

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

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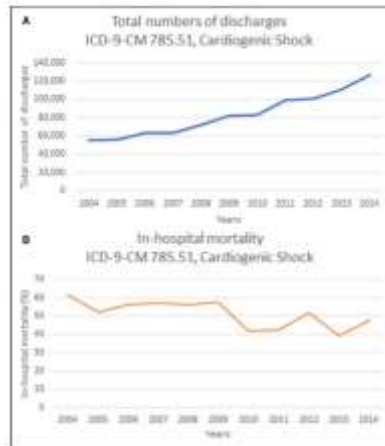
AHA SCIENTIFIC STATEMENT

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

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On behalf of the American
Heart Association
Council on Clinical
Cardiology; Council
on Cardiovascular and
Stroke Nursing; Council
on Quality of Care and
Outcomes Research;
and Mission: Lifeline

Circulation. 2017;136:e232–e268



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 (Nohypotensive)	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 shockoly 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/m ² . 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

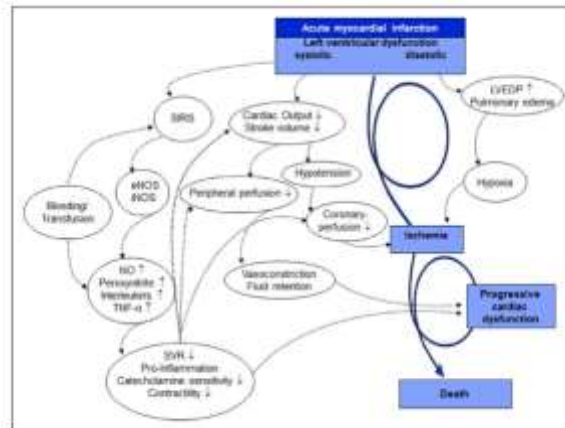
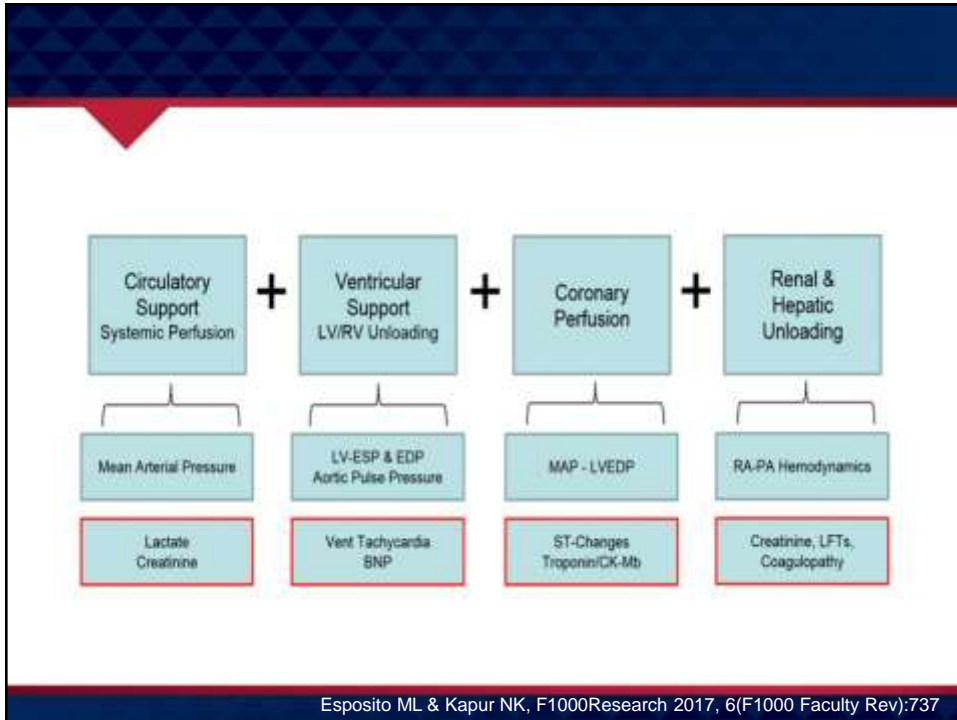


Figure 1. The pathophysiological concept of the expanded cardiogenic shock spiral.

