POST CARDIOTOMY SHOCK ASSESSMENT

BACKGROUND

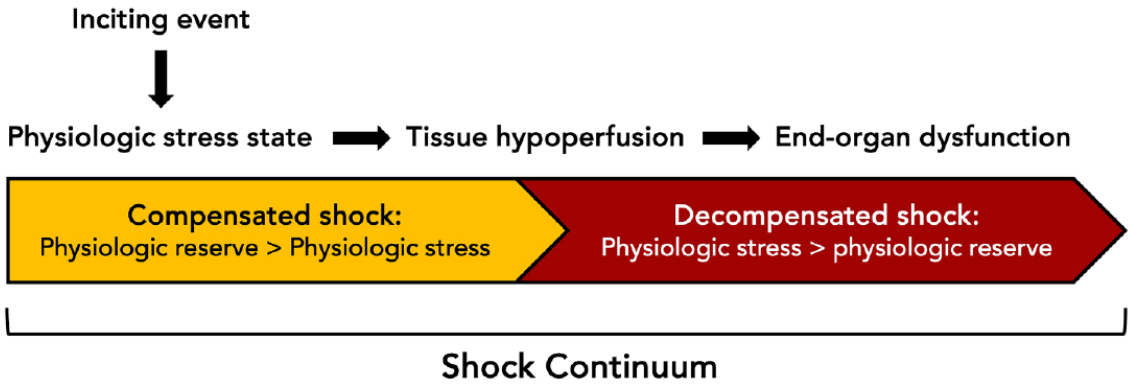
The combination of a non-pulsatile low flow state, cross clamping of aorta, activation of a systemic inflammatory response (SIR), coagulopathy, need for blood products transfusion, and anesthesia along with potential surgical technical issues, predispose patients to multiple pathophysiological abnormalities and higher risks for cardiac and multiorgan complications including shock and multiorgan failure.

Normal Hemodynamic Variables

Hemodynamic Variable	Formula/ Normal Range
MAP (Mean Arterial Pressure)	70 – 100 mmHg
mPAP (Mean Pulmonary Artery Pressure)	10 – 20 mmHg
CVP (Central Venous Pressure)	2 – 8 mmHg
PCWP (Pulmonary Capillary Wedge Pressure)	4 – 12 mmHg
SV (Stroke Volume)	1 ml/kg/beat
SVI (Stroke Volume Index)	1 ml/kg/beat/m^2
CO (Cardiac Output)	SV × HR = 4 – 8 L/min
CI (Cardiac Index)	SVI × HR = 2.5 – 4 L/min/m^2
SVR (Systemic Vascular Resistance)	(MAP − CVP) / CO = 800 – 1200 dyn·s/cm^5 or 10 – 15 WU
PVR (Pulmonary Vascular Resistance)	(mPAP − PCWP) / CO = 100 – 250 dyn·s/cm^{-5} or 1 – 2 WU
CPO (Cardiac Power Output)	(MAP × CO) / 451 = >1 W
CPI (Cardiac Power Index)	(MAP × CI) / 451 = >0.4 W/m^2
PAPI (Pulmonary Artery Pulsatility Index)	(sPAP − dPAP) / CVP = >2 mmHg
RVSWI (Right Ventricular Stroke Work Index)	(mPAP − CVP) × SVI = 0.5 – 0.6 mmHg/L/m^2

DEFINITION OF SHOCK

- **Physiological:** Relative or absolute decreased tissue perfusion resulting in organ dysfunction
- **Clinical:** Hypotension defined as MAP <65 mmHg or SBP <90 mmHg sustained for 15 min



ASSESSMENT OF CARDIOGENIC SHOCK

- Integration of clinical assessment with hemodynamics and ultrasound is essential (no single modality is sufficient in isolation).
- The initial therapeutic strategy should be based on the identification of one of the four Forrester-Kenny phenotypes and assessing the pressures interfaces.
    - Several decades ago, based on cardiac index on one axis and pulmonary edema on the other, cardiogenic shock was classified by **Forrester** in four phenotypes (see figure 1):

