"Mechanical circulatory support in cardiogenic shock"
The Cardiologist's view

ACCA Masterclass 2017

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Disclosure of Interest

Pascal Vranckx has the following potential conflicts of interest to report:

Speaking or consulting fees from: AstraZeneca, Bayer Health Care and Daiichi-Sankyo.

outside this presentation.


T
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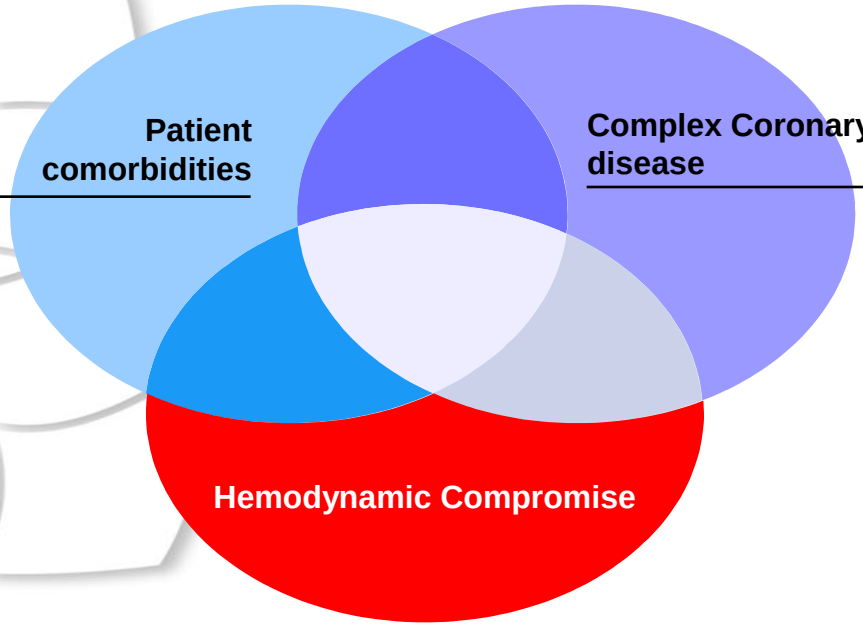
M

background

- Cardiogenic shock is the most severe form of acute heart failure.
- It is defined as pump failure despite adequate preload, leading to tissue hypoxia and organ dysfunction.
- Low mixed venous oxygen saturations and elevated lactate levels are surrogates for tissue hypoxia, while encephalopathy and low urine output indicate organ dysfunction.
- Patients with acute myocardial infarction complicated by acute heart failure or cardiogenic shock have high mortality with conventional therapy (7-10% /50%).

