypotension or shock:** reduced forward cardiac output from dynamic obstruction.
- **Pulmonary edema:** SAM-related mitral regurgitation can raise left atrial pressure.
- **Paradoxical worsening:** inotropes, vasodilators, diuresis, tachycardia, and IABP unloading may worsen obstruction in DLVOTO.

Predisposing factors and triggers

Predisposing substrate	Physiologic triggers
LV hypertrophy, basal septal hypertrophy, or sigmoid septum	Reduced preload: hypovolemia, hemorrhage, diuresis, positive pressure ventilation
Small LV cavity	Tachycardia or atrial fibrillation with loss of atrial kick
Mitral valve apparatus abnormalities	Reduced afterload: sepsis, anesthesia, vasoplegia, post-AVR afterload loss
Hypertrophic cardiomyopathy or hypertensive cardiomyopathy	Increased contractility: endogenous sympathetic tone or exogenous inotropes/inodilators
Post-surgical mitral valve geometry changes	RV failure causing decreased LV filling and leftward septal shift

Common clinical scenarios

- Vasodilatory shock, including sepsis and post-cardiotomy systemic inflammatory response.
- Hypovolemia, particularly in a small LV or during positive pressure ventilation.
- Hypertrophic phenotypes, including hypertensive cardiomyopathy with sigmoid septum or HOCM.
- Stress or ischemic cardiomyopathy with apical hypokinesis and basal hypercontractility, such as Takotsubo or LAD infarction.
- RV failure with reduced LV preload or ventricular interdependence, including high-risk or intermediate-high-risk PE, pulmonary hypertension with RV-PA uncoupling, and post-cardiotomy RV failure.
- Post-cardiac surgery states, especially post-SAVR/TAVR for aortic stenosis and post-mitral valve repair or replacement.

Diagnosis: focused echocardiography approach

Consider dynamic LVOTO in any patient with shock and a “great-looking EF” on echocardiogram and lack of clinical response to increasing dose of vasopressors.

- Dynamic LVOTO can be identified by presence of SAM on M-mode and mosaic of colors suggesting high-velocity flow on continuous flow Doppler.
- Dynamic LVOTO is confirmed and the severity can be assessed by estimating the LVOT gradient by using combination of pulse and continuous flow Doppler.

Use 2D imaging, color Doppler, M-mode, pulsed-wave Doppler, and continuous-wave Doppler to confirm dynamic obstruction, localize it, and estimate severity.

1. **2D imaging:** look for a small, hyperdynamic LV; LV hypertrophy; basal septal hypertrophy >15 mm; or sigmoid septum.

[Clip - image 2D A3C view- hyperdynamic hypertrophied LV with pronounced basal septal hypertrophy; source EMCrit Scott Weingart and Trina Augustin](#)

2. **Color Doppler:** look for LVOT aliasing and asymmetric posteriorly directed MR.

Clips from EMCrit - Scott Weingart and Trina Augustin

- [Aliasing in The Out Flow Tract-LVOT Obstruction by western Sono](#)
- [Posteriorly directed MR & aliasing in LVOT](#)

3. **M-mode:** place the cursor through the anterior mitral leaflet to identify SAM during systole (the anterior MV leaflet moves toward and or touches the septum); also assess for aortic valve mid-systolic notching, fluttering, or closure in severe obstruction.

4. **Pulsed-wave Doppler:** sample sequentially from the LV cavity toward the LVOT to localize the obstruction and distinguish LVOT from mid-cavitary obstruction.

5. **Continuous-wave Doppler:** measure high-velocity flow and estimate the LVOT gradient. The classic pattern is late systolic peaking with a dagger-shaped contour.