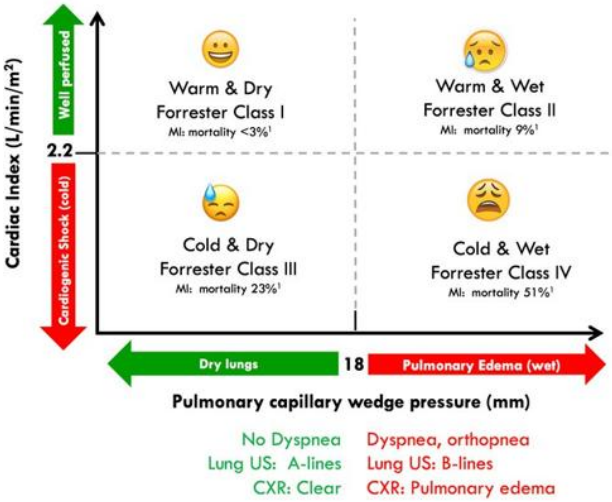


Figure 1. Forrester diagram showing the four phenotypic quadrants

- More recently, **Kenny** reworked the Forrester concept, utilizing stroke volume (SV) or surrogates on one axis and VEXUS for evaluation of venous congestion on the other, for the initial assessment of shock, and subsequently to track the progress during resuscitation using varying parameters of forward flow vs. congestion, as shown in figure 2.