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Three Objectives of Shock Care

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



Integrated Shock Team

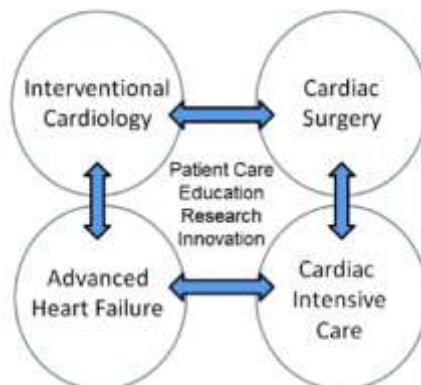
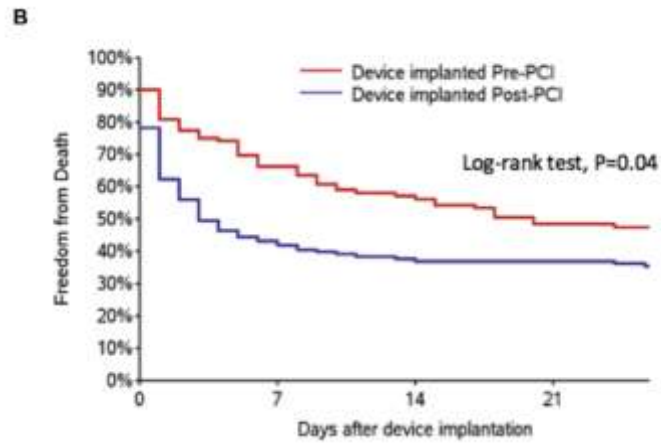


Fig. 2. Components of a cardiogenic shock team.



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

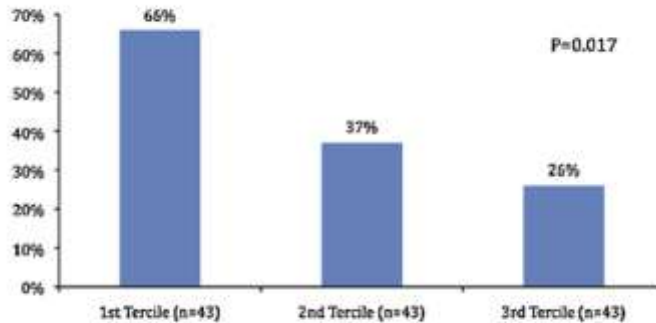


Figure 3. In-hospital survival rates as a function of shock onset to MCS implantation.

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

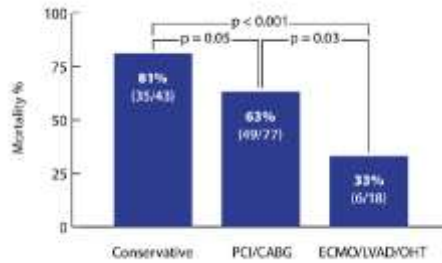
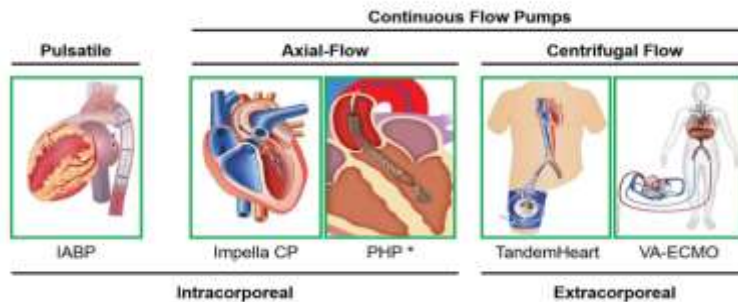


Figure 1. In-hospital mortality according to treatment strategy. Mortality varied significantly among 3 alternative primary treatment strategies in ST segment elevation acute myocardial infarction complicated by cardiogenic shock. CABG, coronary artery bypass graft; ECMO, extracorporeal membrane oxygenation; 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



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

The diagram illustrates two types of extracorporeal circulation pumps: Axial Flow and Centrifugal Flow.

- Axial Flow:** Shown with the Impella RP pump, which is a catheter-based device inserted into the heart.
- Centrifugal Flow:** Shown with three examples:
 - VA-ECMO (Venous-Arterial Extracorporeal Membrane Oxygenation)
 - Tandem pRVAD (Pump in Right Ventricular Assist Device)
 - Protek Oxy-RVAD (Right Ventricular Assist Device)

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

Harvi: A comprehensive, educational cardiovascular simulation and online textbook

The screenshot displays the Harvi online cardiovascular simulation and textbook interface. The main content area is titled "Cardiovascular Physiology & Hemodynamics" and "Part I: Basic Physiological Principles" by Daniel Dinkhof MD PhD. A table of hemodynamic parameters is shown on the right, and a pressure-volume loop graph is displayed in the center. Below the graph, a list of topics is provided for study.

