The Next Stage in Cardiogenic Shock

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Timing is
everything.

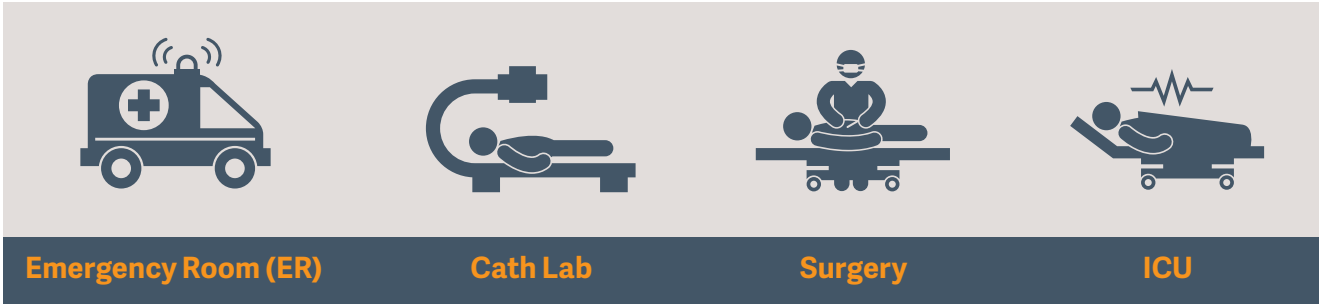
Cardiogenic Shock

The unresolved clinical challenge

The problem

Cardiogenic shock (CS) can present in many different stages. It ranges from those at high risk of developing shock to those critically ill with severe multi-system organ failure, hemodynamic collapse and on-going cardiac arrest.[1]

It can happen anywhere



Cardiogenic shock can present along multiple points in the pathway of care and develops more frequently after initial presentation to the hospital.[2]

Cardiogenic shock patients represent a wide spectrum of disease that requires tailored therapy to improve hemodynamic derangements.[3]

Develop a treatment plan

Identify

Any attempt to improve outcomes in CS should begin with its early identification. Models of care including a multi-disciplinary CS team, hold potential for the early identification and individualized treatment of CS.[4]

Initiate

Experts suggest use of advanced hemodynamic monitoring to diagnose and/or manage patients with CS.[1] To avoid the negative impact of inotropes, consideration should be given to early initiation of intra-aortic balloon pumping.[5,7]

Evaluate

Quick feedback loops incorporating patient status and hemodynamics are required to assess the response to initial therapies.[3]

Escalate

When patients do not respond to treatments initiated, consider the next level of support and transfer to experienced shock centers if required.[1]

Identification is critical

Stages of Cardiogenic Shock[1]

Cardiogenic Shock Stages	At Risk	Beginning	Classic	Deteriorating	Extremis
	A patient with risk factors for cardiogenic shock who is not currently experiencing signs or symptoms. For example, large acute myocardial infarction, prior infarction, acute and/or acute on chronic heart failure.	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	A patient presenting with hypoperfusion requiring intervention beyond volume resuscitation (inotrope, pressor, or mechanical support including ECMO). These patients typically present with relative hypotension.	A patient who fails to respond to initial interventions. Similar to Stage C and getting worse.	A patient being supported by multiple interventions who may be experiencing cardiac arrest with ongoing CPR and/or ECMO.
Physical Signs	• Normal JVP • Lung sounds clear • Strong distal pulses • Normal mentation	• Elevated JVP • Rales in lung fields • Strong distal pulses • Normal mentation	• Ashen, mottled, dusky • Volume overload • Extensive rales • Killip class 3 or 4 • Non-invasive or invasive ventilation • Acute alternation in mental status • Urine Output < 30 mL/h	• Any of stage C	• Near pulselessness • Cardiac collapse • Mechanical ventilation • Defibrillator used
Biochemical Markers	• Normal renal function • Normal lactic acid	• Normal lactate • Minimal renal function impairment • Elevated BNP	• Lactate ≥ 2 • Creatinine doubling **OR** > 50% drop in GFR • Increased LFTs • Elevated BNP	• Any of stage C **AND** deteriorating	• Lactate ≥ 5 • pH ≤ 7.2
Hemodynamics	• Normotensive (SBP > 100 **OR** normal for pt.) If hemodynamics done: • Cardiac index ≥ 2.5 • CVP < 10 • PA Sat ≥ 65%	• SBP < 90 **OR** MAP <60 **OR** > 30 mmHg drop • Pulse ≥ 100 If hemodynamics done: • Cardiac index ≥ 2.2 • PA Sat ≥ 65%	• Drugs/device used to maintain BP above stage B values If hemodynamics done: • Cardiac Index < 2.2 • RAP/PCWP > 0.8 • PCWP > 15 • PAPI < 1.85 • CPO ≤ 0.6	• Any of stage C **AND** requiring multiple pressors **OR** addition of mechanical circulatory support devices to maintain perfusion	• No SBP without resuscitation PEA **OR** Refactory VT/VF • Hypotensions despite maximal support