6. **Always rule out the RV as the primary driver of DLVOTO:** RV dilation, septal shift, or RV failure may be the primary driver of an underfilled and compressed LV causing both decreased LV preload and narrowing of LVOT. Clips from EMCrit – Scott Weingart and Trina Augustin:

- [RVF DLVOTO](#)
- [RVF DLVOTO](#)

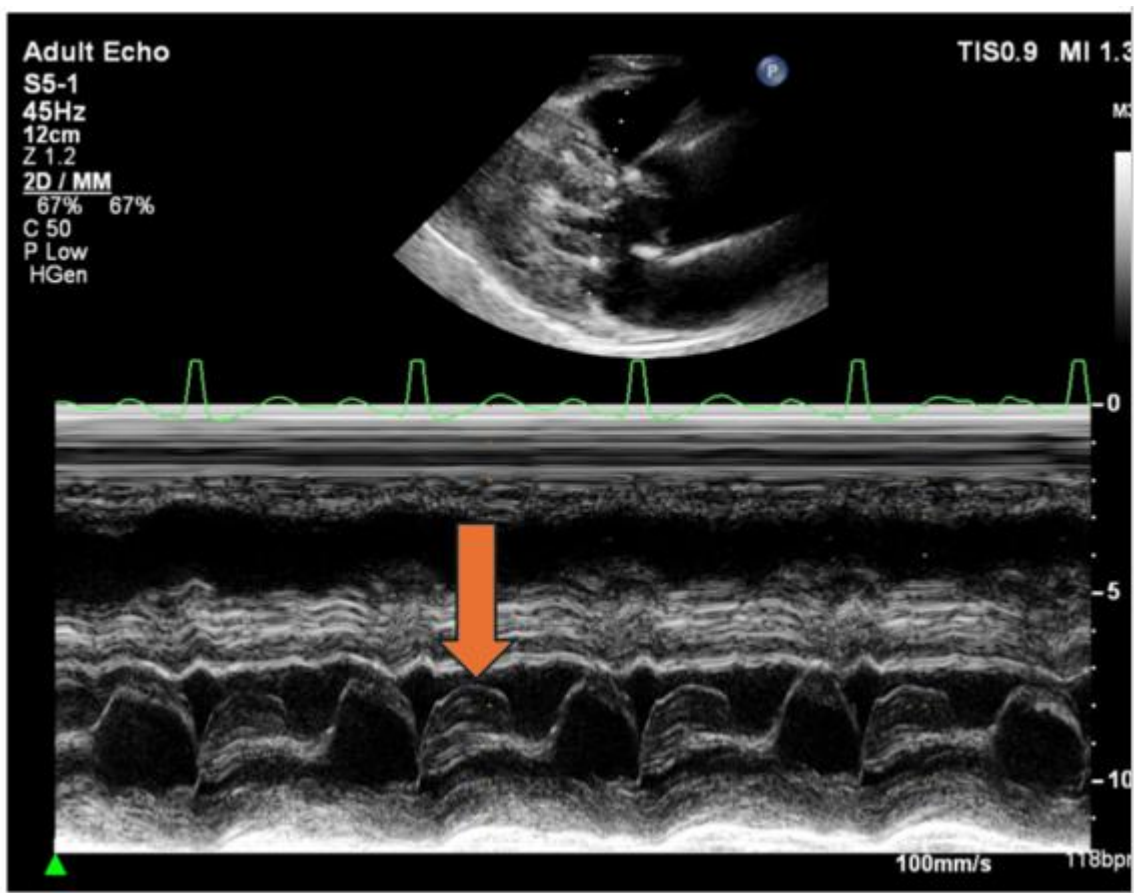
Diagnostic thresholds

- Peak instantaneous gradient >30 mmHg supports DLVOTO.
- Gradient >50 mmHg is generally hemodynamically significant.