Pathophysiology

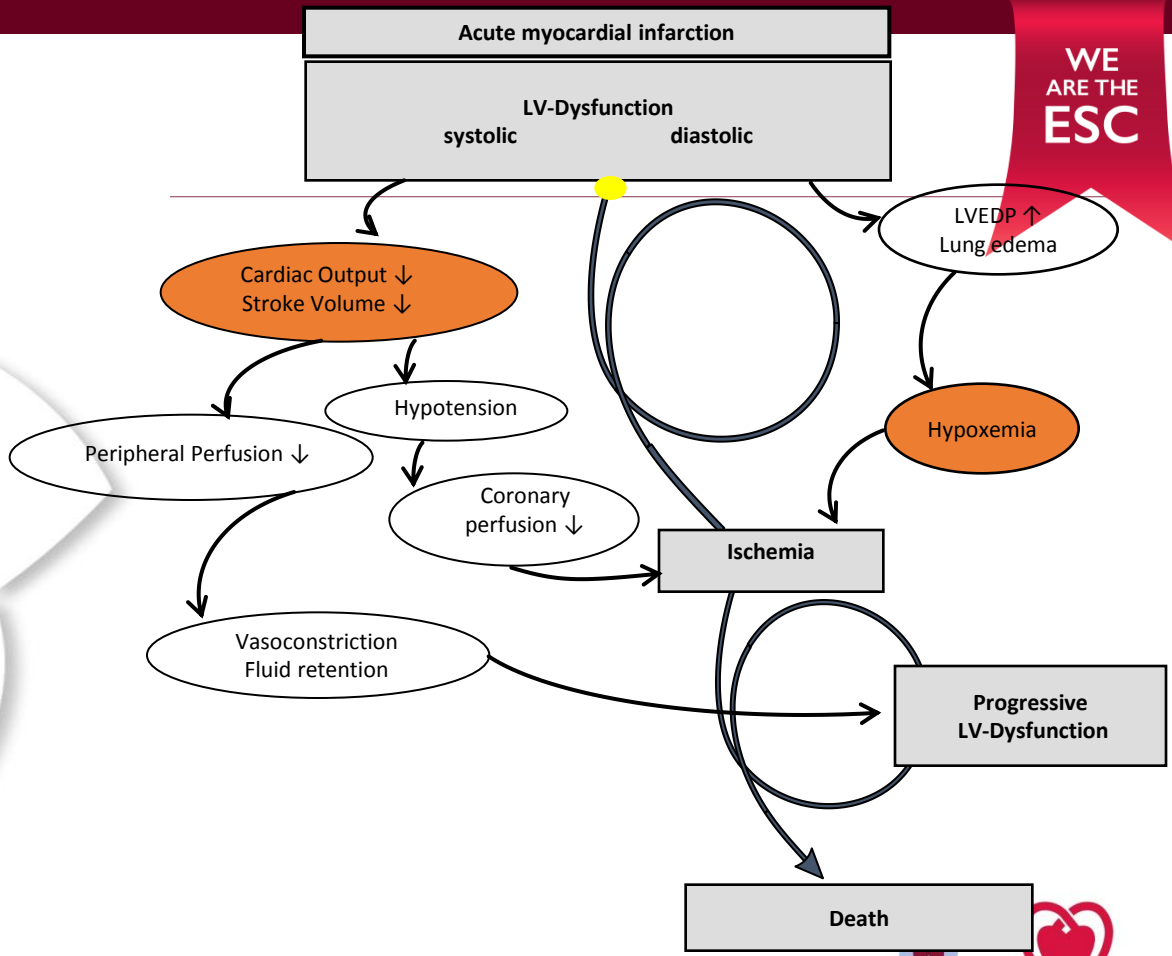
Diabetes
Advanced age
Peripheral vascular disease
Prior surgery



Multi vessel disease
Left main disease

Pathophysiology

Cardiogenic Shock Spiral



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Reynolds HR, Hochman JS. Circulation. 2008;117(5):686-97

