MANAGING MEDICATIONS IN PATIENTS WITHOUT BP

Complications/Follow-Up Destination Therapy Prognosis

YOU MUST UNDERSTAND DISEASE BEING TREATED AND PHYSIOLOGY OF DRUGS BEEN GIVEN

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Overview

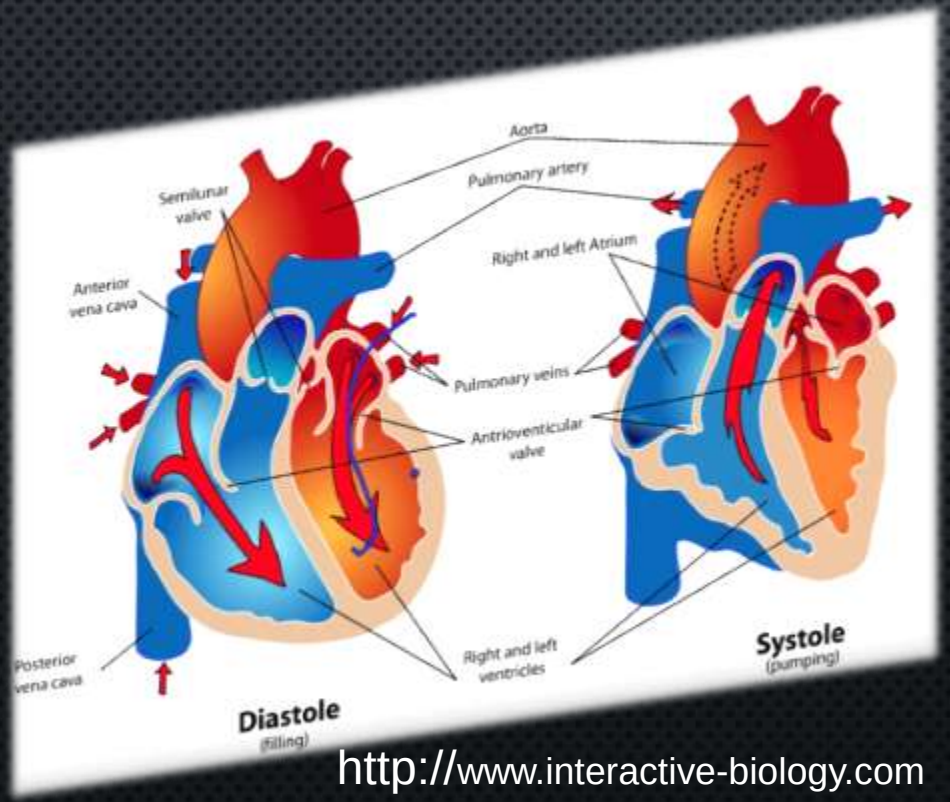
- Physiology of blood pressure-3 slides
- Types of shock-6 slides
  - Mechanisms
- Drugs for shock patient considerations-6 slides
- Destination therapy / complications-4 slides

MAJOR PHYSIOLOGIC DETERMINANTS OF ***TISSUE PERFUSION***

BP = CO X SVR

HR X Stroke Volume (95 cc)

- Preload
- Myocardial contractility
- Afterload



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MAJOR PHYSIOLOGIC DETERMINANTS OF *TISSUE PERFUSION*

BP = CO X SVR

- **Vessel length**
- **Blood viscosity**
- **Vessel diameter (ie, vessel tone)**

HR X Stroke Volume (95 cc)

- Preload
- Myocardial contractility
- Afterload

MIXED VENOUS OXYGEN SATURATION: OXYGEN CONTENT OF BLOOD RETURNING TO HEART AFTER MEETING TISSUE NEEDS

Normal RA blood saturation (mixed venous) 65-70%

Arterial saturation: 95%

Oxygen pulled out into tissue drop in MV02

Normal or high

O2 not removed from RBC (Dissociative shock)(methhemoglobinemia) (Antibotics-nitrates)
IV methylene blue corrects problem

