Cardiogenic Shock: The Evolution of Patient and Device Selection for Acute Circulatory Support

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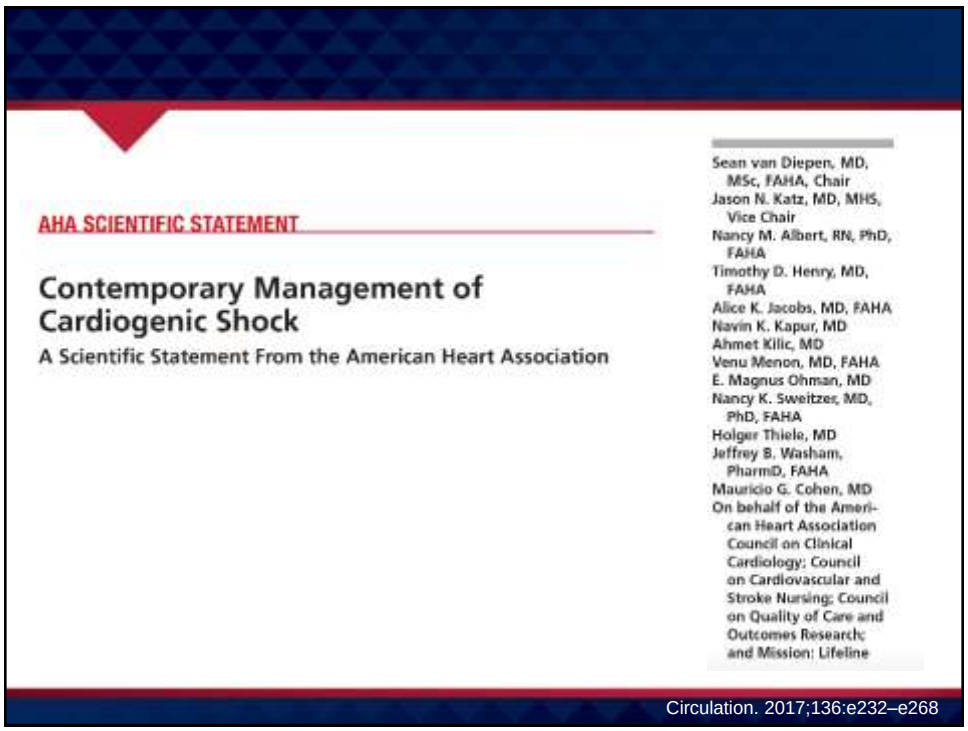
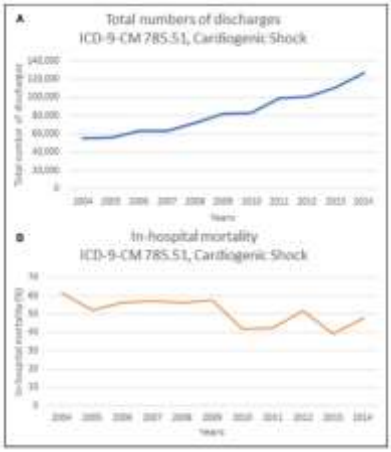




Mandawat A & Rao SV. Circ Cardiovasc Interv. 2017;10:e004337

Table 1
Definitions of pre-cardiogenic shock, cardiogenic shock, and refractory cardiogenic shock according to clinical and hemodynamic criteria and response to therapy

	Pre-CS (Nonhypotensive)	CS	Refractory CS
Clinical criteria	Signs of peripheral hypoperfusion: Oliguria (urine output <30 mL/h) Cold extremities Altered mental status Increased serum lactate	Signs of peripheral hypoperfusion	Signs of peripheral hypoperfusion
Hemodynamic criteria	SBP ≥90 mm Hg without circulatory support	SBP <90 for >30 min or the need for pharmacologic or intra-aortic balloon pump support to maintain a systolic blood pressure >90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline. Cardiac index <2.2 L/min/m². Elevated filling pressures of the left, right, or both ventricles	Same as CS
Response to treatment			Ongoing evidence of tissue hypoperfusion despite administration of adequate doses of 2 vasoactive medications and treatment of the underlying etiology.