Adapted from the SCAI Clinical Expert Consensus Statement on the Classification of Cardiogenic Shock. Endorsed by ACC, AHA, SCCM, and STS.

Any attempt to improve outcomes in cardiogenic shock should begin with early identification.

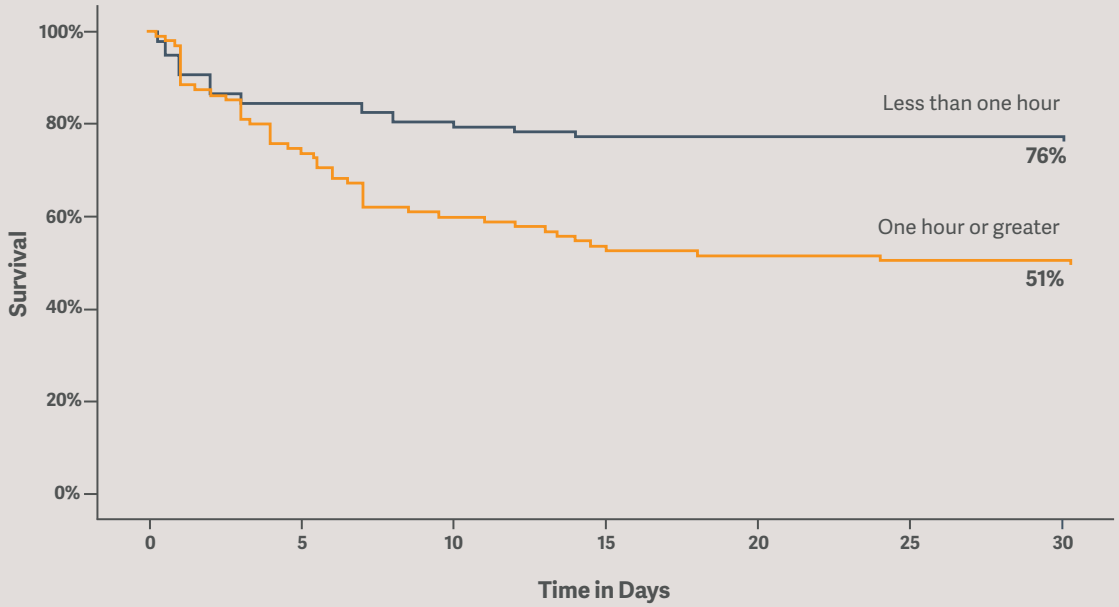
Models of care including a multi-disciplinary CS team hold potential for early identification and individualized treatment.[4]