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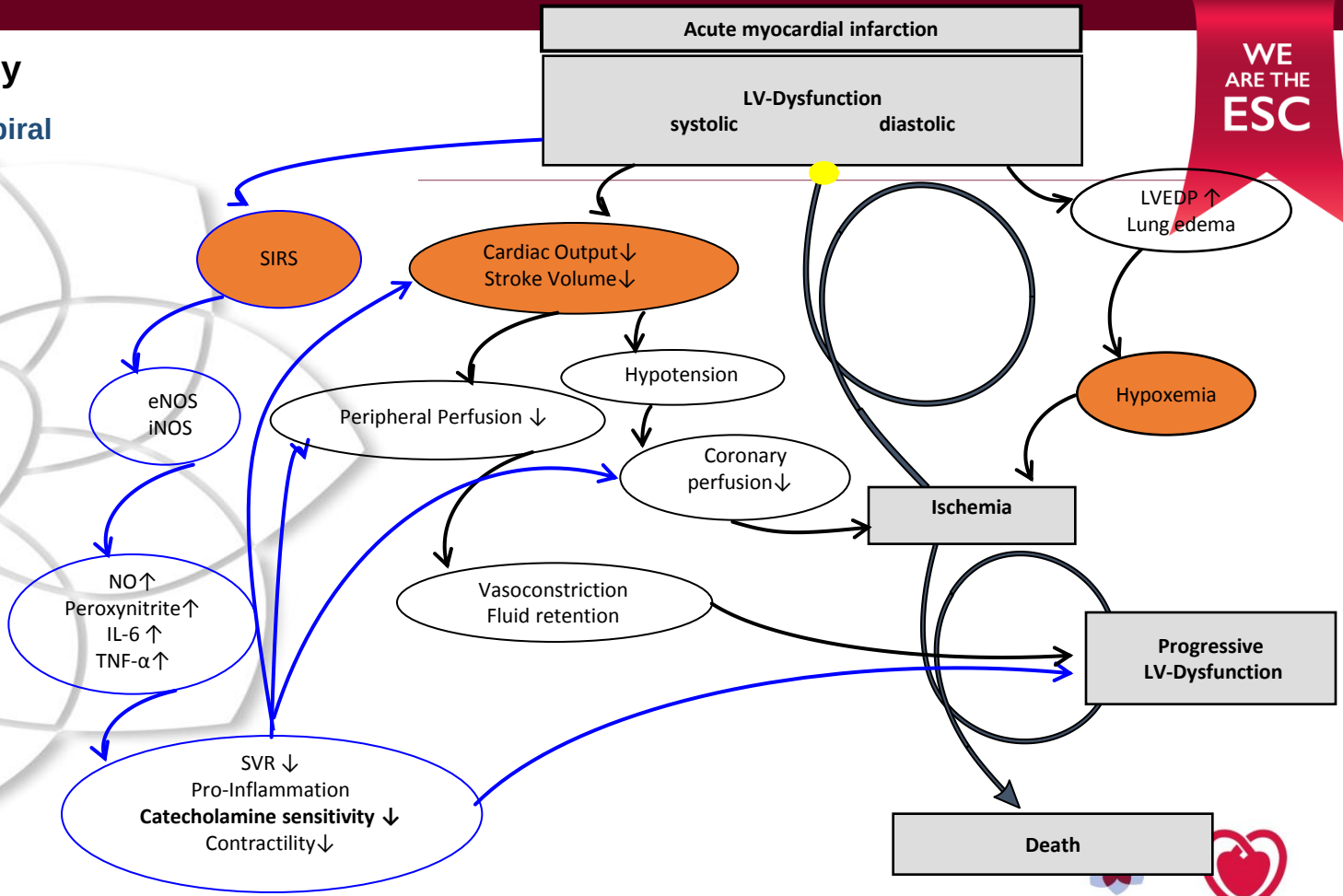
Pathophysiology