Furer A, Wexler J, Burkhoff D, Intervent Cardiol Clin 6 (2017) 359–371

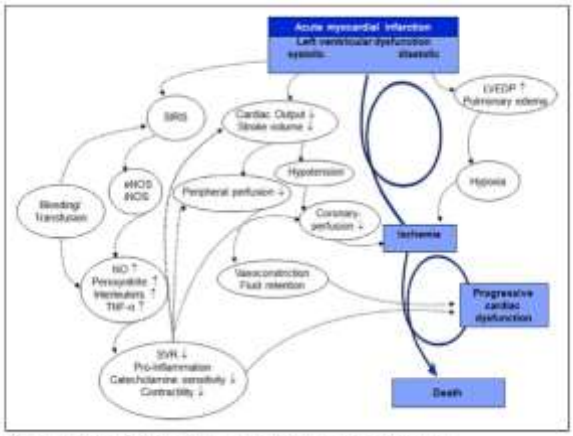
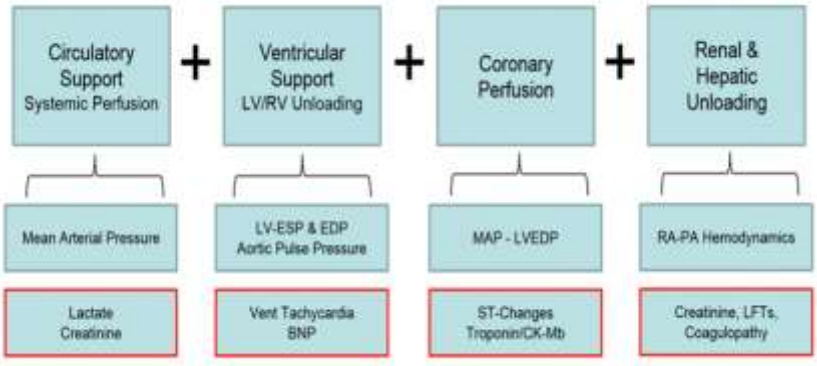


Figure 1. The pathophysiological concept of the expanded cardiogenic shock spiral.

Circulation. 2017;136:e232–e268

Three Objectives of Shock Care

1. Circulatory support
2. Ventricular unloading
3. Coronary perfusion



Esposito ML & Kapur NK, F1000Research 2017, 6(F1000 Faculty Rev):737

Integrated Shock Team

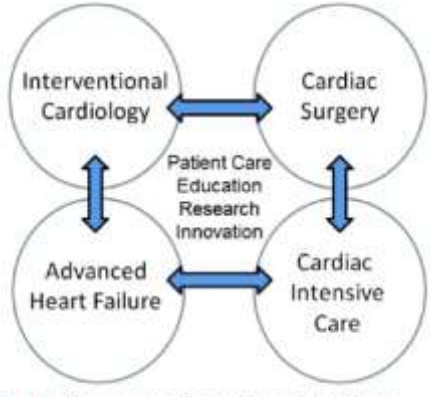
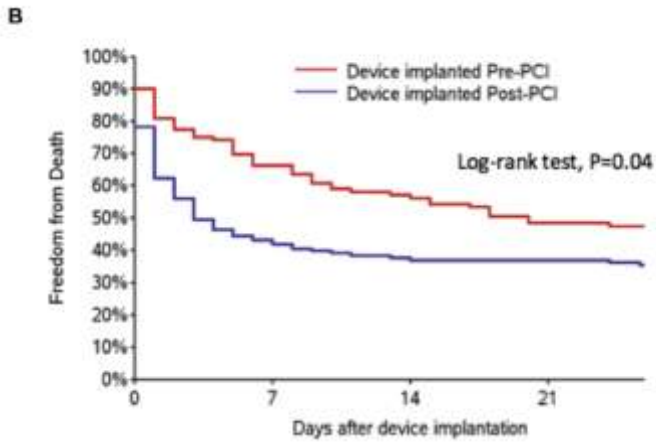


Fig. 2. Components of a cardiogenic shock team.

