Low Cardiac Output Syndrome

ASHLEE FERNANDEZ, BSN, RN, CCRN
ANDREA TORZONE MSN, CPNP, PCNS

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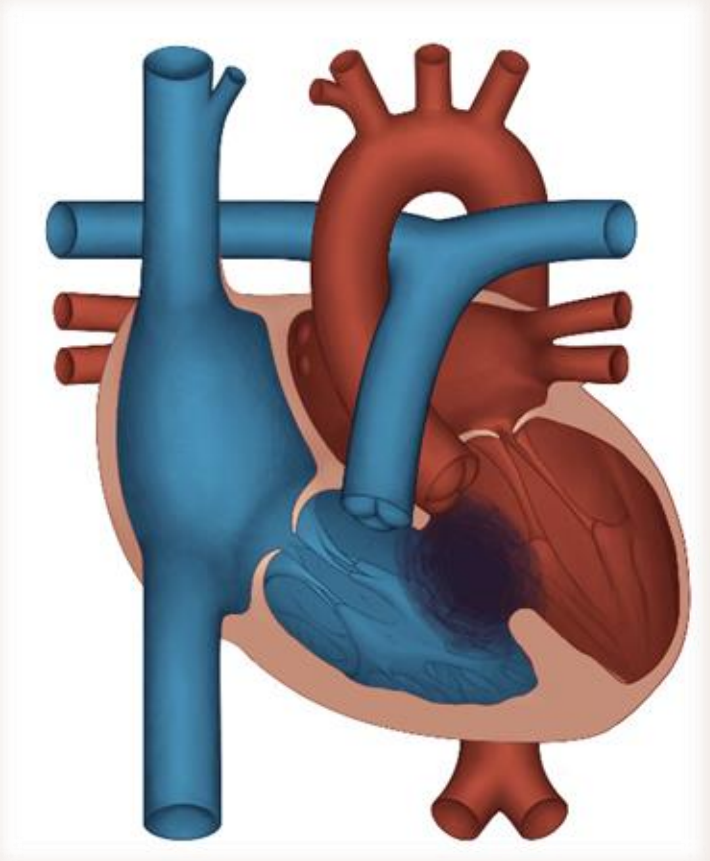
children's health | UT Southwestern Medical Center

Clinical situation

- 10-year-old with VSD and aortic insufficiency due to aortic valve prolapse
- Fatigue and poor exercise tolerance
- Pre-op echocardiogram:
  - large perimembranous VSD
  - severe AI
  - dilated LV



Clinical situation contniued

- Immediately s/p VSD closure and aortic valve repair
- Post-op echocardiogram: good function, no residual VSD, mild AI
- VS:
  - HR 120
  - CVP 9
  - BP 87/58
  - Intubated; Spo2 98%
  - Temp 35.5 C
- Arterial line, CVL in RIJ, Chest tubes
- Adrenaline 0.03 mcg/kg/min

Clinical Assessment: what is important?

- HR & rhythm
- Pulses
- Cap refill
- Oxygenation
- Urine output
- Chest tube output

Clinical situation: 4 hours later

- HR: 140 bpm
- BP: 70/40 mmHg
- Urine output: 0.3 mL/kg/hr
- Extremities cool
- Adrenaline 0.04 mcg/kg/min

What is the most likely diagnosis?

A. Hypovolemic shock
B. Low cardiac output syndrome
C. Sepsis
D. Pulmonary embolism

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Low cardiac output: what is it?

Imbalance between O2 delivery and consumption!

Decrease in systemic perfusion

Clinical presentation:

Low cardiac output: what is it?

Imbalance between O2 delivery and consumption!

Decrease in systemic perfusion

Clinical presentation:

- poor perfusion: pulses, cap refill, color, temperature
- tachycardia
- elevated RA/CVP/LA pressures (poor ventricular function)
- hypotension
- metabolic acidosis
- Increased lactate, decreased MVO2 (mixed venous oxygen saturation)
- decreased urine output

Which is the earliest sign of worsening perfusion?

A. Low blood pressure
B. Decreased urine output
C. Cool extremities
D. Cardiac arrest

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Why is this patient at risk for this condition after surgery?

A. Increased right-to-left shunting
B. Sudden removal of chronic volume overload
C. Increased pulmonary vascular resistance
D. Excess oxygen delivery

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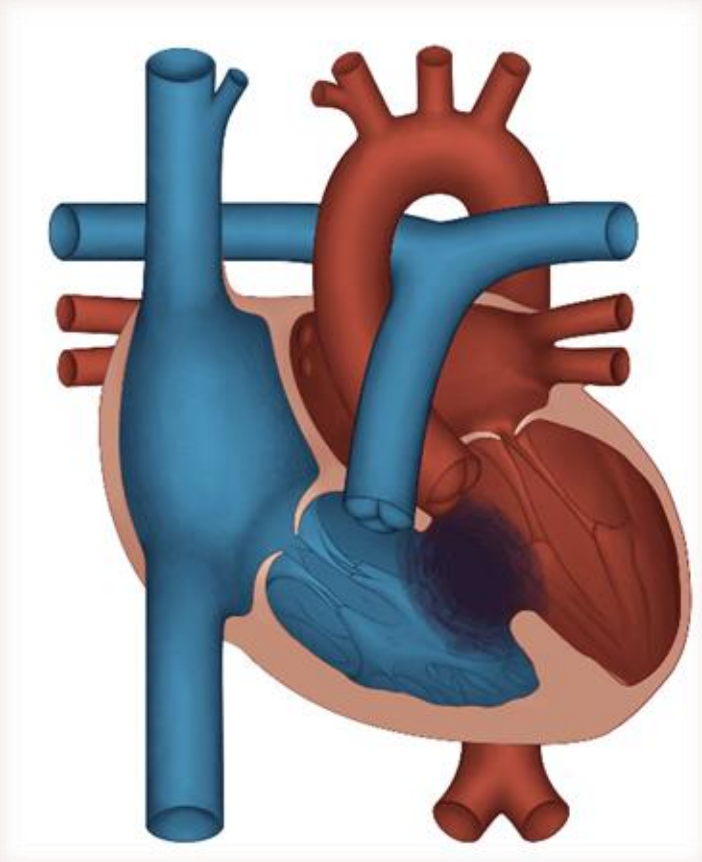