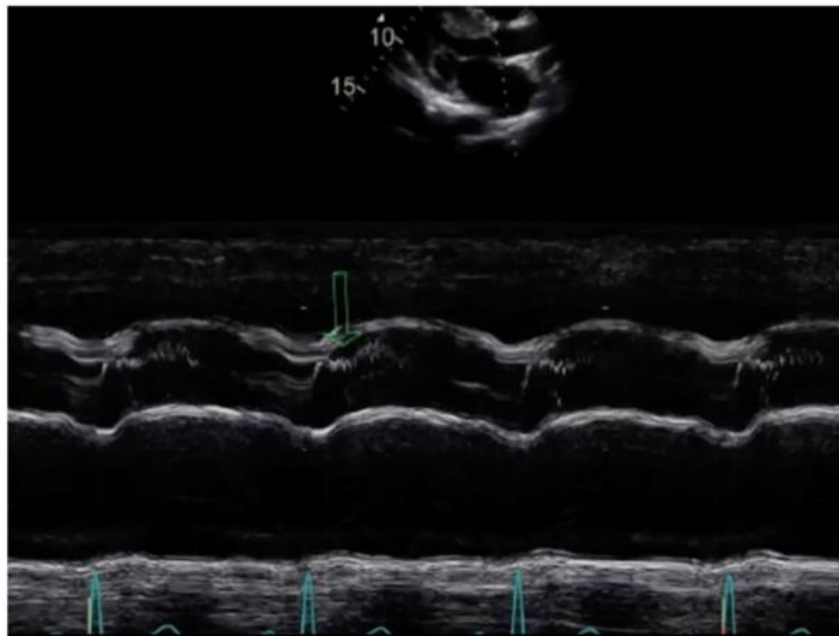
Representative echo findings

Pictures and clips from EMCrit Scott Weingart and Trina Augustin

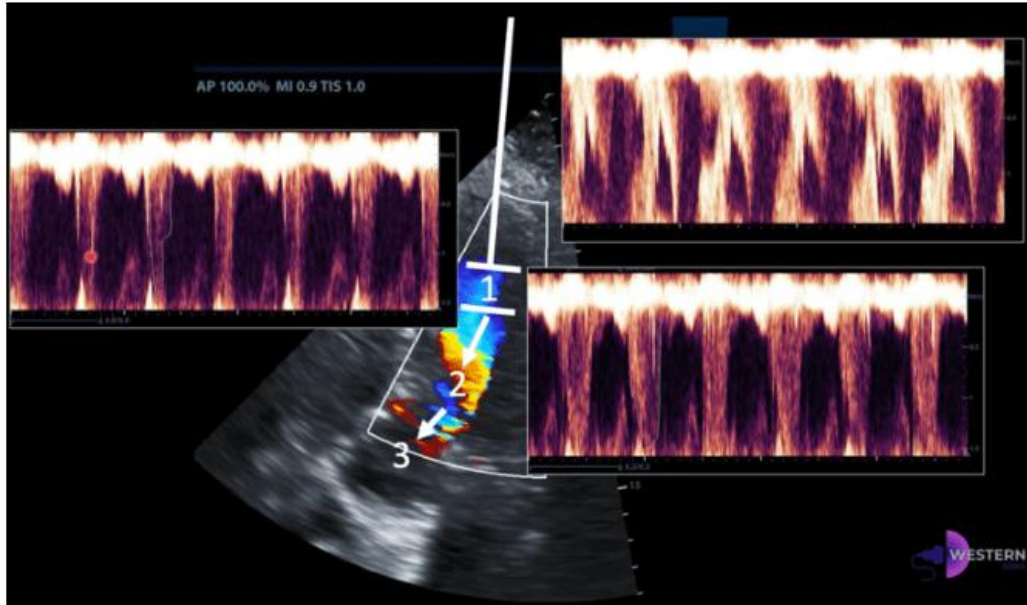
M-mode example demonstrating systolic anterior motion (SAM).