Kapur NK, Davila CD & Jumean MF. Intervent Cardiol Clin 6 (2017) 481–485



Basir MB et al, Am J Cardiol 2017;119:845 - 851

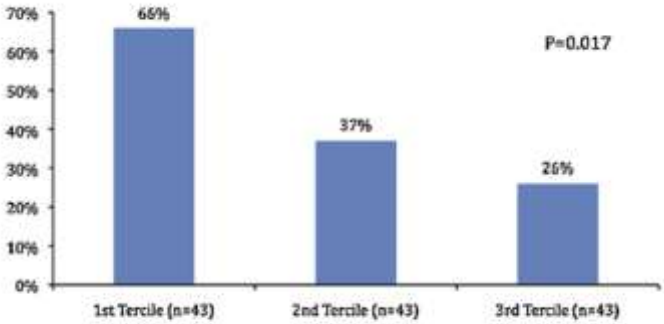


Figure 3. Inhospital survival rates as a function of shock onset to MCS implantation.

Basir MB et al, Am J Cardiol 2017;119:845 - 851

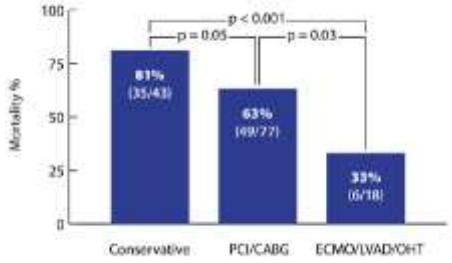
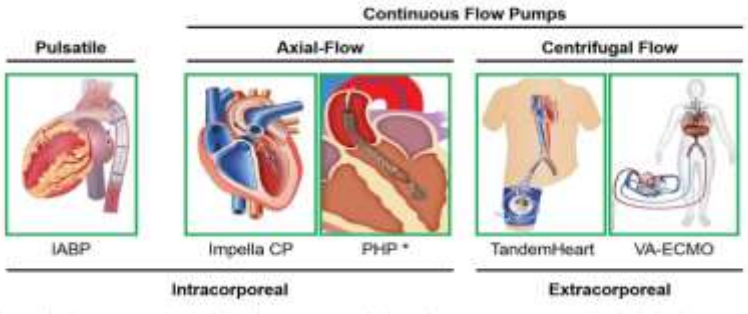


Figure 1. In-hospital mortality according to treatment strategy. Mortality varied significantly among 3 alternative primary treatment strategies in ST segment elevation acute myocardial infarction complicated by cardiogenic shock. CABG, coronary artery bypass graft; ECMO, extracorporeal membrane oxygenation; LVAD, left ventricular assist device; OHT, orthotopic heart transplantation; PCI, percutaneous coronary intervention. Modified from Tayara et al.[11] with permission from Elsevier.

Kalavrouziotis D et al, Canadian Journal of Cardiology 33 (2017) 36e43



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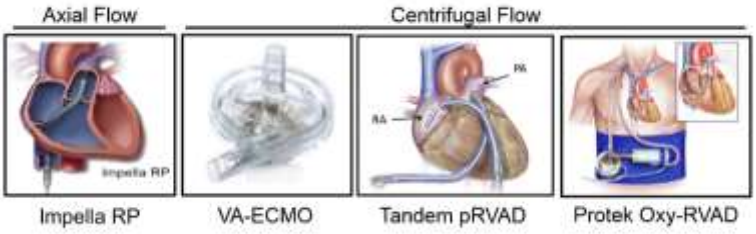