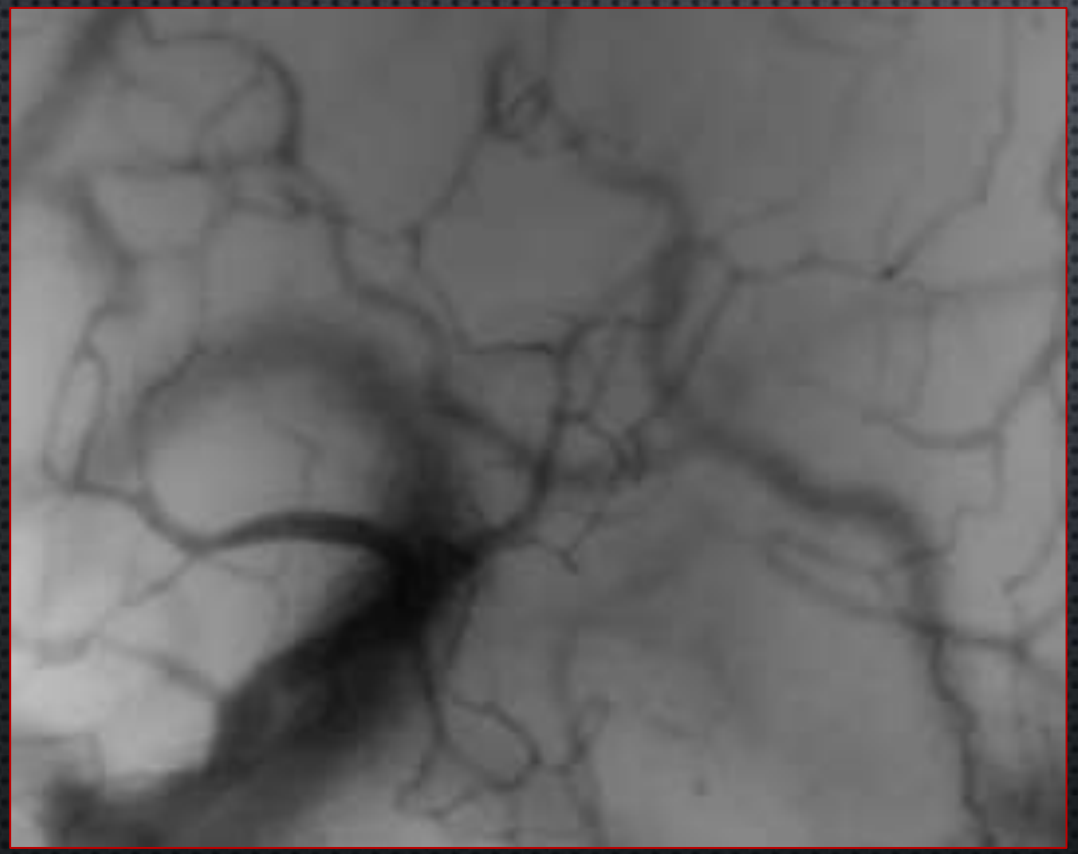
Reduced SV02

Anemia, hypoxemia, or reduced cardiac output during cardiogenic shock

Increased SV02

Reaction to analgesia, sedation, mechanical ventilation, or hypothermia

TISSUE PERFUSION ≠ ? MIXED VENOUS SATURATION



Perfusion= flow / amount of tissue

Blood -improve tissue oxygenation through increasing the oxygen-binding capacity of the blood

Negative effect on the microcirculation (transfused erythrocytes have low deformability, low binding capacity for oxygen –binding to nitric oxide is increased, (vasoconstriction).

Increase in lactate can indicate poor tissue perfusion

SHOCK	Cardiac output	SVR	PCW-LVEDP	Mixed venous 02
Cardiogenic	↓↓↓↓	↑↑↑	↑↑↑	↓↓↓

1. Hypovolemic
2. Cardiogenic
3. Septic shock
4. Dissociative shock
5. Obstructive shock
6. Anaphylactic shock

Different types of shock: focus on cardiogenic
Dissociative-oxygen cannot get off RBC (nitrates)

CASE

52 male emergency room

BP 50/30

CO-3 liter/min

HR 140 sinus tachycardia

Cold extremities

RA pressure 0
RV 20/ 0-2

PA 20/3
PCW 3 mm Hg

Systemic vascular resistance-2300

MV02 45%

Echo-60% EF

1. **Hypovolemic**
2. **Cardiogenic**
3. **Septic shock**
4. **Dissociative shock**
5. **Obstructive shock**
6. **Anaphylactic shock**

MIXED venous saturation=MVO2

CASE

62 female ICU

BP 50/30

CO-7 liters/min

HR 140 sinus tachycardia

Cold extremities

RA pressure 2
RV 30/ 0-2

PA 30/6
PCW 6 mm Hg

Systemic vascular resistance-400

MV02 85%

Echo-60% EF

1. **Hypovolemic**
2. **Cardiogenic**
3. **Septic shock**
4. **Dissociative shock**
5. **Obstructive shock**
6. **Anaphylactic shock**