Parameter	Value
MAP	1200
HR	14.67
CO	2.21
CI	0.026
SV	25
PR	60

Basic Hemodynamics:

HR	124/91 (83)
MAP	30/12 (10)
PCWP	13
CO	6
CI	5.38
SV	141
EDV	16
EF	64

Part I: Basic Physiological Principles

Daniel Dinkhof MD PhD

Questions in collaboration with Marc L. Haken MD

The original 2013 work

Preface: About Harvi and Part I

1. Introduction

2. The Cardiac Cycle and Pressure-Volume Loops

3. Pressure-Volume Relations

4. Preload

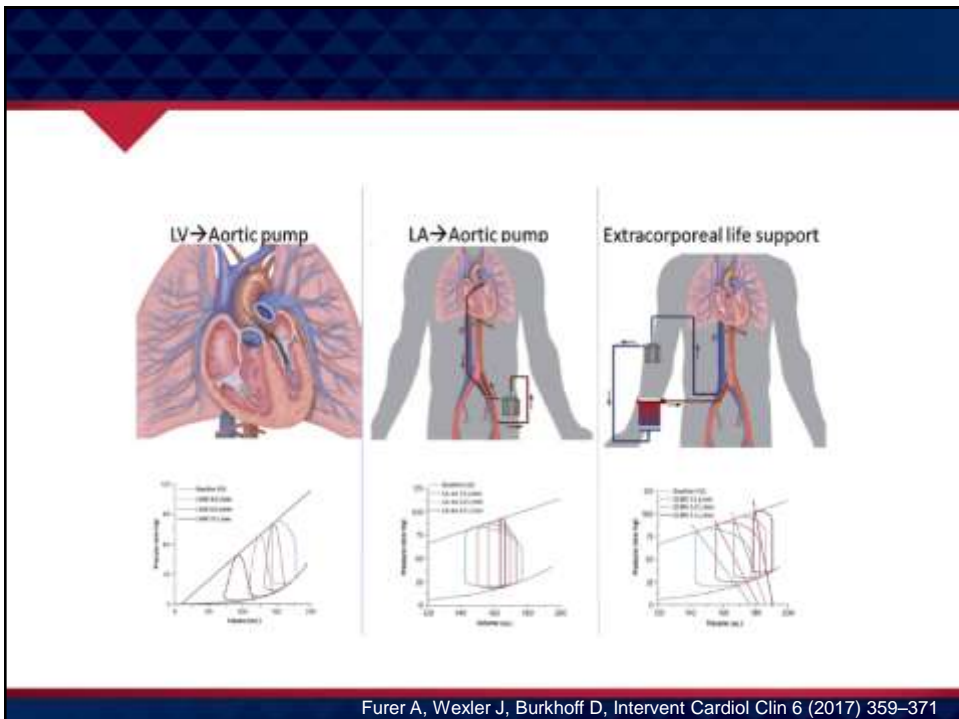
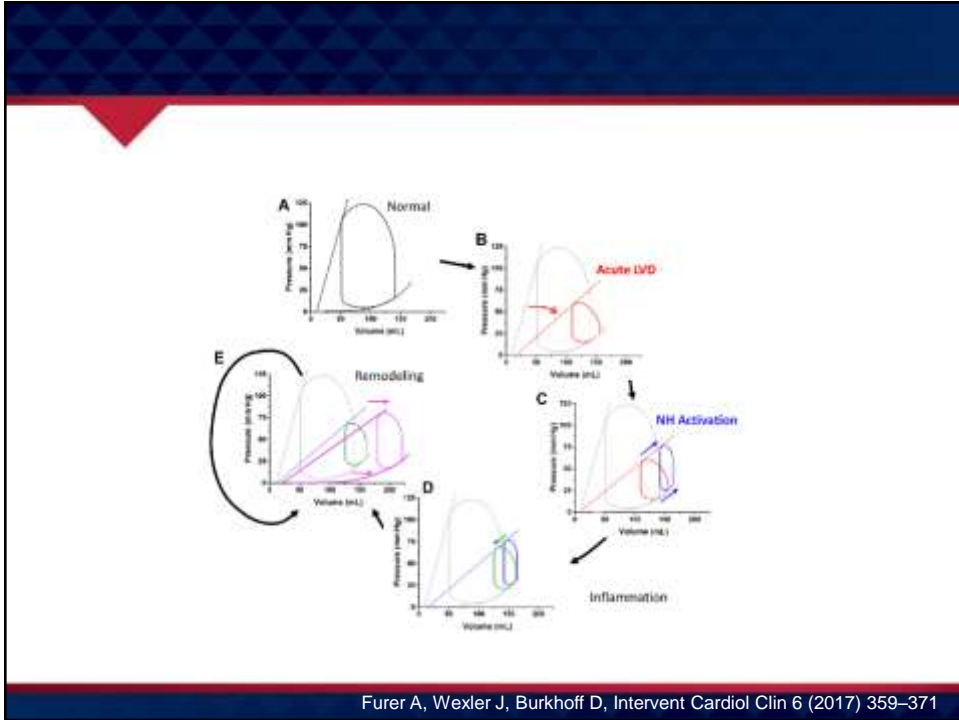
5. Afterload