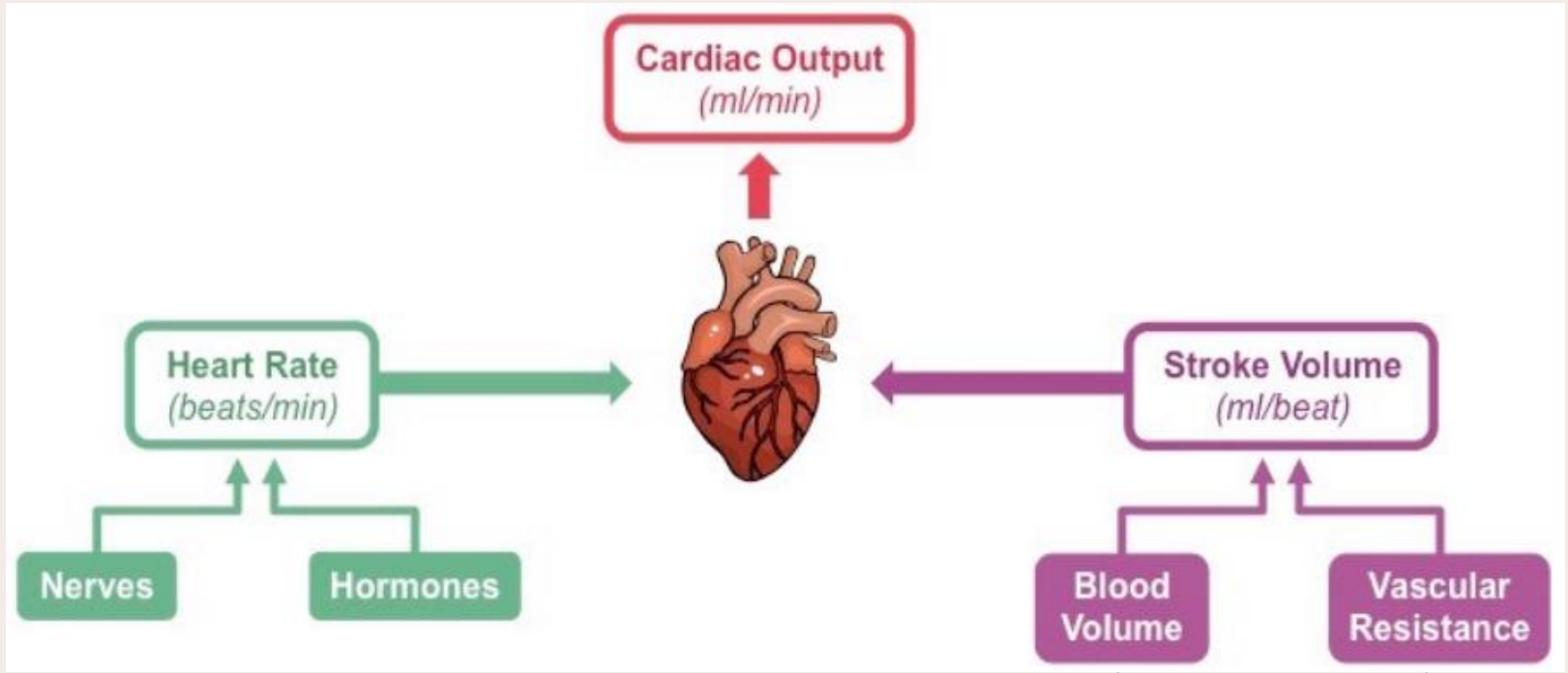
How do we manage LCOS in this patient?

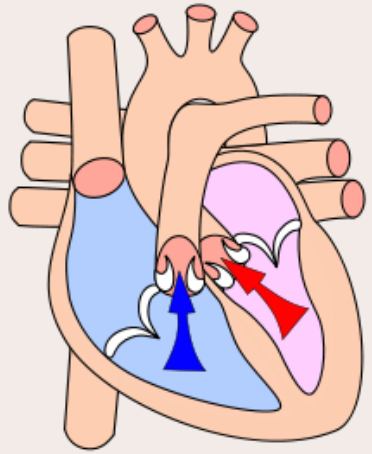
Support the LV:

- Afterload reduction (milrinone)
- Careful volume management
- Optimize inotropic support (adrenaline)