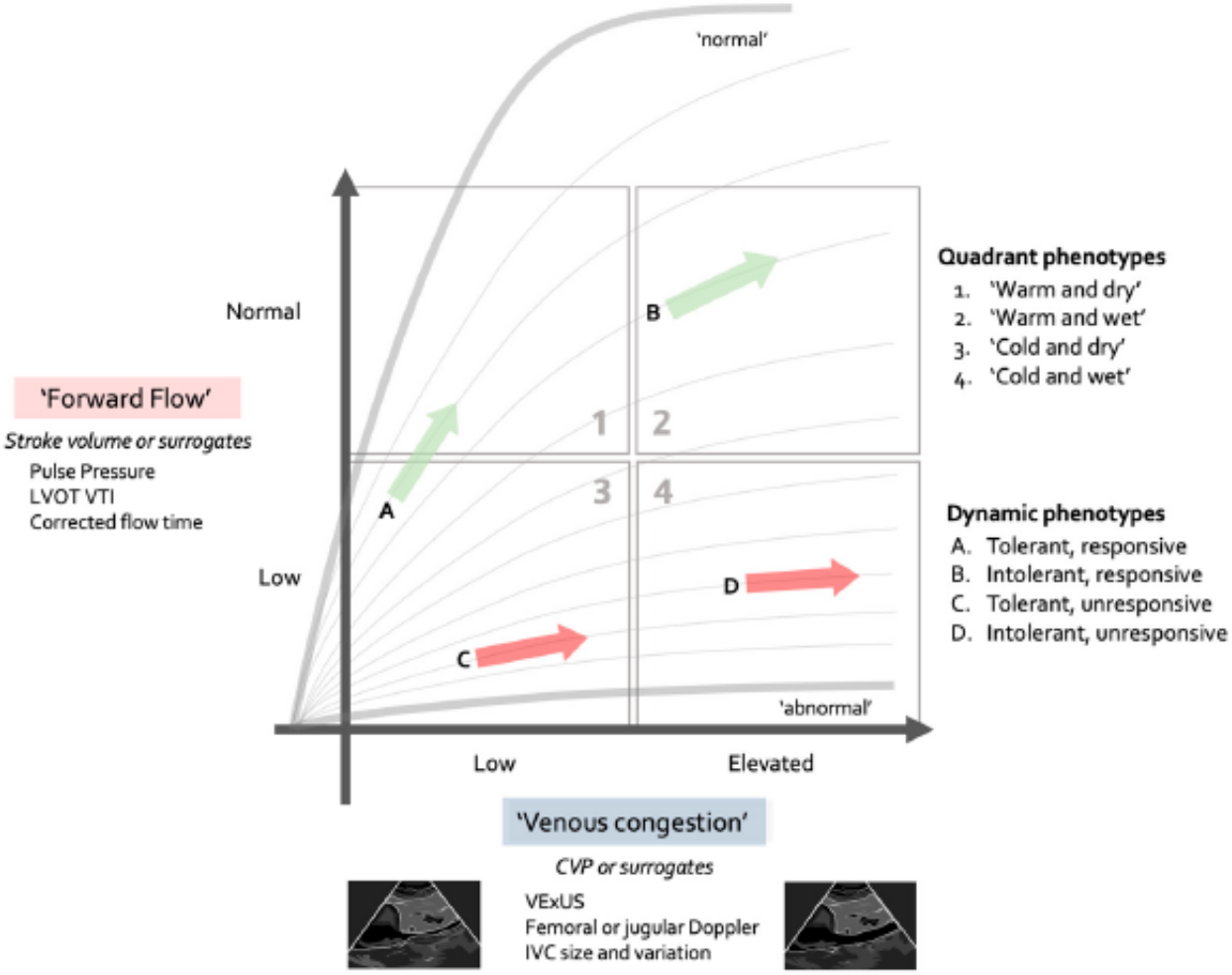


Figure 2. Forrester–Kenny diagram showing the four phenotypic quadrants as well as dynamic. From J. Pers. Med. 2025, 15, 207. https://doi.org/10.3390/jpm15050207

Forward flow (SV) assessment

- In the absence of PA catheter, SV can be estimated via LVOT cross-sectional area × velocity-time integral (LVOT-VTI), measured from the apical 5-chamber view using pulsed-wave Doppler ~5 mm proximal to the aortic valve (https://pubmed.ncbi.nlm.nih.gov/34710882).
    - *Normal LVOT VTI* in healthy adults is approximately **22 ± 4 cm**.

- VTI <18 cm is generally considered low and associated with reduced forward flow.
- It is recommended tracking VTI alone for serial measurements, as LVOT diameter remains constant and its measurement introduces additional error.

Clinical and ultrasonographic assessment of venous congestion

- Physical examination, fluid balance, elevated proBNP and the presence of vascular congestion on CXR are useful tests to identify patients with volume overload but have several limitations.

- Ultrasonographic findings of presence of pulmonary B-lines, IVC collapsibility and internal jugular or femoral venous pressure may perform better than clinical assessment but have also several limitations (see figure 3).