6. Contractility

7. Load-tropy

8. Starling Curves

<http://harvi.online> and www.crfteach.com



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 Leisman¹ and Daniel Burkhoff²

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

Submitted 5 January 2017; accepted in final form 12 May 2017



Meet Harvi

Your guide for learning cardiac physiology, hemodynamics, pathophysiology and therapeutics through the combined power of a novel e-textbook series and a dynamic, interactive simulation.

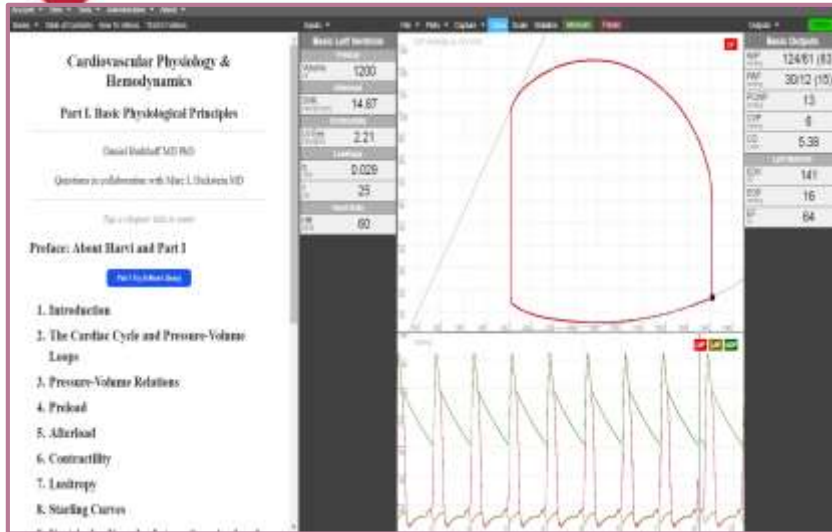
This website is under construction and the current content provides basic information. Please [email us](#) for more information and to get access to the simulation.

You now have two options:

Harvi iPad App

Harvi Online

Harvi: A comprehensive, educational cardiovascular simulation and online textbook



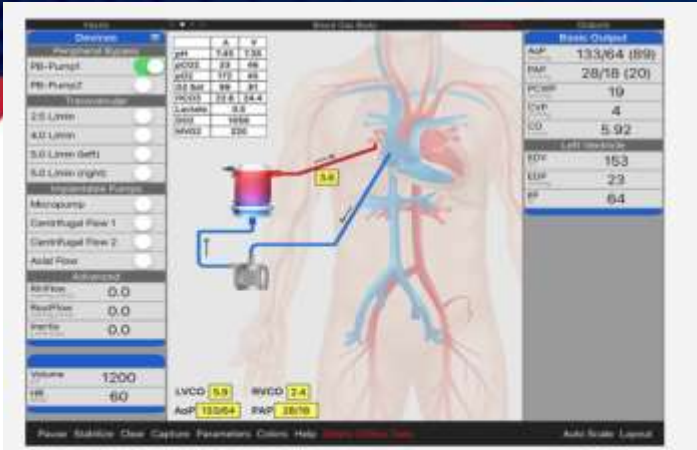
<http://harvi.online> and www.crfteach.com

Harvi: A comprehensive, educational cardiovascular simulation and online textbook