- Don’t forget…
  - Check inotropes and connections

Cardiac output = HR x SV





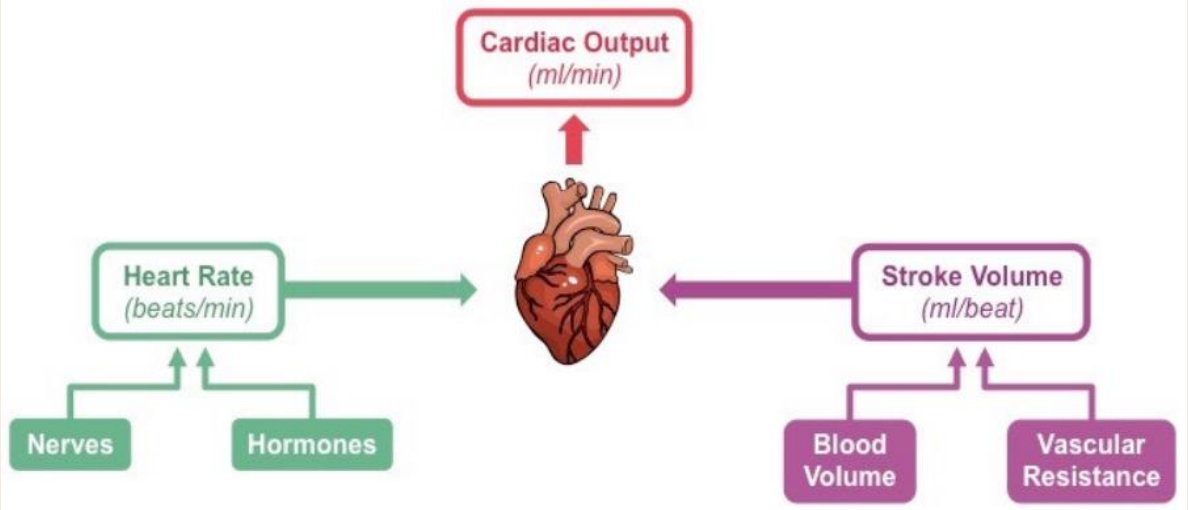
Stroke volume influenced by:

- **Preload**
- **Afterload (there's 2)**
- **Contractility**

Let’s match management to the Cardiac Output equation

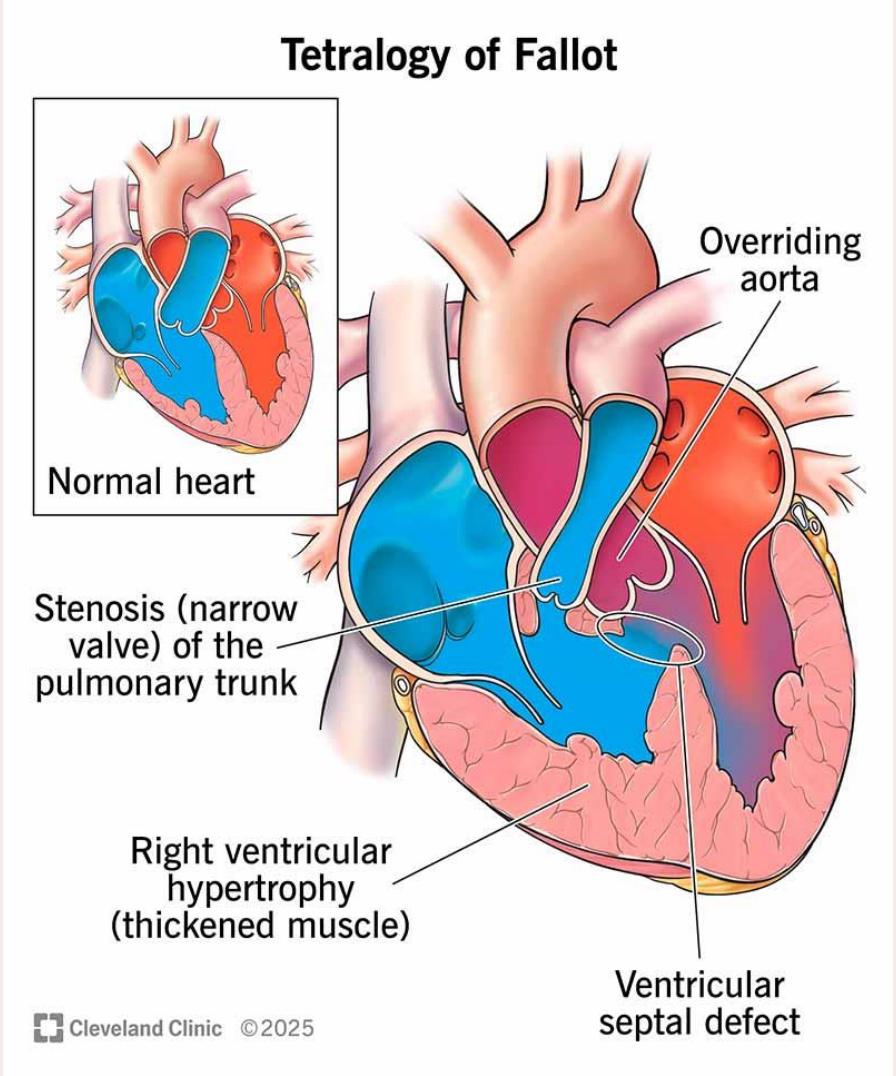
Support the LV:

- Afterload reduction (milrinone) ------ decrease LV **afterload** (SVR)
- Careful volume management ------ optimize **preload**
- Optimize inotropic support (adrenaline) ----- optimize **contractility**



Clinical situation #2:

- 7-year-old with Tetralogy of Fallot/pulmonary stenosis
- Anatomy of ToF
  - VSD
  - Overriding aorta
  - Pulmonary stenosis
  - RV hypertrophy
- Physiology of ToF
- What is important when taking care of a child following repair of ToF?