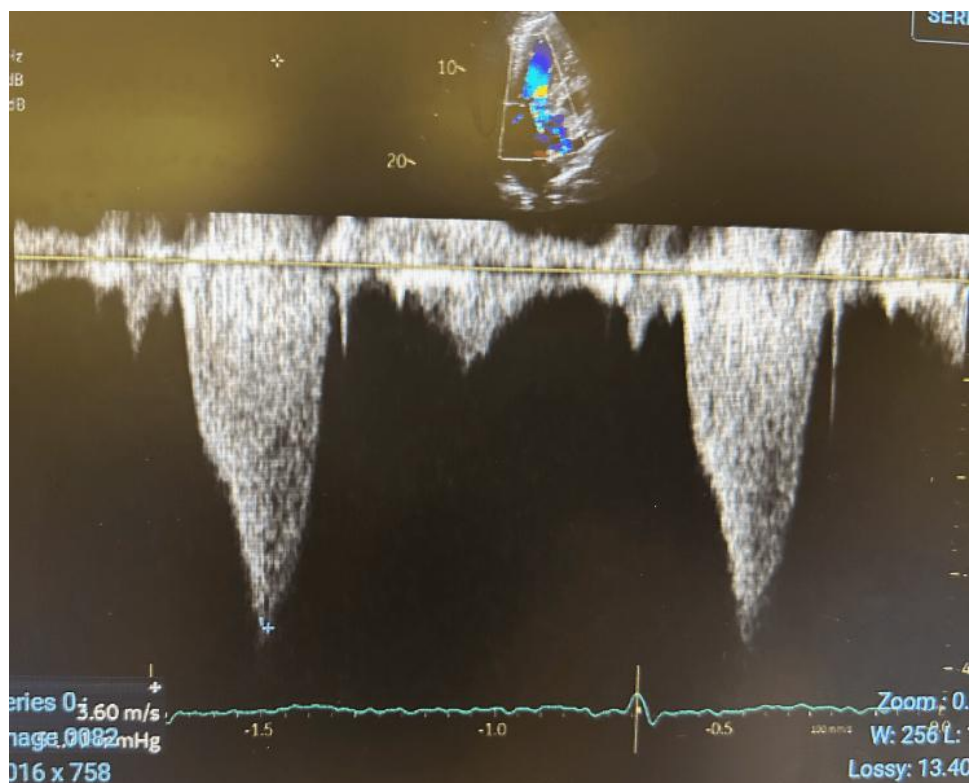
M-mode example demonstrating **aortic valve mid-systolic notching or fluttering**.



Aliasing in The Out Flow Tract-LVOT Obstruction by westernsono, note prominent aliasing (bidirectional waveform) that can be a clue of a dynamic LVOTO



Color/PWD example showing prominent LVOT aliasing and bidirectional high-velocity flow. Image courtesy noted in the source document.



Continuous-wave Doppler example with late-peaking, dagger-shaped dynamic LVOT obstruction profile. Image courtesy noted in the source document.

Always evaluate the RV

Dynamic LVOTO - concise review

Rule out RV failure as the primary driver of dynamic LVOTO

Features of RV failure associated with LVOTO: increased RV to LV ratio, marked septal shift (causing both decreased LV preload and narrowing of LVOT.)

- [RVF DLVOTO](#)
- [RVF DLVOTO](#)

Management framework

Treatment depends on the dominant physiology. First decide whether DLVOTO is primary, with preserved ventricular function and an underfilled hyperdynamic LV, or secondary to RV failure.

Clinical pattern	Usual physiology	Management priority
Primary DLVOTO with preserved ventricular function	Small hyperdynamic LV, reduced preload/afterload, tachycardia, high catecholamine state	Increase preload and afterload, slow heart rate, stop inotropes, reduce contractility
Secondary DLVOTO driven by RV failure	RV dilation/failure reduces LV filling and shifts septum leftward, narrowing the LVOT	Treat RV failure and RV afterload; avoid interventions that worsen RV dilation or contractility

Primary DLVOTO with preserved ventricular function

Common settings include sepsis, vasoplegia, hypovolemia, and post-cardiotomy inflammatory states in patients with susceptible LV anatomy.

Goal	Actions
Optimize preload	Stop diuretics when appropriate. Give 250-500 mL crystalloid and reassess frequently.
Increase afterload	Phenylephrine or vasopressin are preferred because they raise afterload without beta-agonism. Norepinephrine may be second line; monitor for beta-1 effects. Avoid epinephrine when possible.
Reduce contractility	Stop inotropes and inodilators that worsen hypercontractility and SAM.
Slow heart rate	Target a slower rate than presentation, often 55-70 bpm if tolerated. Consider short-acting beta-blockade such as esmolol only after fluids, afterload optimization, and stopping inotropes.
Maintain sinus rhythm	Treat atrial fibrillation promptly. Use amiodarone or cardioversion as indicated. In post-cardiac surgery patients, prefer AAI pacing if AV conduction is intact; use DDD if needed; avoid VVI pacing when possible.
Avoid LV unloading that worsens obstruction	IABP reduces afterload and can worsen DLVOTO. If discontinuing, be mindful that thrombus may form quickly; pause only briefly or remove as soon as feasible.