MIXED venous saturation=MVO2

CASE

62 female ICU

BP 50/30

CO- 2.5 liter/min

HR 140 sinus tachycardia

Cold extremities

RA pressure10
RV 40/ 0-12

PA 40/22
PCW 36 mm Hg

Systemic vascular resistance-1900

MV02 45%

Echo-20% EF

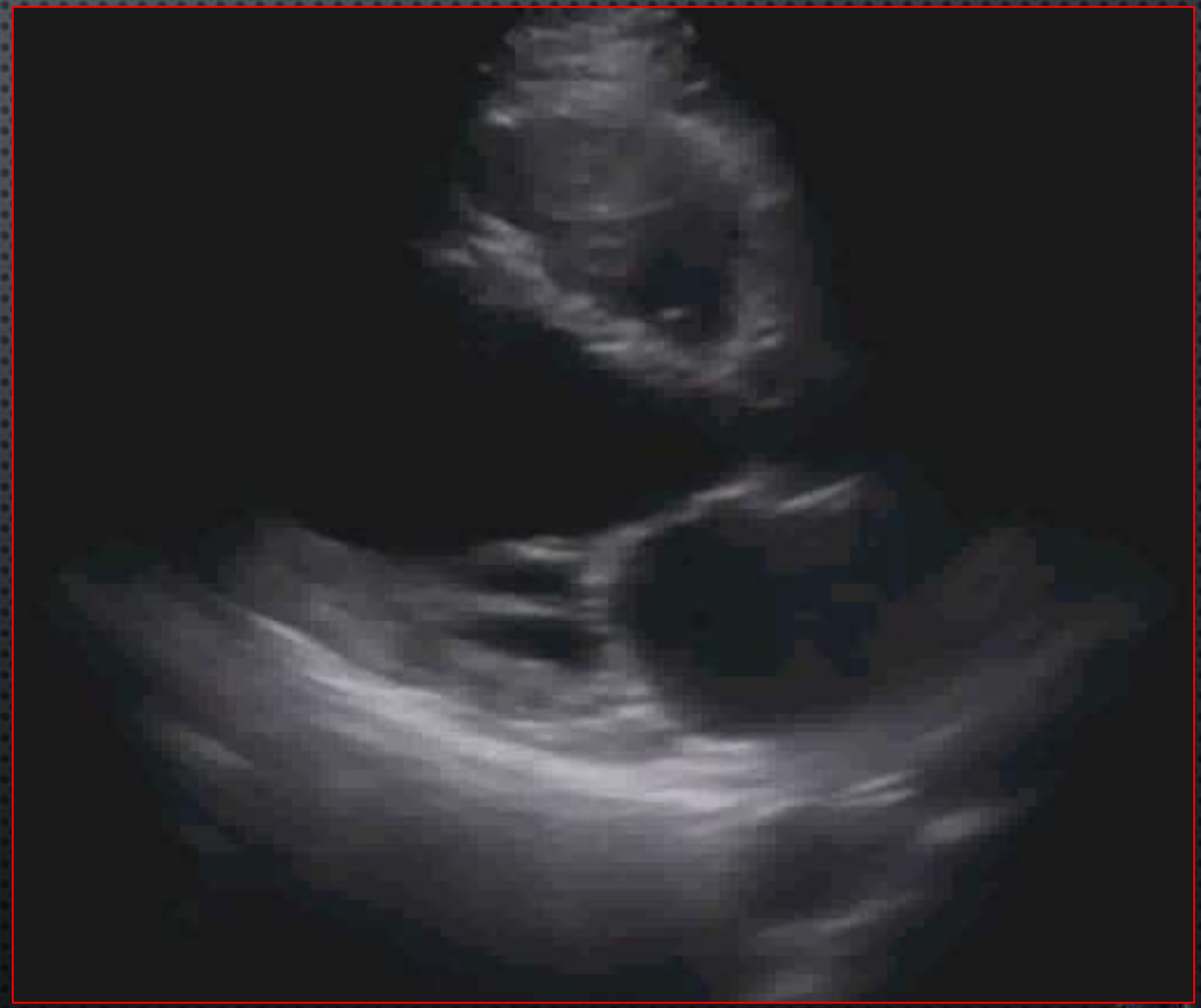
1. **Hypovolemic**
2. **Cardiogenic**
3. **Septic shock**
4. **Dissociative shock**
5. **Obstructive shock**
6. **Anaphylactic shock**

MIXED venous saturation=MVO2

Knowing the etiology is most important to picking agents

..example anaphylaxis

Focus: cardiogenic shock



Drugs

Cardiogenic shock

LOW-CARDIAC-OUTPUT STATE WITH LIFE-THREATENING END-ORGAN HYPOPERFUSION AND HYPOXIA

In-hospital mortality remains high (27%–51%)

Diamond and Forrester subgroup IV with a pulmonary capillary wedge pressure (PCWP) >18 mm Hg and a cardiac index (CI) <2.2 L min−1 m−2, Cardiogenic shock: mortality of 51%.