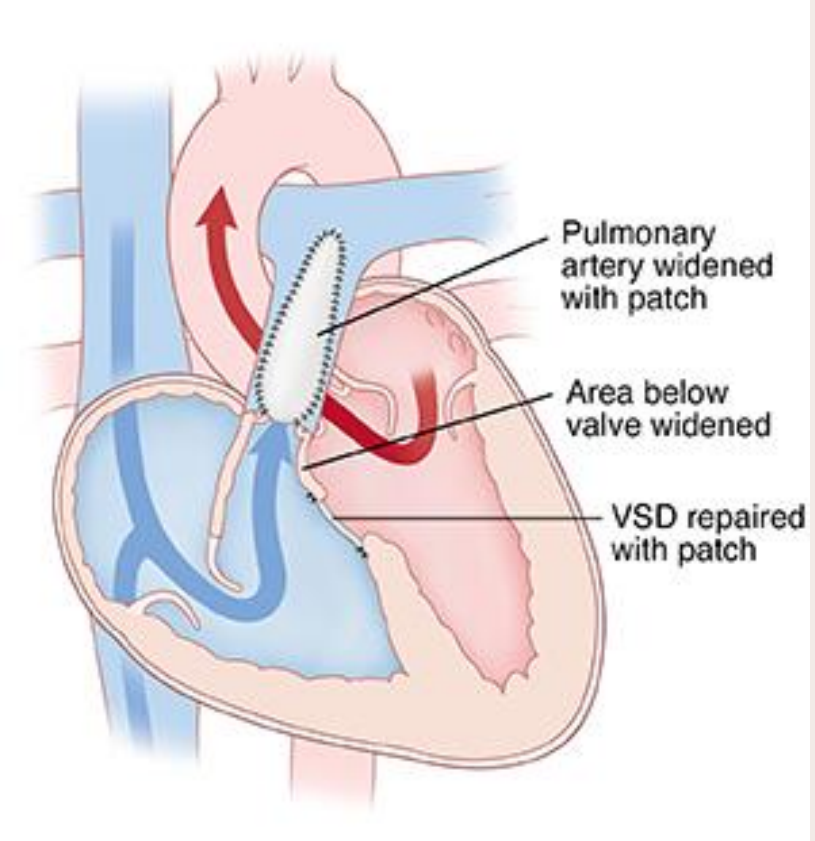


Clinical situation #2: ToF

- Now s/p repair of ToF with transannular patch
- Postop echo: Mildly diminished RV function, free pulmonary valve regurgitation, no residual VSD

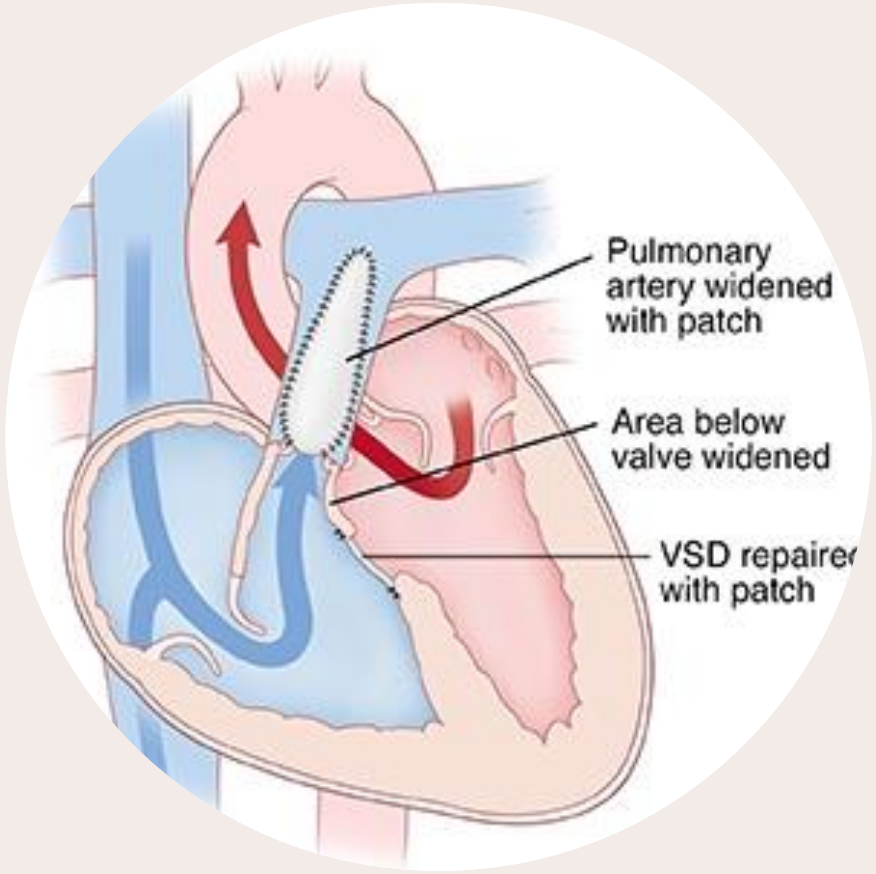
- VS:
  - HR 96
  - BP 105/65
  - CVP 9
  - SpO2 98%
  - Adrenaline 0.04 mcg/kg/min

- 6 hours after arrival the patient has cool extremities and VS are as follows
  - HR 124
  - BP 82/50
  - CVP 15
  - SpO2 95%