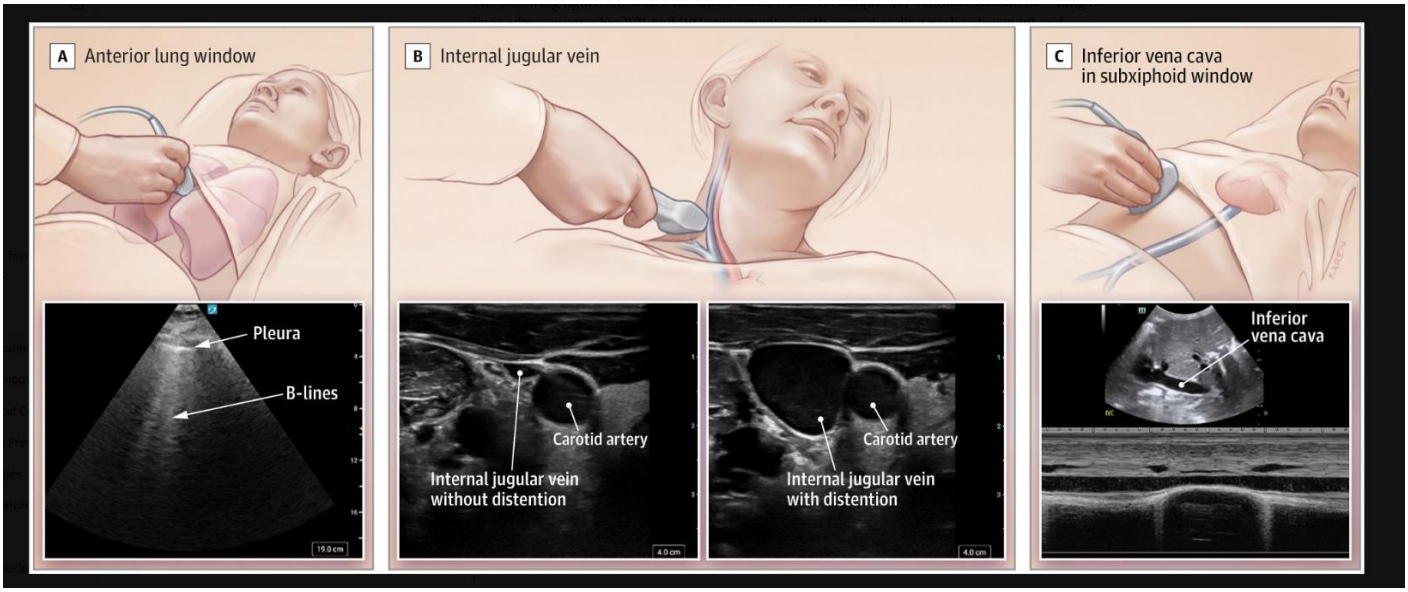


Figure 3. Ultrasonographic findings of pulmonary B-lines, IVC collapsibility and IJ or femoral venous pressure.

- VExUS (Venous Excess Ultrasound Score)
  - It was developed by Beaubien-Souligny et al in 2020 to quantify systemic venous congestion by combining IVC diameter with pulsed-wave Doppler of three venous beds. https://pubmed.ncbi.nlm.nih.gov/32270297
  - It reflects the interaction between venous return and cardiac function, **not pure volume status**.

Components and Grading

The scan involves four assessments: (1) IVC diameter (gatekeeper), (2) hepatic vein Doppler, (3) portal vein Doppler, and (4) intrarenal vein Doppler. Each Doppler component is classified as normal, mildly abnormal, or severely abnormal:

VExUS Grade	Criteria	Interpretation
0	IVC < 2 cm (regardless of Doppler)	No congestion
1	IVC ≥ 2 cm + no severely abnormal Doppler	Mild congestion
2	IVC ≥ 2 cm + **1** severely abnormal Doppler	Moderate congestion
3	IVC ≥ 2 cm + **≥ 2** severely abnormal Doppler	Severe congestion

Severely abnormal patterns include:

- Hepatic vein systolic flow reversal
- Portal vein pulsatility ≥50%
- Monophasic diastolic intrarenal venous flow.

The following figure shows representative Doppler waveforms for each component across the normal-to-severe spectrum:

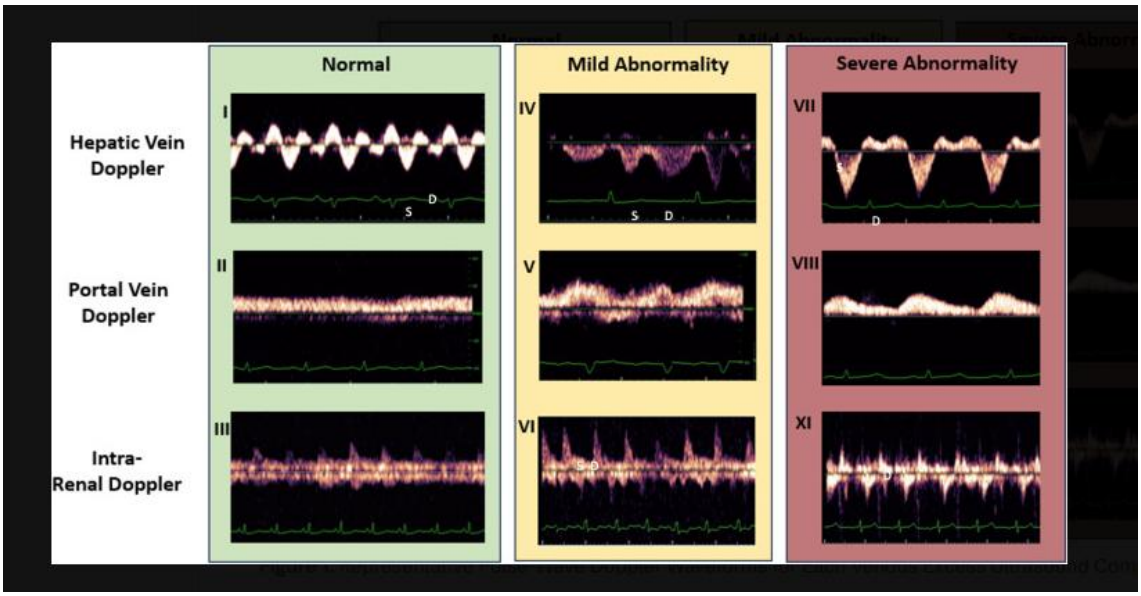
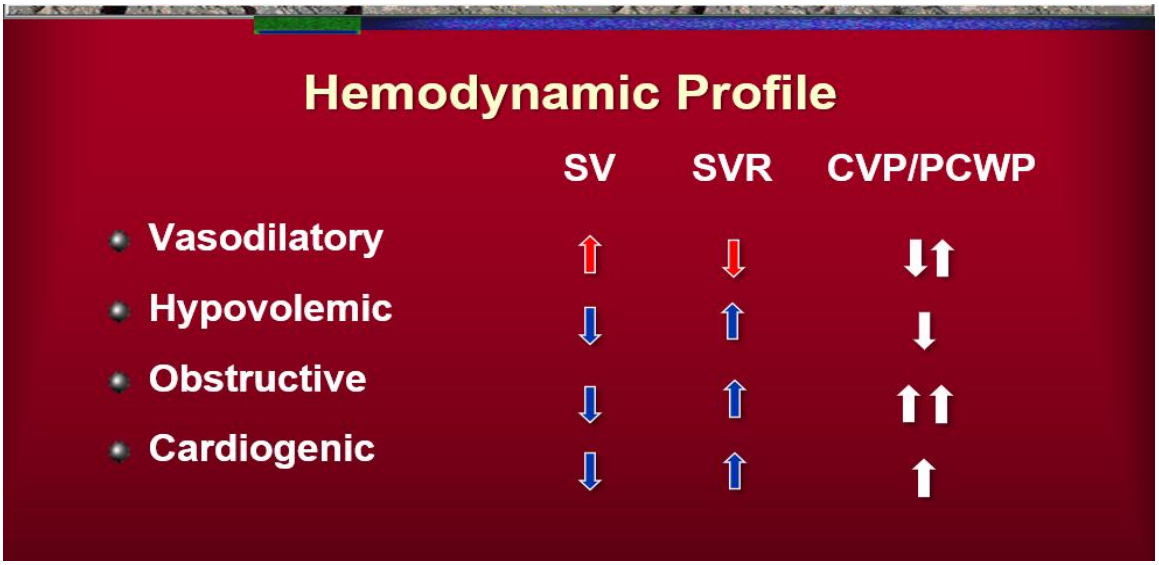