Most common etiology: AMI

SHOCK Trial[9]*	IABP-SHOCK II[11]†	ESC HF Guidelines[15]
Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of ≤2.2 L·min^{-1}·m^{-2} AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

AHA SCIENTIFIC STATEMENT

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

ABSTRACT: Cardiogenic shock is a high-acuity, potentially complex, and hemodynamically diverse state of end-organ hypoperfusion that is frequently associated with multisystem organ failure. Despite improving survival in recent years, patient morbidity and mortality remain high, and there are few evidence-based therapeutic interventions known to clearly improve patient outcomes. This scientific statement on cardiogenic shock summarizes the epidemiology, pathophysiology, causes, and outcomes of cardiogenic shock; reviews contemporary best medical, surgical

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Peripheral Circulation	Volume Status: Wet	Volume Status: Dry
Cold	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

Use NE first

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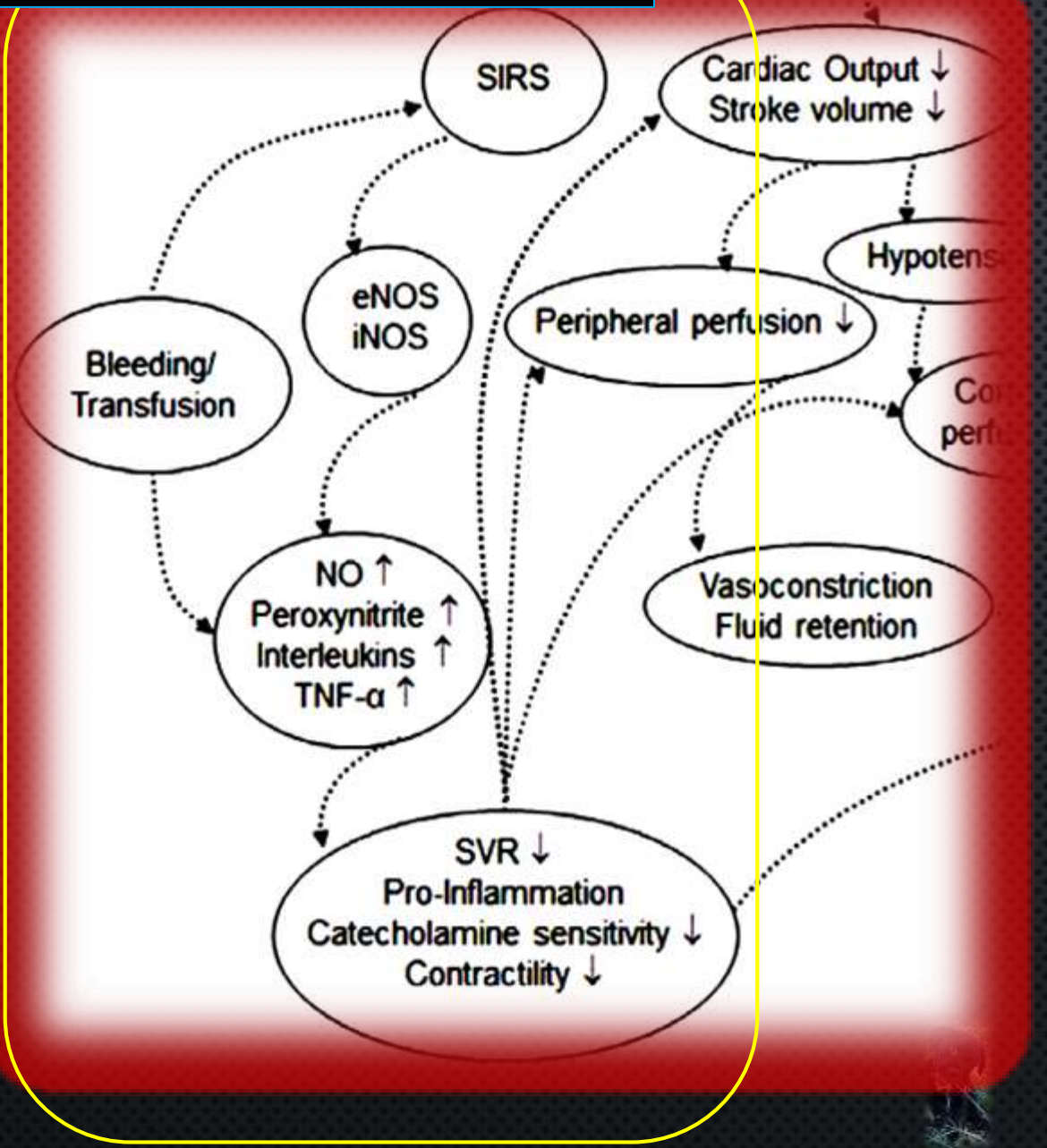
SIRS, systemic inflammatory response syndrome