Graphic: The StayWell Company

Causes of LCOS after ToF repair

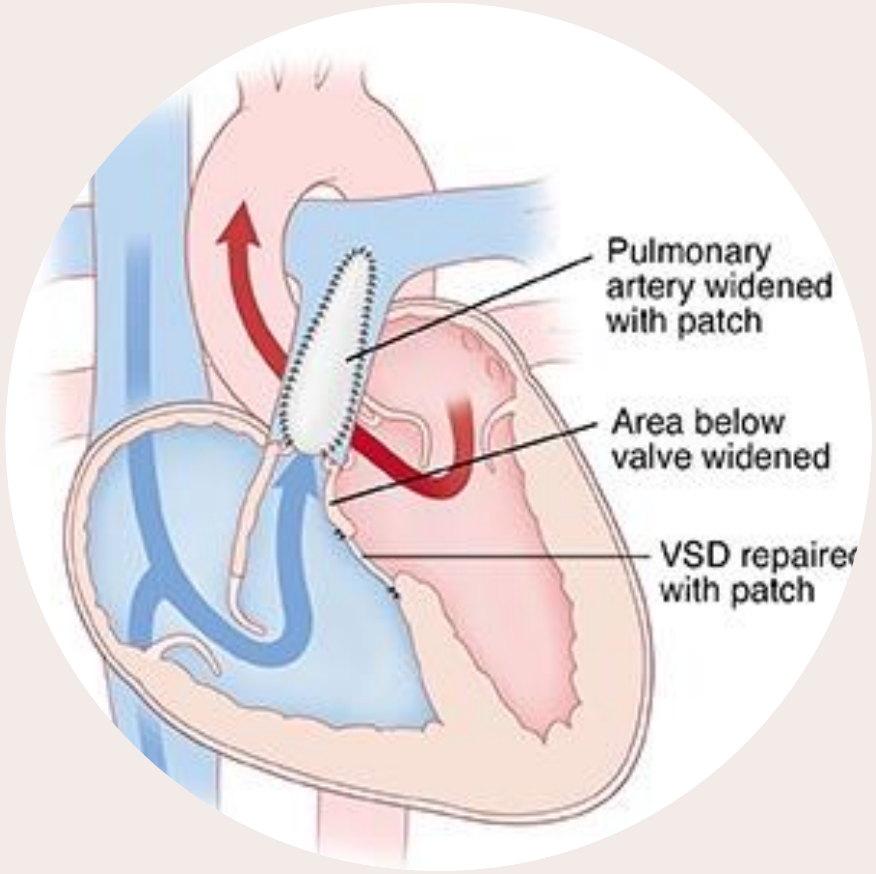


Pre-existing RV hypertrophy
→ Chronic obstruction before surgery leads to a stiff, noncompliant RV

Surgical factors
- Ventriculotomy or patch → myocardial injury
- Cardiopulmonary bypass → global myocardial stunning
- Residual lesions (e.g., RVOT obstruction or pulmonary regurgitation)

Acute postoperative physiology
- Increased pulmonary vascular resistance (PVR)
- Ischemia-reperfusion injury
- Arrhythmia (JET)

Causes of LCOS after ToF repair: it's usually the right side



RV is failing to pump forward (lungs)

Decreased LV preload

Systemic output drops (LCOS)

Increased right atrial pressure: systemic venous congestion

LCOS after ToF repair: what does it look like?