Initiate early

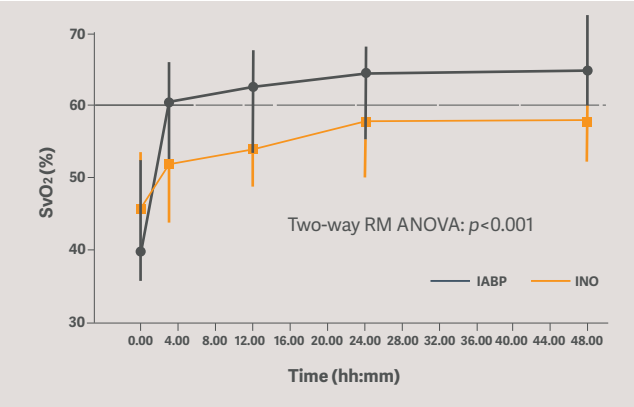
Retrospective analysis indicates early use of mechanical circulatory support (MCS) is an important therapeutic intervention.

Early use of intra-aortic balloon counterpulsation is associated with survival benefits, regardless of the etiology.[5]

30-day survival was 76% when IABP was placed within < 1 hour of onset of cardiogenic shock.[5]



Early initiation of IABP may provide hemodynamic benefit as primary treatment for advanced decompensated heart failure.[6]



Primary circulatory support with the Sensation Plus 50 cc IABP showed a significant increase in improved organ perfusion assessed by SVO_2.[6]

Secondary Endpoints	IABP (n=16)	Inotropes (n=16)	*P* value
Δ Cardiac power output	+0.27 (+0.17; +0.45)	+0.09 (-0.04; +0.21)	0.004
Δ NTproBNP	-59.3 (-78.5; -46.7)	-16.0 (-40.4; +3.3)	<0.001
Cumulative fluid balance	-3,066 (-3,876; -2,205)	-1,198 (-2,251; -70)	0.006
Δ Dyspnea severity score	-4 (-6; -3)	-2 (-3; 0)	0.02
MACE 90 days	6 (38%)	11 (69%)	0.16
90 day mortality	4 (25%)	9 (56%)	0.15

Starting support immediately reduces stroke work, possibly decreasing myocardial oxygen consumption.

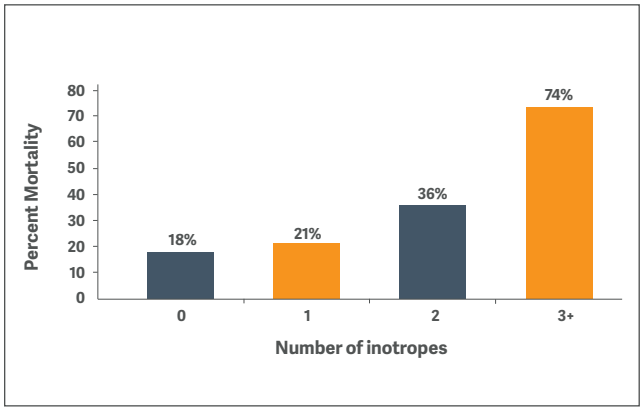
IABP counterpulsation decreases LV afterload, preload and intraventricular dyssynchrony.[6]

To avoid the negative impact of vasoactive drugs, consideration should be given to early initiation of IABP therapy.[5, 7]

Evaluate effectiveness

Tailor the care to the patient and escalate as needed.

Mortality with use of inotropes post-IABP[5]



Requirement for minimal pressors/ inotropes after IABP placement were predictors of lower mortality.

Escalation of therapy to devices that can offer greater hemodynamic support should be considered in patients requiring increasing inotropes after IABP placement.

Significant predictors of 30-day mortality on mulitvariate analysis[5]

Variables	Odds ratio	95% Confidence limits	*P* value
Age	1.07	1.03 – 1.10	0.001
Inotropes post-IABP	2.03	1.44 – 2.84	0.000
Time to IABP	1.05	1.01 – 1.09	0.009
Cardiopulmonary resuscitation	2.44	10.4 – 5.72	0.041

Identification of predictors of mortality would allow clinicians to tailor therapy and reserve use of more powerful MCS devices for patients that have more advanced stages of CS.[5]

Evaluating the response to therapy is critical in making adjustments to the plan of care.[3]

IABP: the safe first-line MCS option

Minimizing complications is critical to maximize the benefit of treatment.