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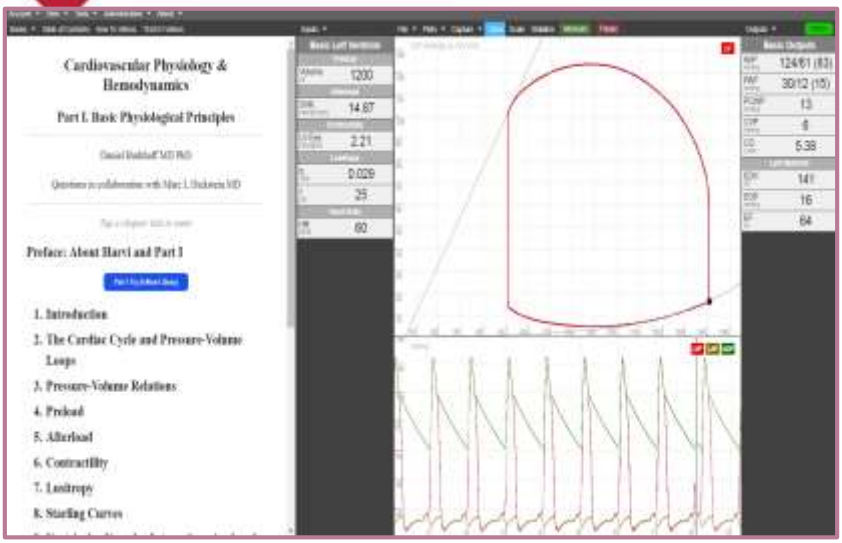

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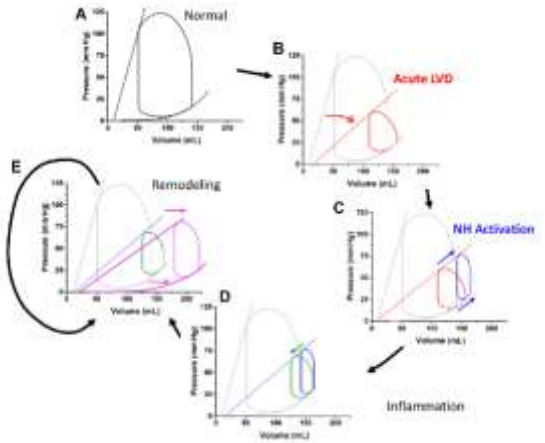

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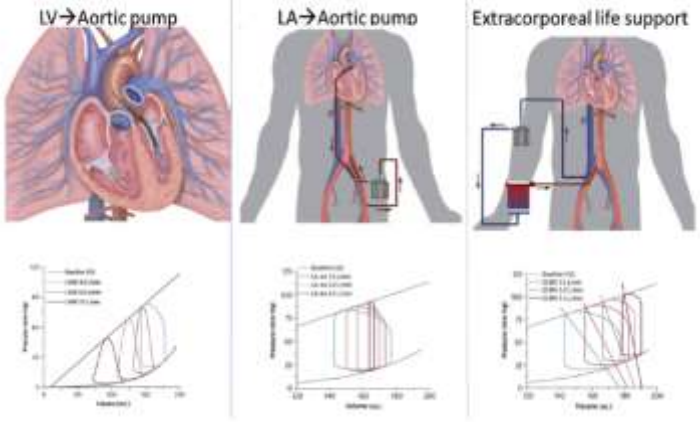

Furer A, Wexler J, Burkhoff D, Intervent Cardiol Clin 6 (2017) 359–371

Adv Physiol Educ 41: 415–424, 2017;
doi:10.1152/advan.00204.2016.

SOURCEBOOK OF LABORATORY ACTIVITIES IN PHYSIOLOGY

Use of an iPad App to simulate pressure-volume loops and cardiovascular physiology

Staci Leisman[1] and Daniel Burkhoff[2]

[1]Department of Medicine and Medical Education, Icahn School of Medicine at Mount Sinai, New York, New York; and [2]Cardiovascular Research Foundation and Columbia University Division of Cardiology, New York, New York