Hemodynamic: rising CVP, hypotension, tachycardia

Perfusion: prolonged capillary refill, cool extremities

Enlarged liver

Elevated jugular venous pressure

Lab: rising lactate, metabolic acidosis

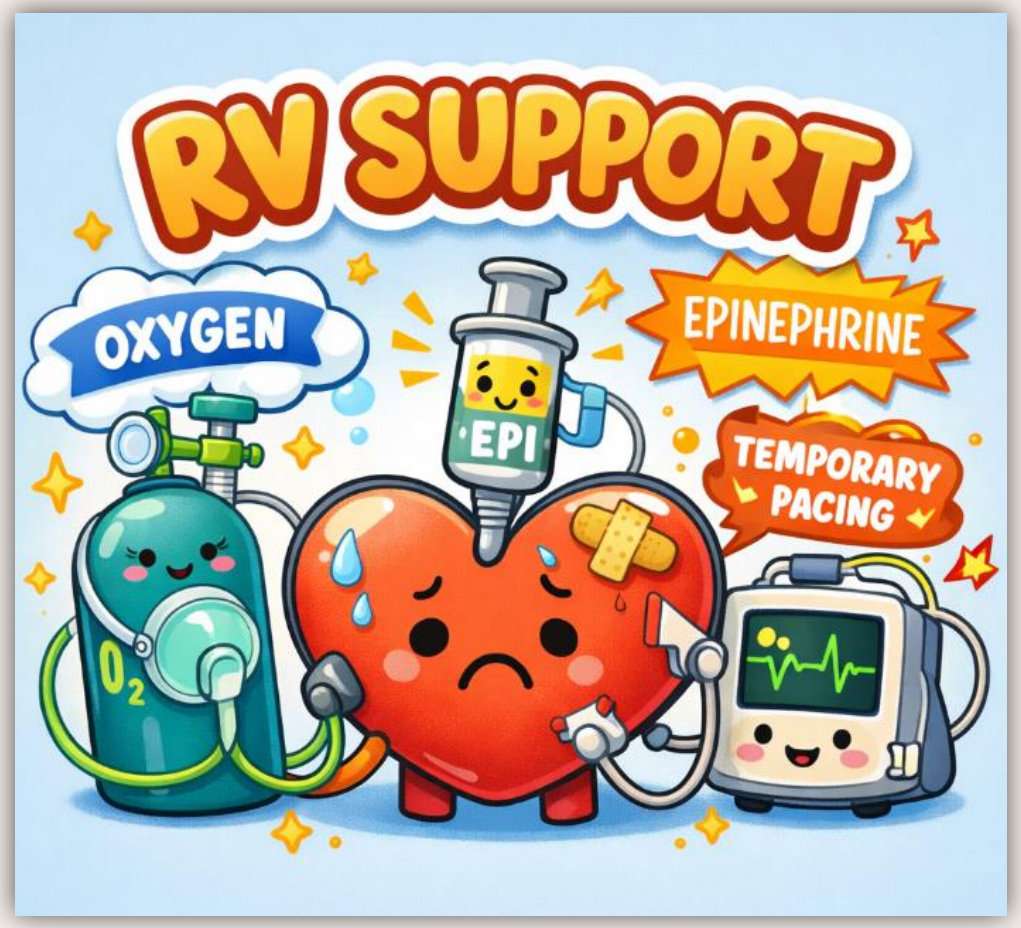
Management of LCOS following ToF repair

Optimize preload
- Avoid both underfilling and overfilling
- Use cautious fluid administration
- Monitor CVP and echocardiography
- Extubate

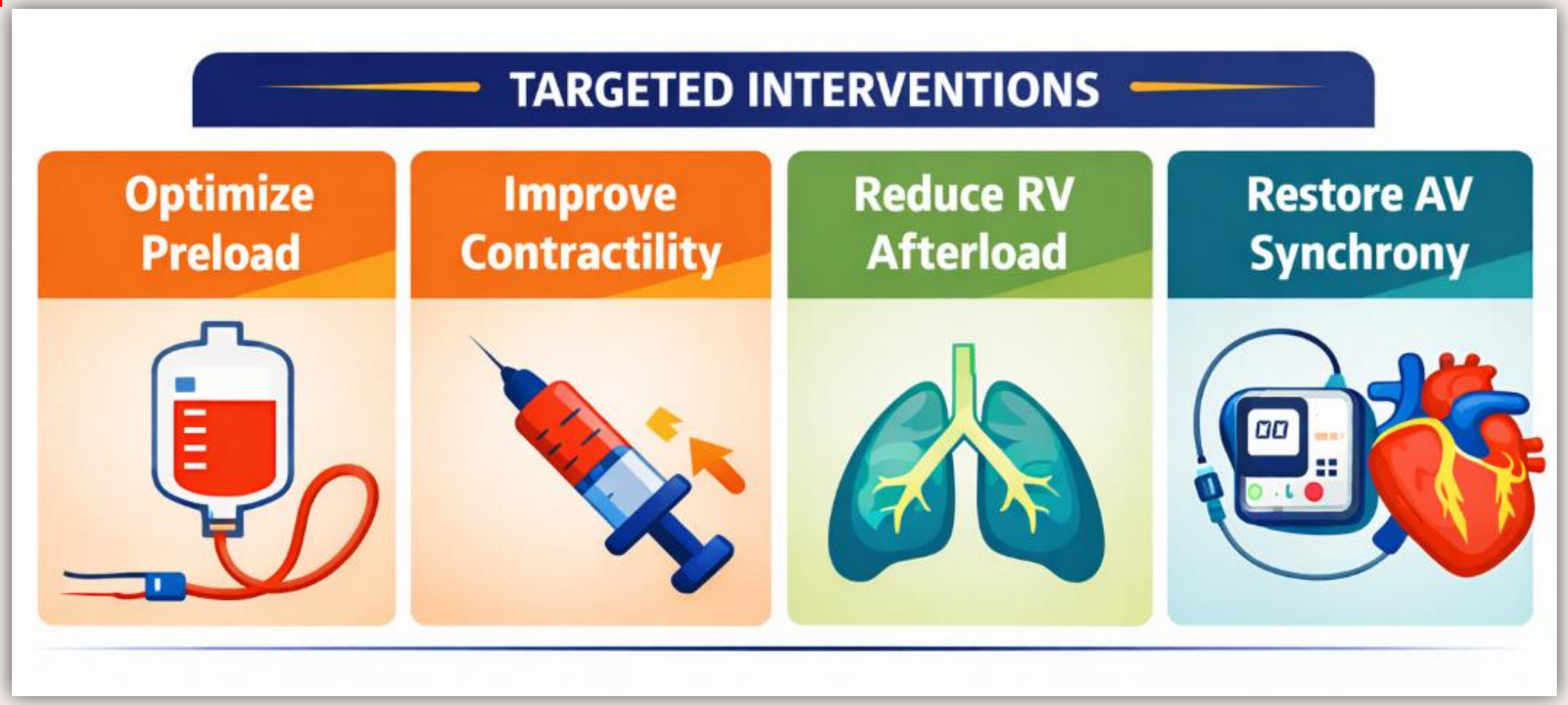
Inotropic support:
- Low-dose adrenaline

Reduce PVR
- Adequate oxygenation & ventilation
- Avoid hypercarbia
- Treat acidosis