Figure 4. Doppler waveforms for each of the three venous beds. From JACC Adv, January 2026.

ETIOLOGY OF POST CARDIOTOMY SHOCK

Typical hemodynamic patterns in common shock states



- *Vasodilatory*
  - Early: due to well-established post cardiotomy SIR
  - Late: Usually, sepsis related
- *Hypovolemic*
  - Usually due to severe bleeding but also due to decreased effective intravascular volume
- *Obstructive*
  - Acute pulmonary embolism
  - Cardiac tamponade
  - Tension pneumothorax
- *Cardiogenic*
  - Acute myocardial infarct (type 5 MI)
    - Graft or non-graft related (see figure 1 below)
  - Acute decompensated heart failure (ADHF) in patients with underlying cardiomyopathies
  - Arrhythmias
  - Dynamic LVOT obstruction
    - Post AVR for AS due to sudden loss of afterload in a small, hypertrophied LV
    - Post MV repair; it cal alco occur MVR
      - After MV repair, approximately 5% of cases may be complicated by SAM and dynamic LVOTO

        - It is also well described after MVR, particularly when the anterior mitral leaflet or subvalvular apparatus is preserved
    - 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.
        - It can be identified by presence of SAM on M-mode and mosaic of colors suggesting high-velocity flow on continuous flow Doppler
        - It is confirmed and the severity can be assessed by estimating the LVOT gradient by using combination of pulse and continuous flow Doppler
        - Physiologic triggers
            - Decreased afterload
            - Sudden loss of fixed afterload with repair of the AS
            - SIR/vasoplegia post CPB
            - Vasodilating effect of sedating medications such as propofol/anesthetics
            - Decreased preload
            - Hypovolemia/bleeding/polyuria/SIR post CPB and rewarming
            - Increased inotropy
            - Exogenous catecholamines used commonly in waning from CPB
- *Mixed*
    - Vasodilatory due to post cardiotomy SIR or concomitant sepsis and primary cardiac including myocardial stunning
    - Neurogenic due to spinal cord infarction due to ischemia or embolic phenomena
        - Bradycardia and warm skin (can be seen below the level of spinal cord lesion)