THE BASICS: Preload, Afterload, Contractility, Diastolic Pressure



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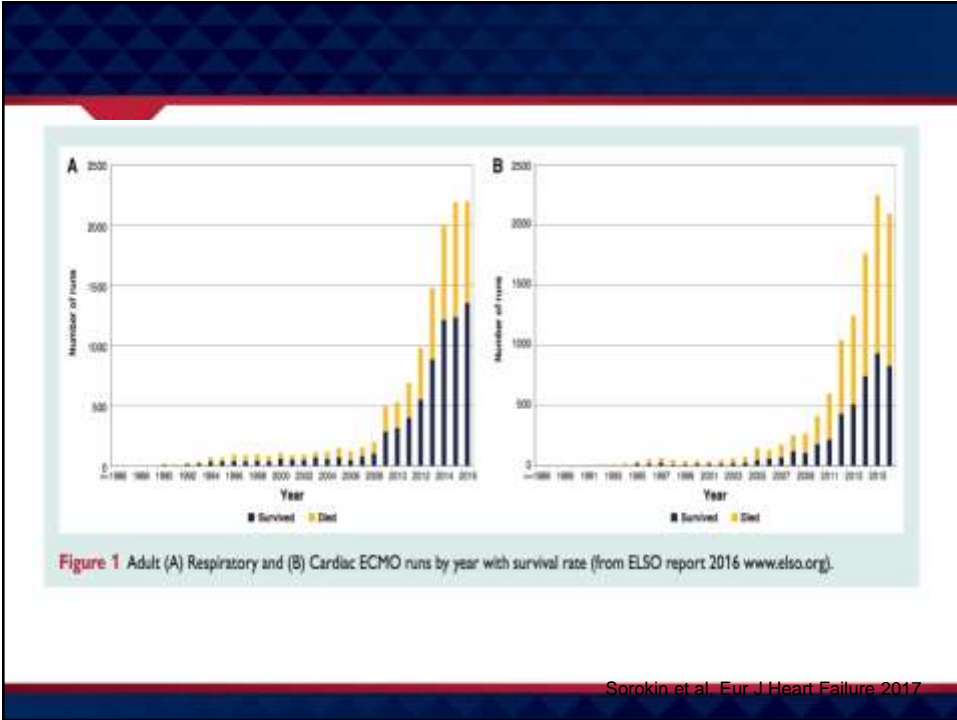
An alternate view shows the anatomic placement of inflow and outflow ports of different devices. There is a full range of devices and configurations to choose from.

Harvi: A comprehensive, educational cardiovascular simulation and online textbook

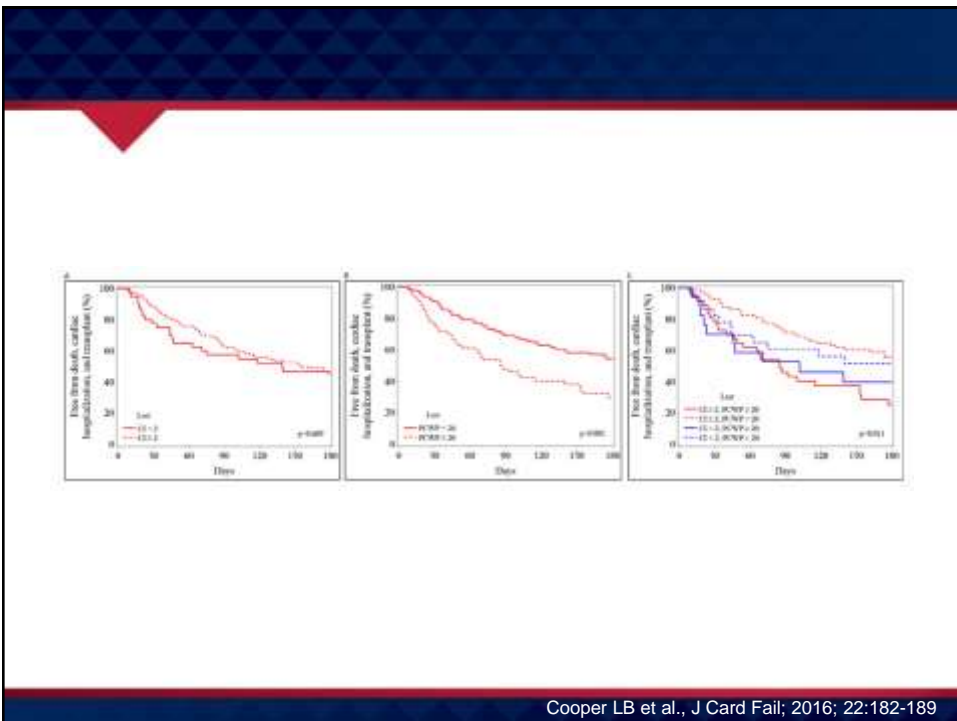
AND THERAPEUTICS: CHF, VADs, ECMO, Valves, Shunts, Drugs