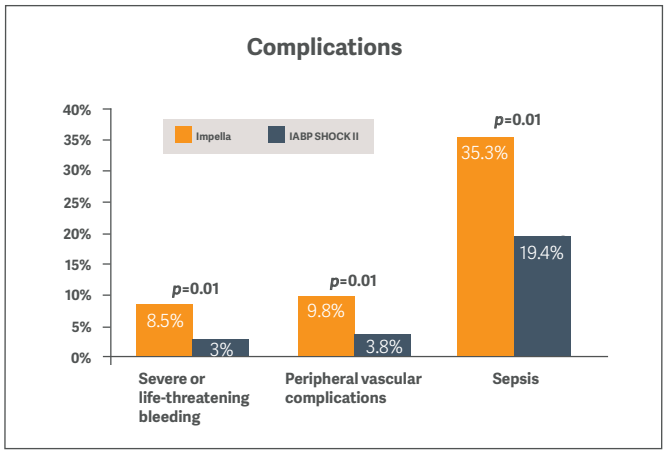
IABP is unique in its safety profile, cost efficiency and retains its position as the most widely used hemodynamic support device.[8]

Article	Number of patients	Mortality	Bleeding	Stroke	Vascular Complications
Dhruva 2019[9]	1680 Matched pairs from NCDR*	Favors IAB Absolute difference 10.9%	Favors IAB Absolute difference 15.4%	NA	NA
Amin 2019[10]	48,306 Premier database*	Favors IAB $p < 0.0001$	Favors IAB $p = 0.045$	Favors IAB $p < 0.0001$	NA
Wernly 2019[11]	588 Meta-analysis from 4 RCT**	No difference $p = 0.38$	Favors control $p = 0.002$	No difference $p = 1.00$	Favors control $p = 0.01$
Schrage 2019[12]	237 Matched pairs from IABP-Shock II**	No difference $p = 0.64$	Favors control $p < 0.01$	NA	Favors control $p = 0.01$

* Impella vs. IABP
** Impella vs. control (IABP and/or medical treatment)

Recent observational studies from large national, independent databases have shown a decrease in mortality, lower bleeding complications, lower stroke rates and a lower cost with IABP's compared to pVADS.[9, 10]

Complications matter



The use of IABP is associated with significantly fewer complications compared to pVADs.[12]

Cost of complications[13]

Outcome pVAD	With bleeding	Without bleeding	*P* value
In-hospital Mortality	166/469 (35.4%)	399/1346 (29.6%)	<0.001
Length of Stay	10 days (4-18)	6 days (2-12)	<0.001
Mean Health Care Cost	$79,518	$55,484	<0.001

No increased bleeding with IABP

Trial	IABP	No IABP	*P* value
CRISP AMI: major bleeding[14]	3.1%	1.7%	0.49
CRISP AMI: major vascular[14]	4.3%	1.1%	0.09
SHOCK II: moderate bleeding[15]	17.3%	16..4%	0.77
SHOCK II: major bleeding[15]	3.3%	4.4%	0.51
SHOCK II: major vascular[15]	4.3%	3.4%	0.53

Trial enrollment: CRISP AMI, n = 337; SHOCK II, n = 600

IABP therapy remains the predominant MCS device, a trusted, valuable first-line option[9, 10, 16]

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Identify

Emphasis should be on rapid identification of the patient's hemodynamic and critical care needs and deployment of appropriately tailored interventions.[5]

Initiate

Early placement of an appropriate MCS may be considered in those who fail to stabilize or show signs of improvement quickly after initial interventions.[17]

Evaluate

Similar to the collection, and analysis of battlefield intelligence, patients should be continually assessed, adjustments made, reassessed and readjusted.[18]

Escalate

If there is a need for increasing inotropes, consideration should be given to escalation of therapy to more invasive support devices.

Consideration for transfer to a facility with higher powered devices may be necessary.[5]

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