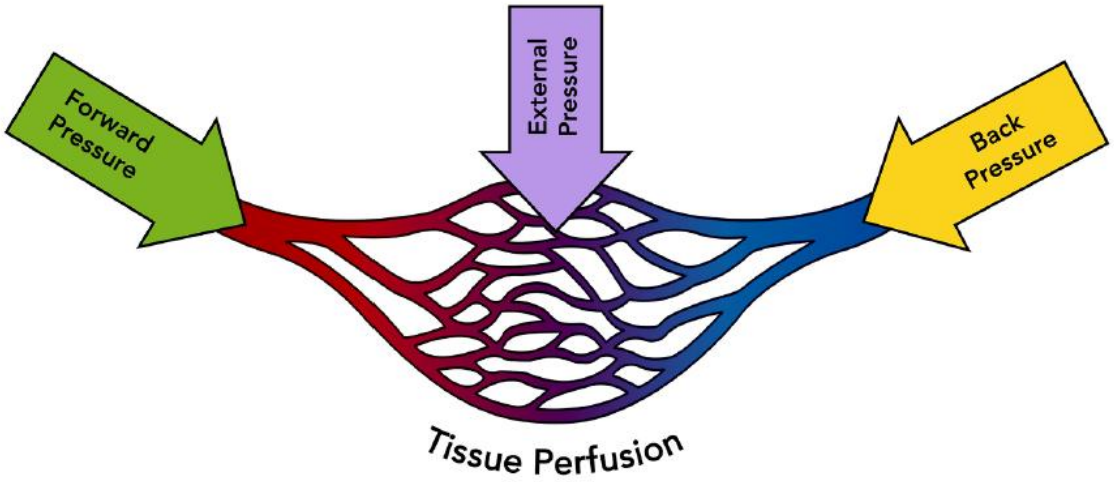
Reframing conceptualization of shock

Proposed by Sara Crager https://doi.org/10.1016/j.emc.2025.09.003) and Phillipe Rola et al https://doi.org/10.3390/jpm15050207 . Summarized in EMCrit by Scott Weingart.

Shock is a dynamic interplay between physiologic stress and physiologic reserve.

- It is essential to recognize that compensated shock represents a physiologic stress state wherein sympathetic drive often maintains normotension, or even hypertension, which can mask underlying hemodynamic instability.

- Progression to decompensated shock is not necessarily associated with the development of hypotension, and that a patient may have severe tissue hypoperfusion and evolving end-organ dysfunction while maintaining a normal MAP.
    - Tissue perfusion requires adequate forward flow across capillaries to maintain adequate organ function.
    - Forward flow may be conceptualized as the net sum of competing forward, backward, and external pressures.



From Emerg Med Clin N Am 44 (2026) 315–331
https://doi.org/10.1016/j.emc.2025.09.003

Rather than considering macrohemodynamic variables in isolation, forward, backward, and external pressures are organized around pressure interfaces, with the net vector sum at each interface determining the resultant forward flow. Significant impairment at any one of these interfaces can precipitate a shock state.

- This framework requires a shift away from a narrow focus on forward pressures (BP, LV systolic function, and intravascular volume status) as the central organizing principles of shock evaluation and management.

PRESSURE INTERFACES

Interface 1: *LV to systemic arterial resistance coupling Interface*

- Forward pressure: LV CO.
- Forward and back pressure: SVR.
    - Assess SV, cardiac power output (CPO), LVEF and SVR

Interface 2: Arteriolar to capillary (macro to arterial microcirculation)

- Forward pressure: MAP and DBP (for the coronaries).
- Backward pressure: Arteriolar tone and external pressure (peripheral tissue edema causes external pressure on capillaries leading to collapse).
    - Assess skin perfusion (capillary refill time-CRT, mottling), mixed SvO2 and mixed venous to arterial PCO2 gap.