Cardiogenic Shock Spiral



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Pathophysiology

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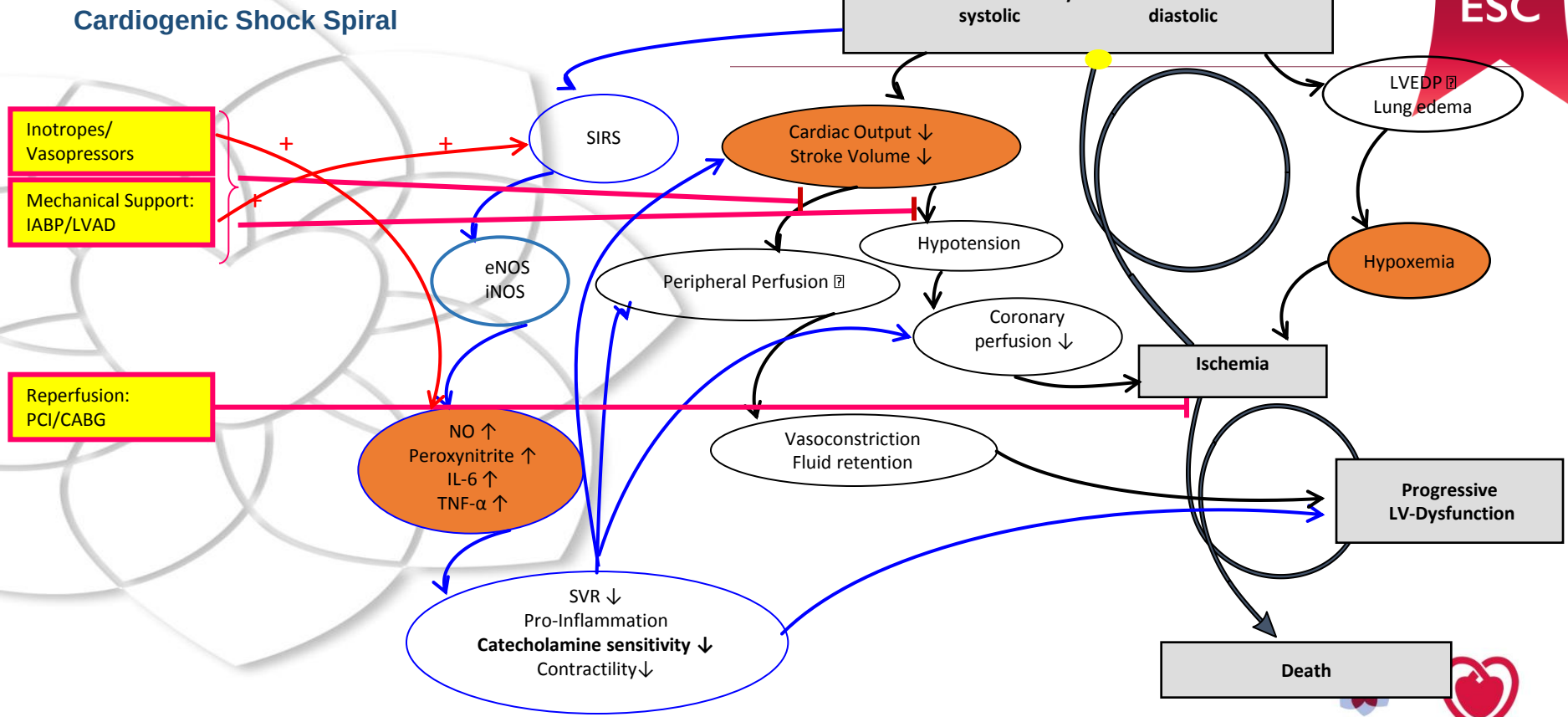