Medication	Usual Infusion Dose	Receptor Binding				Hemodynamic Effects
		α_1	β_1	β_2	Dopamine	
Vasopressor/inotropes						
Dopamine	0.5–2 μg·kg^{-1}·min^{-1}	–	+	–	+++	↑CO
	5–10 μg·kg^{-1}·min^{-1}	+	+++	+	++	↑↑CO, ↑SVR
	10–20 μg·kg^{-1}·min^{-1}	+++	++	–	++	↑↑SVR, ↑CO
Norepinephrine	0.05–0.4 μg·kg^{-1}·min^{-1}	++++	++	+	–	↑↑SVR, ↑CO
Epinephrine	0.01–0.5 μg·kg^{-1}·min^{-1}	++++	++++	+++	–	↑↑CO, ↑↑SVR
Phenylephrine	0.1–10 μg·kg^{-1}·min^{-1}	+++	–	–	–	↑↑SVR
Vasopressin	0.02–0.04 U/min	Stimulates V_1 receptors in vascular smooth muscle				↑↑SVR, ↔PVR
Inodilators						
Dobutamine	2.5–20 μg·kg^{-1}·min^{-1}	+	++++	++	–	↑↑CO, ↓SVR, ↓PVR
Isoproterenol	2.0–20 μg/min	–	++++	+++	–	↑↑CO, ↓SVR, ↓PVR
Milrinone	0.125–0.75 μg·kg^{-1}·min^{-1}	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Enoximone	2–10 μg·kg^{-1}·min^{-1}	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Levosimendan	0.05–0.2 μg·kg^{-1}·min^{-1}	Myofilament Ca^{2+} sensitizer, PD-3 inhibitor				↑CO, ↓SVR, ↓PVR

Cause or Presentation of CS	Vasoactive Management Considerations
Classic wet and cold	Norepinephrine or dopamine[144] Inotropic agent[210,211]*
Euvolemic cold and dry	Norepinephrine or dopamine[144] Inotropic agent[210,211] Small fluid boluses
Vasodilatory warm and wet or mixed cardiogenic and vasodilatory	Norepinephrine Consider hemodynamics-guided therapy

Why not try NE first

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RV shock	Fluid boluses[144,145] Norepinephrine, dopamine, or vasopressin[144,212,213] Inotropic agents[144]* Inhaled pulmonary vasodilators[214]
Normotensive shock	Inotropic agent or vasopressor
Aortic stenosis	Phenylephrine or vasopressin In patients with reduced LVEF, echocardiography- or PAC-guided dobutamine titration
Aortic regurgitation	Dopamine Temporary pacing
Mitral stenosis	Phenylephrine or vasopressin Esmolol or amiodarone
Mitral regurgitation	Norepinephrine or dopamine Inotropic agents* Temporary MCS, including IABP[144]

Reducing complications

Bundle	Target	Components
ABCDE bundle[219]	Delirium, weakness, and ventilation liberation	Daily awakening and spontaneous breathing trials Assessment and management of delirium Early and progressive mobility
Ventilator bundle[220–222]	Ventilator-associated pneumonia	Head of bed elevation Sedation protocols targeting light sedation with RASS or SAS scores Daily sedation vacation if light sedation contraindicated Chlorhexidine oral rinse Endotracheal tube with subglottic secretion drainage
Central line bundle[223,224]	Central line–associated bloodstream infection	Hand hygiene Maximal barrier precautions Chlorhexidine skin antisepsis Optimal catheter site selection (avoidance of femoral approach) Ultrasound-guided central line placement Daily review of line necessity
Stress ulcer prophylaxis[225,226]	Stress ulcer	Proton pump inhibitor or H_2 blocker in patients without enteral nutrition In enterally fed patients, the risks of prophylaxis should be balanced with risk of ventilator-associated pneumonia
Deep vein thrombosis prophylaxis[226]	Venous thromboembolism	Routine venous thromboembolism prophylaxis in patients not on anticoagulants