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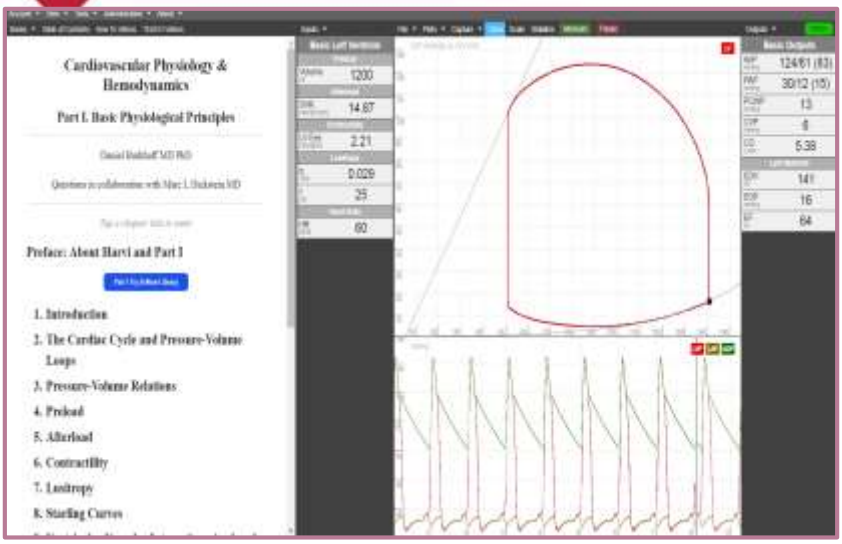

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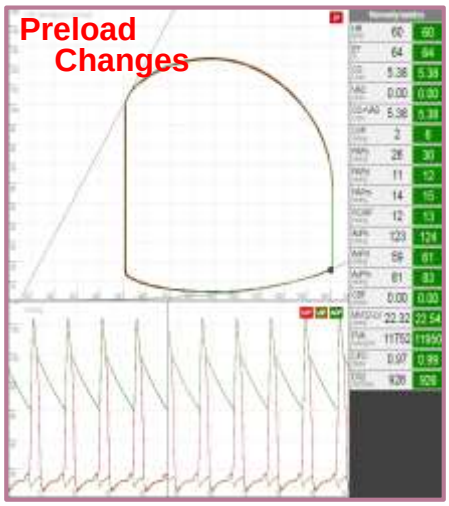
THE BASICS: Preload, Afterload, Contractility, Di



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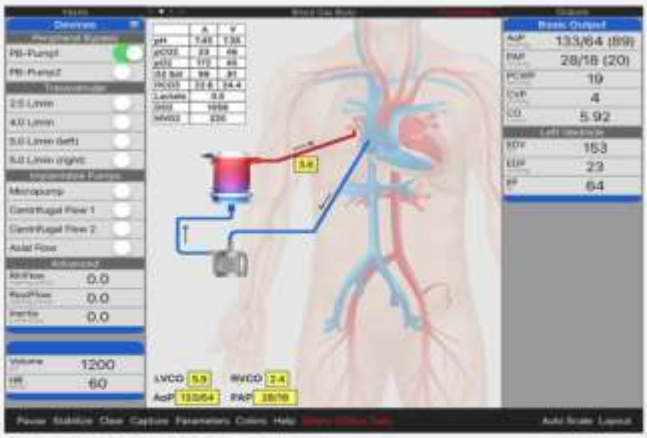

An alternate view shows the anatomic placement of inflow and outflow ports of different devices. There is a full range of devices and configurations to choose from.