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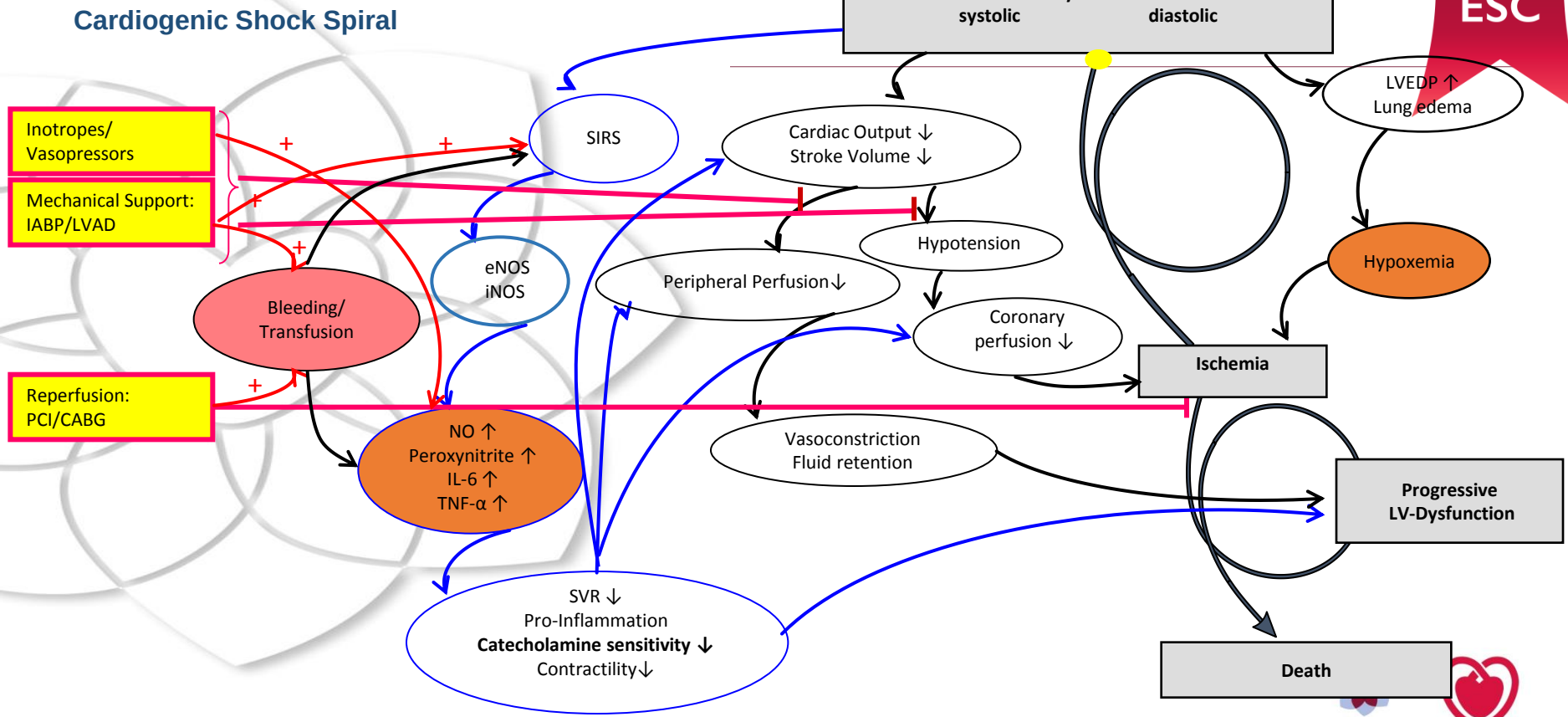
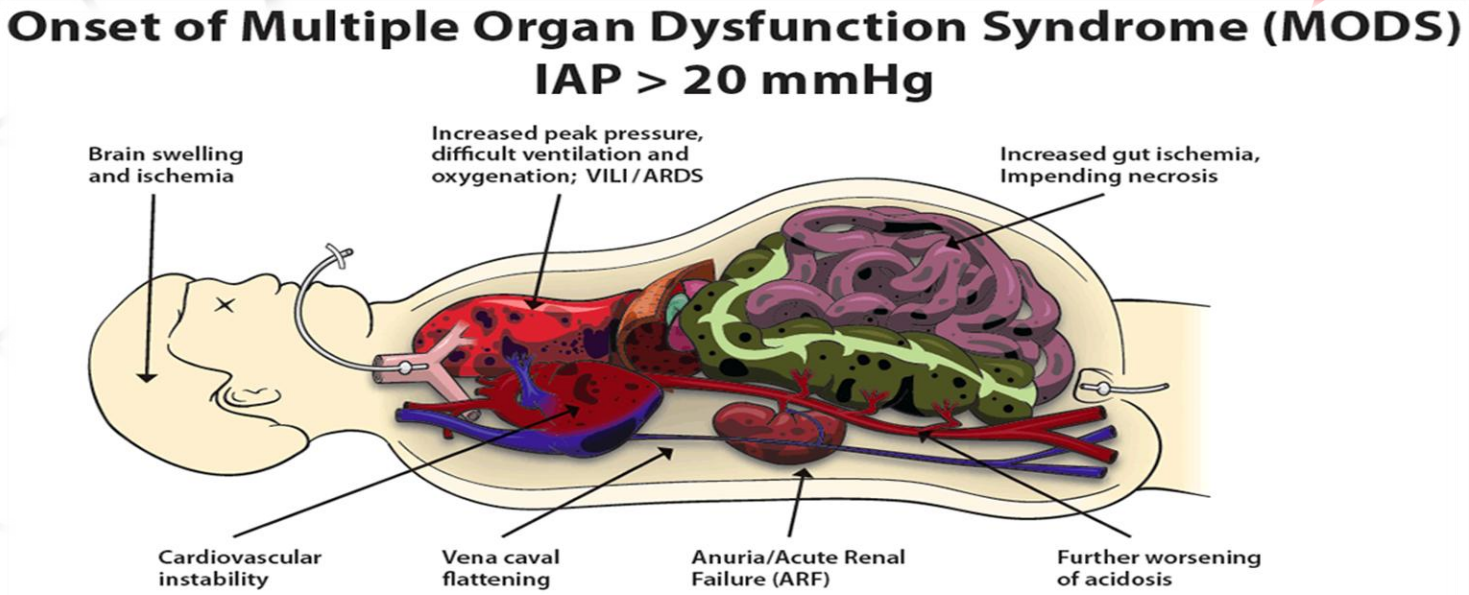
Pathophysiology