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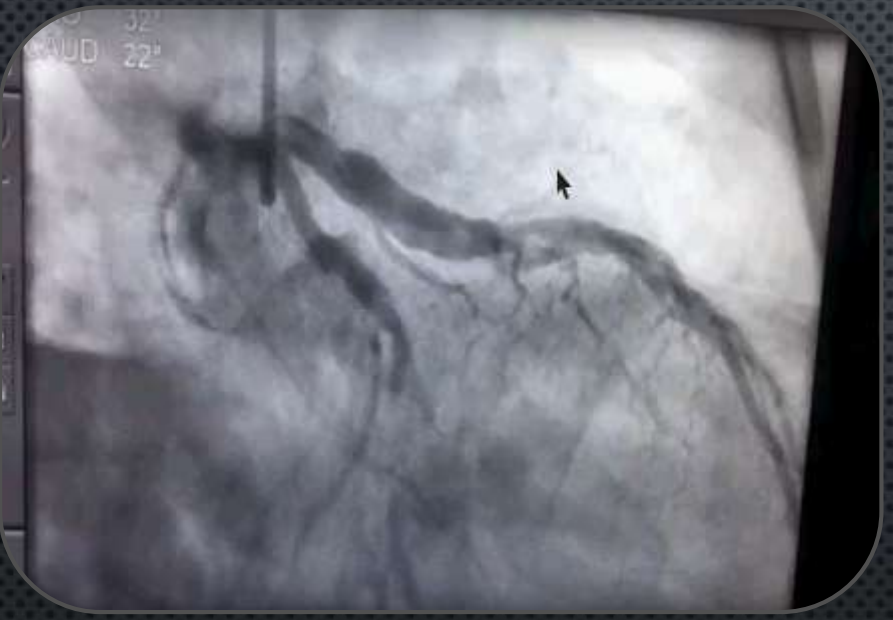
One-Year Outcomes after PCI Strategies in Cardiogenic Shock

H. Thiele, I. Akin, M. Sandri, S. de Waha-Thiele, R. Meyer-Saraei, G. Fuernau, J. Eitel, P. Nordbeck, T. Geisler, U. Landmesser, C. Skurk, A. Fach, A. Jobs, H. Lapp, J.J. Piek, M. Noc, T. Goslar, S.B. Felix, L.S. Maier, J. Stepinska, K. Oldroyd, P. Serpytis, G. Montalescot, O. Barthelemy, K. Huber, S. Windecker, L. Hunziker, S. Savonitto, P. Torremante, C. Vrints, S. Schneider, U. Zeymer, and S. Desch, for the CULPRIT-SHOCK Investigators*

CONCLUSIONS

Among patients with acute myocardial infarction and cardiogenic shock, the risk of death or renal-replacement therapy at 30 days was lower with culprit-lesion-only PCI than with immediate multivessel PCI, and mortality did not differ significantly between the two groups at 1 year of follow-up. (Funded by the European Union Seventh Framework Program and others; CULPRIT-SHOCK ClinicalTrials.gov number, NCT01927549.)