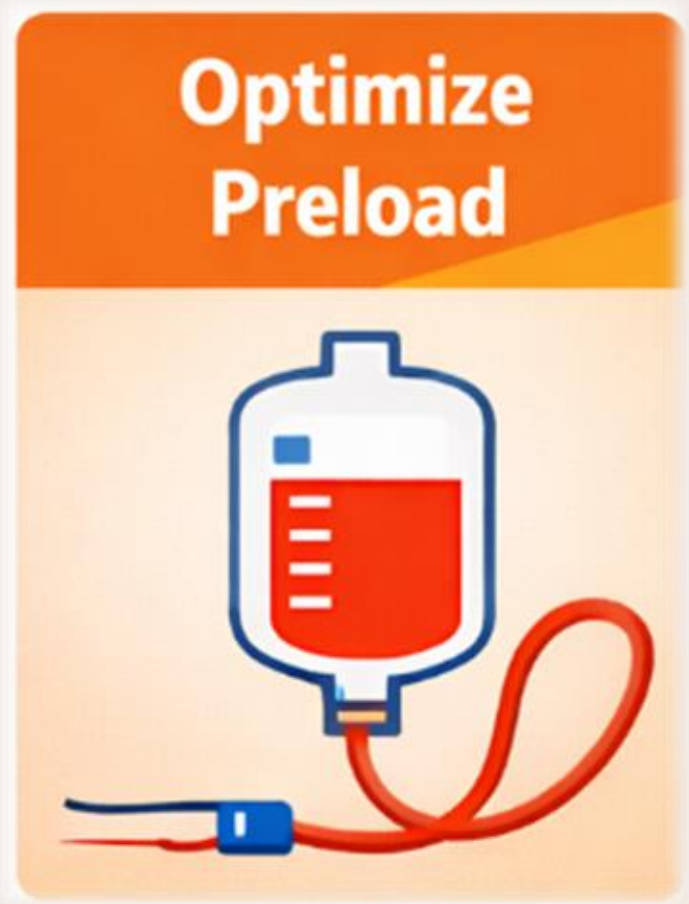
Treat arrhythmias
- JET
- Cooling, sedation, amiodarone
- Pacing to restore AV synchrony



Management of LCOS following ToF repair

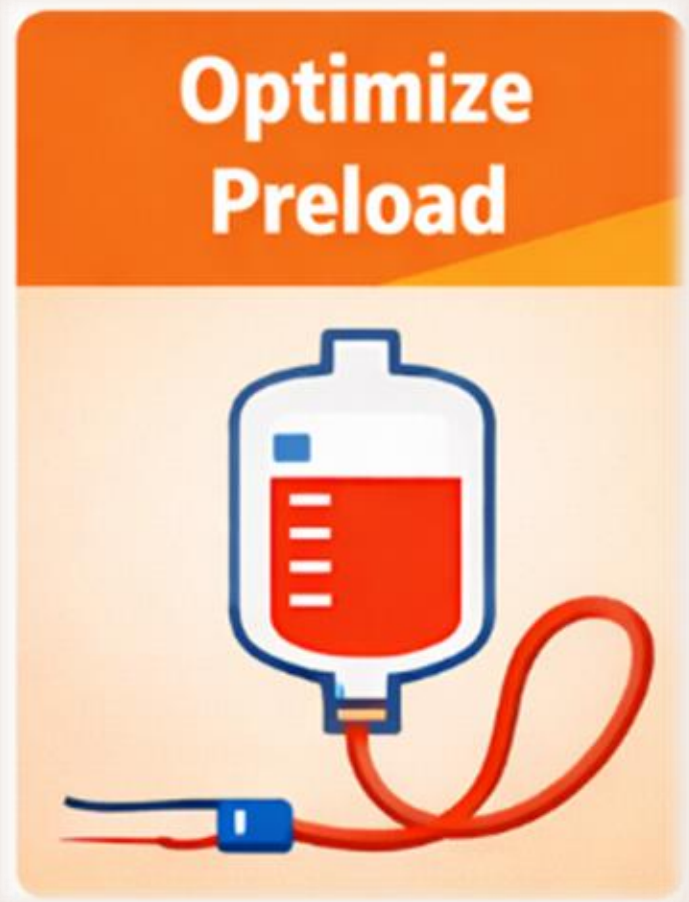


Management of low cardiac output



How do we optimize preload?

Management of low cardiac output



- Monitor CVP
- Use cautious fluid administration
- Avoid underfilling & overfilling

Management of low cardiac output

How do we improve contractility ?

Management of low cardiac output

Inotropes:
Adrenaline (low dose)
Noradrenaline
Dopamine (cautious)
Dobutamine

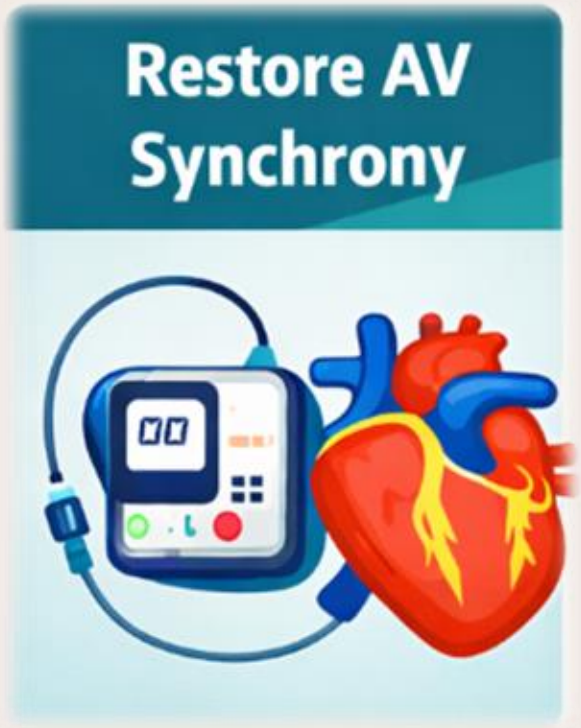
Management of low cardiac output

How do we reduce RV afterload?