<http://harvi.online> and www.crfteach.com



Sorkin et al. Eur J Heart Failure 2017



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

Integrated Shock Team

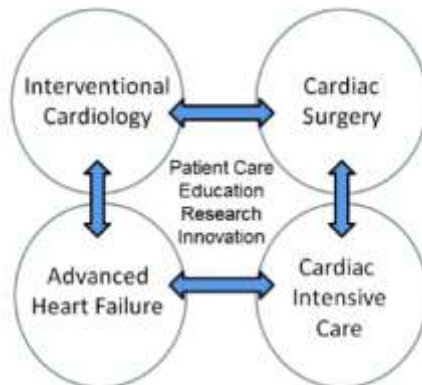


Fig. 2. Components of a cardiogenic shock team.

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

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

	IMP (Ascending Aa)	Impella 2.5 (LV/LVOT)	Impella 5.0 (LV/LVOT)	Tandemheart LA (to Systemic Artery)	ECMO (Pulmonary Artery and Vein)	Tandemheart (RA to Aa)	Impella RP (RA to PV)
Primary hemodynamic effect(s)	LV volume or pressure unloading	LV volume or pressure unloading	LV volume or pressure unloading	LV volume unloading	Decreases pressure and volume unloading	RV pressure unloading	LV volume or pressure unloading
TPR	Decreased	Decreased	Decreased	Mildly increased	Highly increased	None	None
Drugs to recover	Yes	Yes	Yes	Yes	No*	Yes	Yes
Hemodynamic support	Low	Moderate	High	High	High	High	Moderate
Pump mechanism	Pressure	Axial flow	Axial flow	Centrifugal	Centrifugal	Centrifugal	Axial flow
Cannula size	7.3 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

As indicated aorta, ECMO, extracorporeal membrane oxygenation; IMP, intra-aortic balloon pump; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; RA, pulmonary artery; RA, right atrium; RV, right ventricle; TPR, total peripheral resistance.

*Except in extracorporeal cardiopulmonary resuscitation (ECPR).

Adapted from Dumesnil et al.¹ Copyright© 2012, BMJ Publishing Group Ltd and the British Cardiovascular Society. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

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

Contemporary Management of Cardiogenic Shock

Table 5. Initial Vasoactive Management Considerations in Types of CS

Cause or Presentation of CS	Vasoactive Management Considerations	Hemodynamic Rationale
Clinical wet and cold	Norepinephrine or dopamine ¹⁴ Inotropic agent ^{15,16,17}	This subtype has low CI and high SVR. Consider hemodynamic stabilization with norepinephrine (preferred in SH or arhythmias) or dopamine (HR preferred but associated with higher risk of arrhythmias). Consider addition of inotropic agent when stabilized and after revascularization (MI only).
Ischemic cold and dry	Norepinephrine or dopamine ¹⁴ Inotropic agent ^{15,17} Small fluid boluses	Consider hemodynamic stabilization with norepinephrine (preferred in SH or arhythmias) or dopamine (HR preferred but associated with higher risk of arrhythmias). Consider addition of inotropic agent when stabilized and after revascularization (MI only). DUEP may be low, and patients may tolerate fluid boluses.
Vasodilatory warm and wet or mixed cardiogenic and vasodilatory	Norepinephrine Consider hemodynamic-guided therapy	This subtype has low SVR.
RV shock	Fluid boluses ^{18,19} Norepinephrine, dopamine, or vasopressin ^{14,20,21,22} Inotropic agents ^{15,16} Inhaled pulmonary vasodilator ²³	Hemodynamic goals include maintaining preload, lowering RV afterload (PVR), treating absolute or relative bradycardia, and maintaining atrioventricular synchrony. Dopamine (HR preferred but associated with arrhythmia risk). Vasopressin may raise SVR and have neutral effect on PVR. Consider adding or transitioning to inotropes after initial hemodynamic stabilization and revascularization.
Normotensive shock	Inotropic agent or vasopressin	Initial inotropic therapy may be appropriate given that this subtype has SBP >90 mmHg and relatively high SVR.
Aortic stenosis	Phenylephrine or vasopressin in patients with reduced CO, echocardiography or PFC-guided dobutamine titration	Shock caused by aortic stenosis is an afterload-dependent state. Inotropes may not improve hemodynamics if SVR 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.

CLINICAL STATEMENTS AND GUIDELINES

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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Thank You!

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