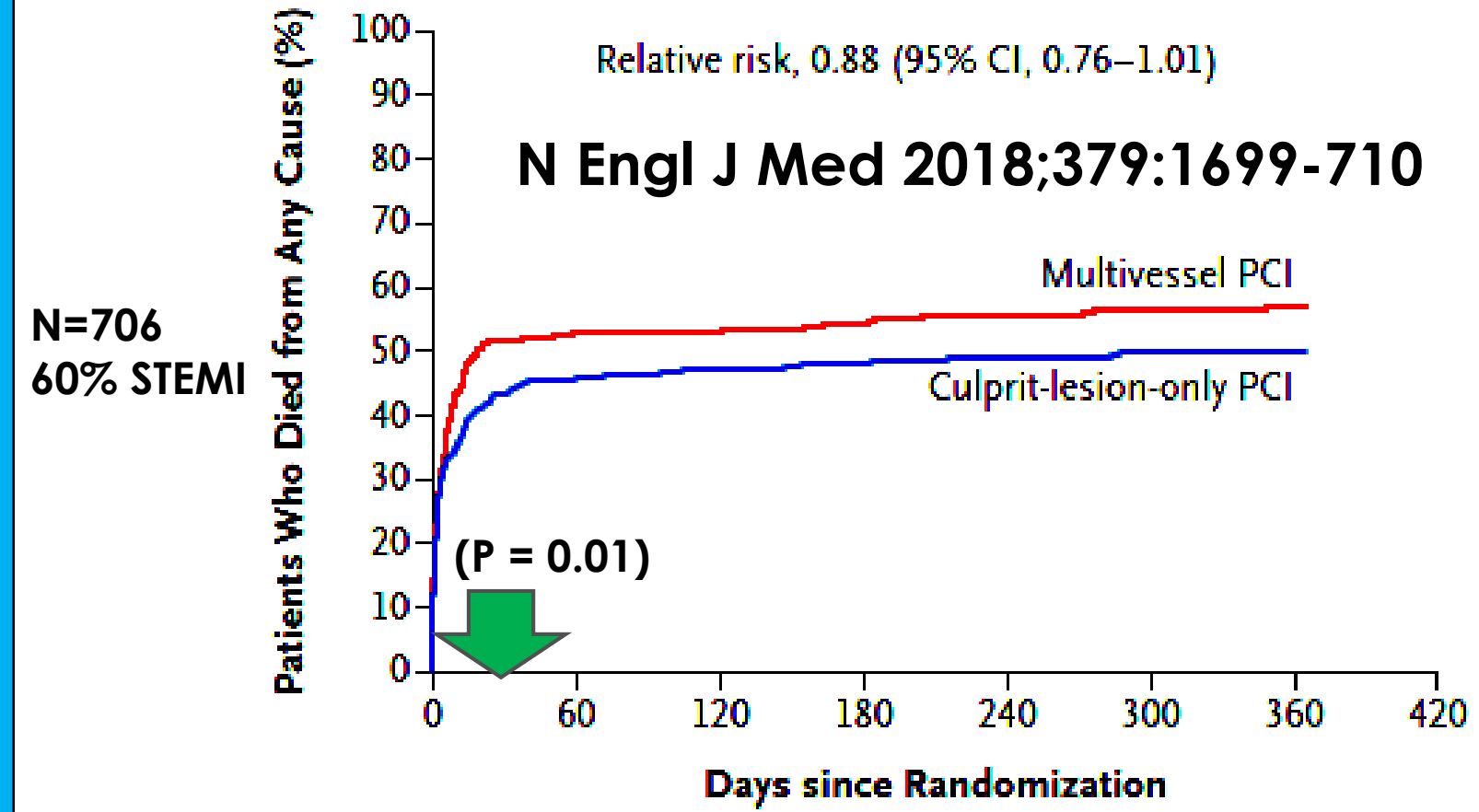
BP 70/50

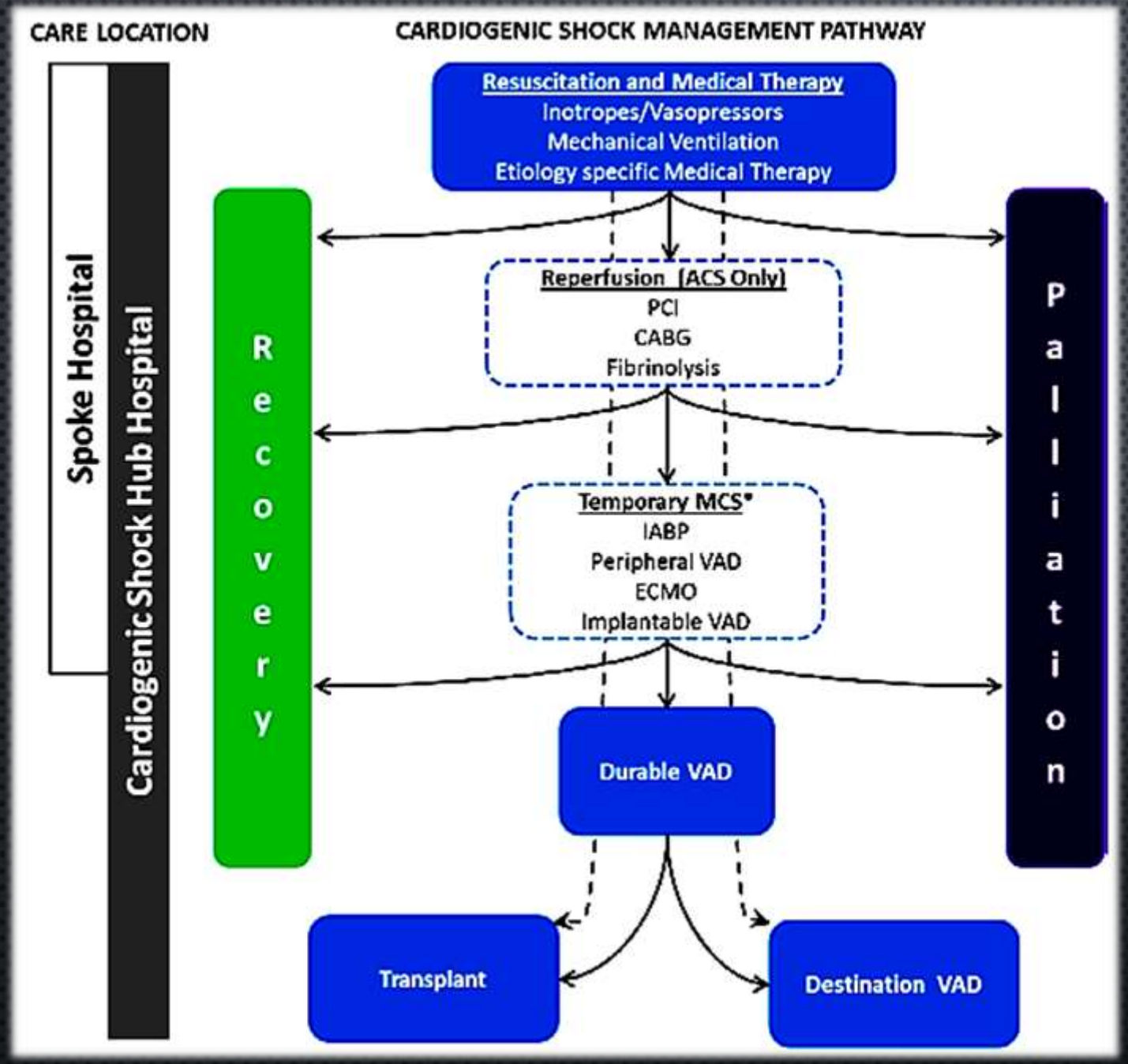
Fix all 3 vessels or just circ?