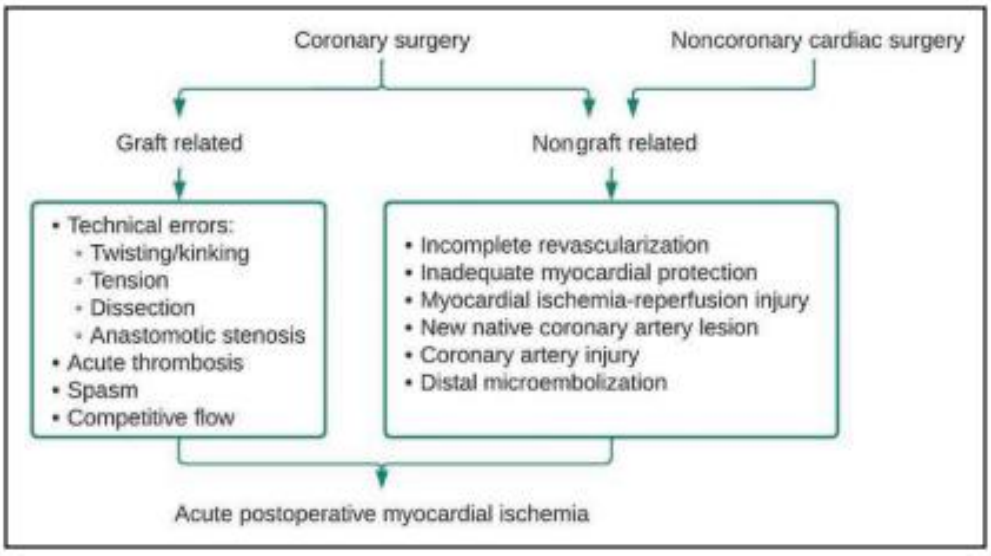
Interface 3: Distal Capillary to venular microcirculation

- Forward pressure: Mean systemic microcirculatory pressure
- Back pressure: CVP
    - Assess CVP, venous congestion (IJ, femoral and IVC collapsability and VExUS)

Interface 4: Right ventricle to pulmonary artery (RV to PA)

- Assess RV size, RV to LV interdependence, TAPSE and TR max vel by echocardiogram and pulmonary artery pulsatility index (PAPI) by PA catheter.

Etiology of postoperative acute myocardial ischemia



From Bernhardt AM, et al. https://doi.org/10.1016/j.healun.2022.10.028

Definition of CABG-related type 5 MI

- Evidence of new myocardial ischemia/new loss of myocardial viability within ≤48 h after the procedure determined by:
    - Elevation of troponin >10 times in patients with normal baseline or 20% variation in patients with elevated pre-procedure values

    - Ischemic ECG, echocardiographic, or angiographic findings
- Caveats
    - There is no consensus on the troponin cutoff points that clearly differentiate cardiac procedural myocardial ischemia from myocardial infarction
        - The Academic Research Consortium-2 expert consensus document, type 5 MI was defined as increases in cTn >35 times the URL with new evidence of ischemia or 70 times the URL as a standalone criterion
    - Interpretation of the ECG is limited by several factors
        - ST-segment and T-wave changes may be less specific, whereas ST-segment elevation with reciprocal changes is a more reliable indicator
        - Postoperative paced rhythm or pericarditis
    - Echocardiogram alone is nondiagnostic in the postoperative setting
        - It can rule out non-ischemic causes of chest pain, aortic dissection, and mechanical complications of myocardial infarction
        - Intraoperative regional wall motion abnormalities on transesophageal echocardiography do not predict graft failure after CABG

- **SCAI shock classification**
    - The Society for Cardiovascular Angiography and Intervention (SCAI) developed in 2019 a staging system for risk stratification that predicts mortality. It was endorsed by the AHA, SCCM, ACC and STS.

        - **Stage A (“at risk”):** Hemodynamically stable but have acute MI or acute on chronic heart failure putting them at risk of developing C
        - **Stage B (“beginning”):** Hemodynamic instability, including hypotension and/or tachycardia, but with normal perfusion
        - **Stage C (“classic”):** Hypoperfusion (hyperlactatemia, oliguria, cool/clammy skin, or altered mentation) that requires intervention (inotrope, vasopressor or MCS) to restore perfusion
        - **Stage D (“deteriorating”):** Hemodynamic instability and/or hypoperfusion fail to respond to initial interventions
        - **Stage E (“extremis”):** Overt or impending circulatory collapse including hypotension despite maximal support or cardiac arrest with ongoing resuscitation