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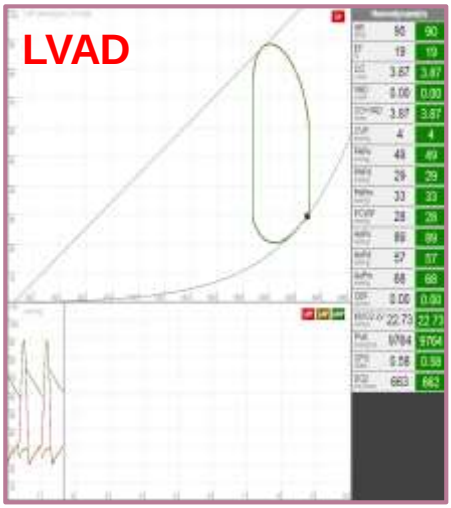
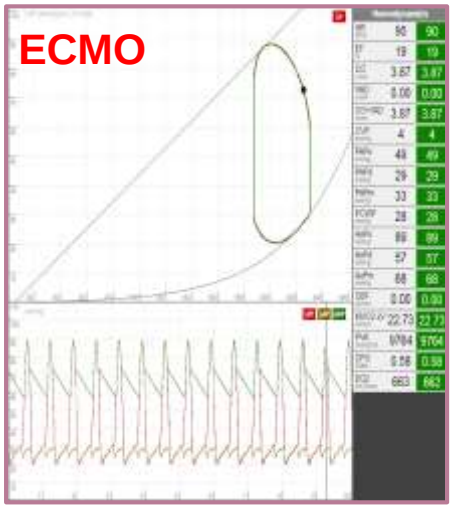
ND THERAPEUTICS: CHF, VADs, ECMO, Valves, Shunts, Drugs





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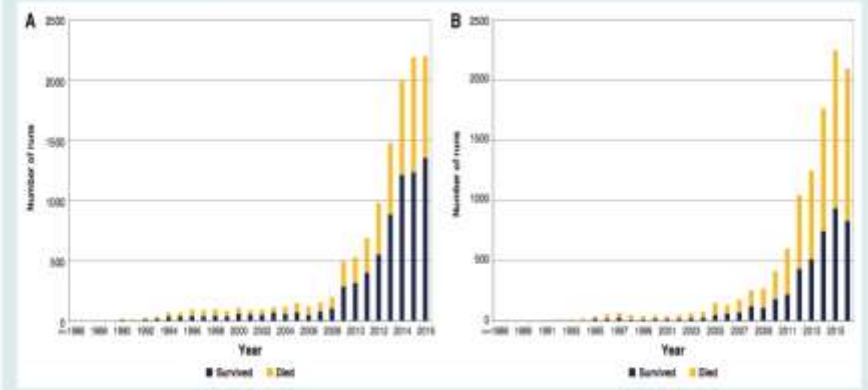
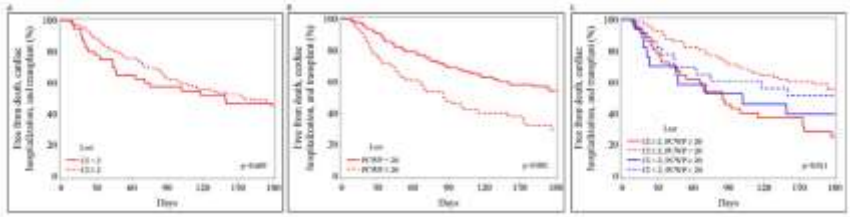


Figure 1 Adult (A) Respiratory and (B) Cardiac ECMO runs by year with survival rate (from ELSO report 2016 www.elso.org).

Sorokin et al. Eur J Heart Failure 2017



Cooper LB et al., J Card Fail; 2016; 22:182-189

Integrated Shock Team

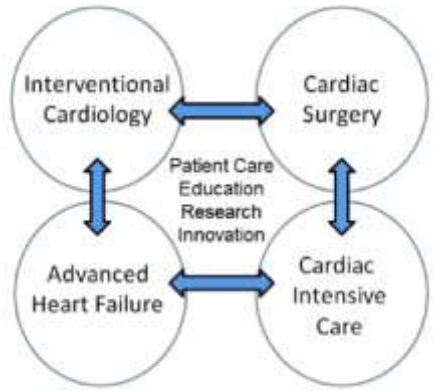


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Table 2. Comparison of Left Ventricular and Right Ventricular Percutaneous Mechanical Circulatory Support Devices