LAD-90% prox

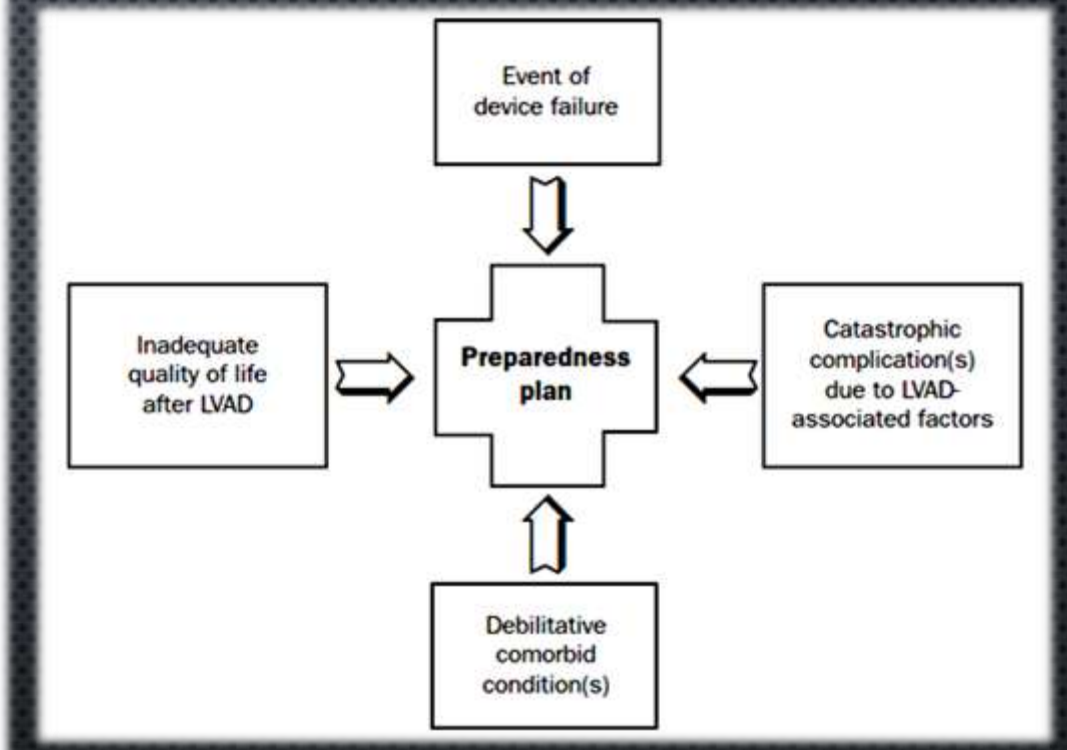
Circ closing

RCA-already closed





Destination Therapy: lots of planning



Summary: Team Approach

- Understanding physiology of BP
- Know patient very well—renal, lung, cardiac, blood
- Know drugs and which is best for that patient
- If planning on transplant: talk with family and transplant referral team very early