Secondary DLVOTO driven by RV failure

In a susceptible LV, RV failure can reduce LV preload and shift the septum leftward, producing an underfilled, compressed LV and precipitating SAM/DLVOTO.

- **Avoid treating it like primary DLVOTO:** indiscriminate fluid loading can worsen RV dilation and pericardial constraint; beta-blockade can depress RV contractility.
- **Core principle:** fix the RV failure and the LVOTO often improves.

Priority	Preferred approach
Treat the cause	Address PE, pulmonary hypertension/RV-PA uncoupling, or post-cardiotomy RV failure.
Support perfusion pressure	Target a higher MAP to support RV coronary perfusion and provide LV afterload.
Choose vasopressors carefully	Vasopressin is useful because it raises SVR without increasing PVR. Norepinephrine may help if mild beta-1 support is needed for RV contractility. Avoid phenylephrine when RV failure is dominant because it may increase PVR.
Reduce RV afterload	Correct hypoxemia, hypercapnia, and acidemia. Consider inhaled pulmonary vasodilators such as epoprostenol. Nitric oxide may not be available; nebulized nitroglycerin can be considered if used locally.
Optimize RV volume	Avoid reflexive large-volume loading. Diuresis may help if the RV is dilated and beyond its optimal Frank-Starling range.
Escalate when needed	In severe RV failure with low contractility, consider low-dose epinephrine as a last resort with close bedside reassessment. Avoid pure inodilators such as milrinone if SVR reduction worsens LVOTO. Consider early ECMO referral or initiation if pharmacologic support fails or worsens LVOTO.

Post-cardiotomy and valve surgery considerations

After SAVR or TAVR for aortic stenosis

Before valve replacement, fixed aortic stenosis can “splint” the LV and prevent dynamic obstruction. After AVR, sudden afterload reduction and a shorter ejection time can unmask DLVOTO or a mid-ventricular gradient.

- Triggers: sudden loss of fixed afterload, vasoplegia/SIR after cardiopulmonary bypass, anesthetic vasodilation, hypovolemia/bleeding/polyuria/rewarming, and catecholamine exposure during separation from bypass.
- Management generally follows the primary DLVOTO approach: restore preload, increase afterload, reduce catecholamines/inotropy when feasible, and control tachycardia.

After mitral valve repair or replacement

SAM and dynamic LVOTO can occur after mitral valve repair and is also described after mitral valve replacement, particularly when the anterior leaflet or subvalvular apparatus is preserved.

Risk factors that promote SAM after mitral surgery
Redundant anterior and/or posterior mitral leaflets
Anterior displacement of the mitral valve or coaptation point
Unfavorable aorto-mitral angle <130 degrees
Papillary muscle displacement or abnormal chordae
Asymmetric septal hypertrophy
Undersized mitral annulus or annuloplasty ring
High-profile or malpositioned prosthetic valve

Bedside treatment algorithm

1. Recognize the pattern: shock + hyperdynamic LV + LVOT aliasing/SAM ± posterior MR.
2. Confirm location and severity with PWD/CWD and calculate the gradient.
3. Evaluate the RV before committing to treatment strategy.
2. If primary DLVOTO: stop inotropes/diuretics, cautiously increase preload, increase afterload with phenylephrine and or vasopressin, slow heart rate, and restore sinus rhythm.
3. If RV-driven DLVOTO: treat RV failure, support MAP, reduce RV afterload, avoid excessive fluids and beta-blockade, and escalate early if RV failure persists.
4. Reassess frequently with bedside echo and hemodynamics after each intervention.