	IABP (Ascending Ao)	Impella 2.5 (LV/LVOT)	Impella 5.0 (LV/LVOT)	TandemHeart (LA to Systemic Artery)	ECMO (Femoral Artery and Vein)	TandemHeart (RA to Ao)	Impella RP (RA to PA)
Primary hemodynamic effect(s)	LV volume or pressure unloading	LV volume or pressure unloading	LV volume or pressure unloading	LV volume unloading	Biventricular pressure and volume unloading	RV pressure unloading	LV volume or pressure unloading
TPR	Decreased	Decreased	Decreased	Mildly increased	Highly increased	None	None
Bridge to recovery	Yes	Yes	Yes	Yes	No*	Yes	Yes
Hemodynamic support	Low	Moderate	High	High	High	High	Moderate
Pump mechanism	Pneumatic	Axial flow	Axial flow	Centrifugal	Centrifugal	Centrifugal	Axial flow
Cannula size	7.5 Fr	13 Fr	22 Fr	21 Fr inflow; 15–17 Fr outflow	18–21 Fr inflow; 15–22 Fr outflow	21 Fr inflow; 15–17 Fr outflow	13 Fr
Implantation time	Very low	Low	Moderate	High	Moderate	High	Low
Risk of limb ischemia	Very low	Low	Low	High	High	High	Low
Anticoagulation	Very low	Very low	Very low	High	High	High	Very low
Hemolysis	Very low	Low	Low	Low	Low	Low	Low
Postimplantation management complexity	Very low	Moderate	Moderate	High	Very high	High	Moderate

Ao indicates aorta; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LA, left atrium; LV, left volume; LVOT, left ventricular outflow tract; PA, pulmonary artery; RA, right atrium; RV, right volume; TPR, total peripheral resistance.

*Except in extracorporeal cardiopulmonary resuscitation (eCPR).

Mandawat A & Rao SV. Circ Cardiovasc Interv. 2017;10:e004337

Table 5. Initial Vasoactive Management Considerations in Types of CS

Cause or Presentation of CS	Vasoactive Management Considerations	Hemodynamic Rationale
Classic wet and cold	Norepinephrine or dopamine[**] Inotropic agent[**,***]	This subtype has low CI and high SVR. Consider hemodynamic stabilization with norepinephrine (preferred in SVR or arrhythmias) or dopamine (CHR preferred but associated with higher risk of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only)
Euvolemic cold and dry	Norepinephrine or dopamine[**] Inotropic agent[**,***] Small fluid boluses	Consider hemodynamic stabilization with norepinephrine (preferred in SVR or arrhythmias) or dopamine (CHR preferred but associated with higher risk of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only) LVEDP may be low, and patients may tolerate fluid boluses
Vasodilatory warm and wet or mixed cardiogenic and vasodilatory	Norepinephrine Consider hemodynamics-guided therapy	This subtype has low SVR
RV shock	Fluid boluses[**,***] Norepinephrine, dopamine, or vasopressin[**,***,†††] Inotropic agents[***] Inhaled pulmonary vasodilators[†††]	Hemodynamic goals include maintaining preload, lowering RV afterload (PVR), treating absolute or relative bradycardias, and maintaining atrioventricular synchrony Dopamine (CHR preferred but associated with arrhythmia risk) Vasopressin may raise SVR and have neutral effect on PVR Consider adding or transitioning to inotrope after initial hemodynamic stabilization and revascularization
Normotensive shock	Inotropic agent or vasopressor	Initial inotropic therapy may be appropriate given that this subtype has SBP >90 mm Hg and relatively high SVR
Aortic stenosis	Phenylephrine or vasopressin In patients with reduced LVEF, echocardiography- or PAC-guided dobutamine titration	Shock caused by aortic stenosis is an afterload-dependent state Inotropy may not improve hemodynamics if LVEF is preserved Definitive therapies will be defined by underlying cause and may include surgical aortic valve replacement or balloon valvuloplasty and/or transcatheter aortic valve replacement

Circulation. 2017;136:e232–e268

1. Early hemodynamic assessment
2. Early use of acute MCS devices
3. Identification of optimal door-to-support time
4. Appropriate acute MCS device selection
5. Early use of decongestive therapy (to reduce metabolic failure)

Esposito ML & Kapur NK, F1000Research 2017, 6(F1000 Faculty Rev):737

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