Management of low cardiac output

- Ensure adequate oxygenation
- Avoid hypoxia, hypercarbia, and acidosis
- Gentle ventilation if intubated
- Treat pain/agitation (reduces PVR)

Management of low cardiac output



How do we restore AV synchrony?

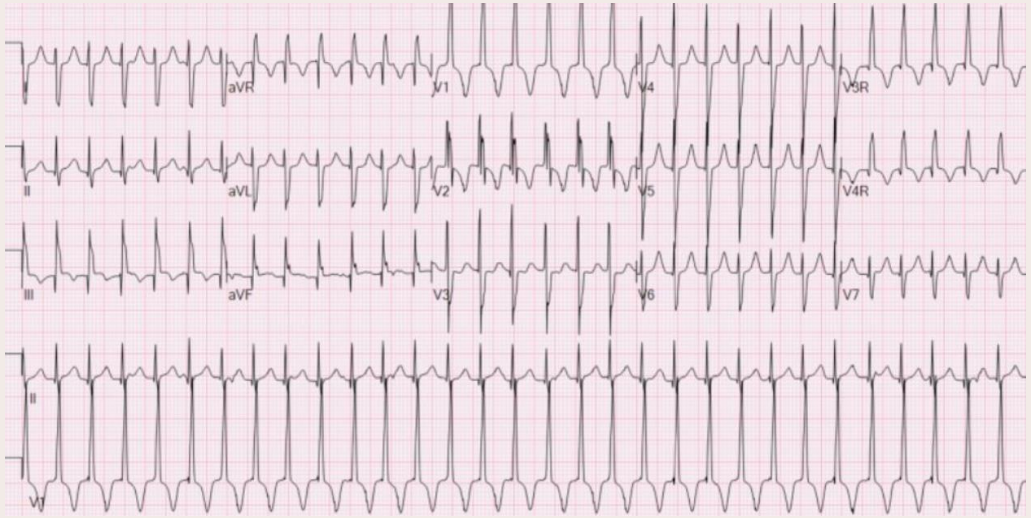
Management of low cardiac output

- Maintain sinus rhythm
- Treat junctional ectopic tachycardia (JET) aggressively

→ AV synchrony is **critical** for RV filling

junctional ectopic tachycardia (JET)

- Typically, transient and occurs within the first 8-48 hours post-operative
- JET: loss of AV synchrony and fast ventricular rate
  - True definition is 30% > resting HR
  - Gradual increase in HR *(important diagnosis)*
    - Regular, narrow QRS complex
    - No P-waves

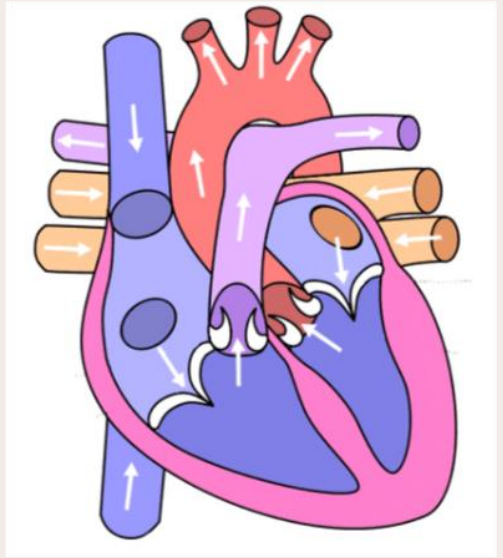


Management

- ↓ inotropes/catecholamines
- Avoid fever / cool
- Sedation +/- neuromuscular blockers
- AAI pacing
- Electrolyte correction
- LCOS management
- Antiarrhythmic medications

Summary:
Low cardiac output: why does it happen?

- Causes:
  - Altered HR or rhythm
  - Decreased preload
  - Increased afterload
    - Increased PVR
  - Systolic dysfunction (poor contractility)
  - Diastolic dysfunction (poor relaxation)
  - Residual surgical lesions



RISK FACTORS

- Preoperative condition – myocardial & end-organ dysfunction
- Complexity of defect
- Younger age < 1 month
- Weight < 2.5kg
- Longer CPB times

Summary:
low cardiac output: what do we do?

- Decrease oxygen consumption:
  - Sedation, neuromuscular blockade
  - Normothermia or cool
- Optimize oxygen delivery
  - Hgb
- Optimize HR & rhythm:
  - Ensure AV synchrony: manage arrhythmia, pace, correct electrolytes
- Optimize preload:
  - Volume
- Decrease afterload:
  - Milrinone
  - Positive pressure ventilation reduces LV afterload
  - Spontaneous ventilation(negative pressure) improves systemic venous return and RV function
- ECHO
  - Systolic dysfunction (decreased contractility)
    - adrenaline, milrinone, correct acid-base balance
  - Diastolic dysfunction (decreased relaxation)
    - milrinone
    - Slow down the HR/avoid tachycardia
- Evaluate for residual surgical lesions
- Re-open sternum

Also try:
Stress dose hydrocortisone

Low cardiac output: key points

Clinical assessment is critical

Recognize trends rather than a single number

Early recognition, notification of team, and intervention