Cardiogenic Shock Spiral



Reynolds HR, Hochman JS. Circulation. 2008;117(5):686-97

Acute heart failure & shock is a 'sepsis like*' condition.

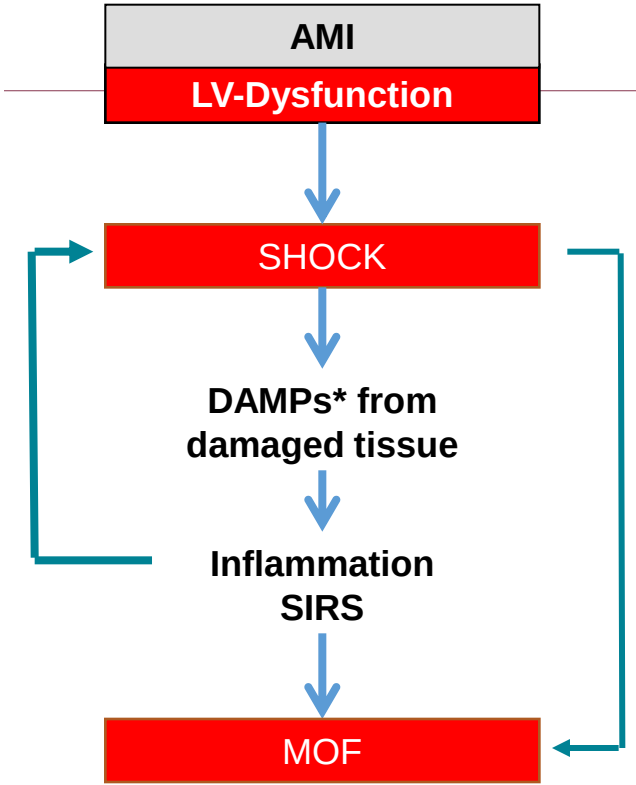


Underperfusion of the intestine and the hematogenous release of endotoxin in patients with HF has been proposed as a mechanism for progression of HF and CRS type 1

Pathophysiology

Cardiogenic Shock Spiral

Shock and SIRS can induce multiple organ failure (MOF), which eventually might cause death, if the condition cannot be reversed promptly by adequate treatment.

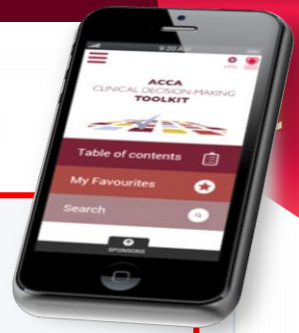


* Damage associated molecular patterns

Rudiger A. Understanding cardiogenic shock. Eur J Heart Failure. 2015;17(5):466-7.

CARDIOGENIC SHOCK: Initial triage and management

This protocol should be initiated as soon as cardiogenic shock/end organ hypoperfusion is recognised and should not be delayed pending intensive care admission.



EMERGENCY DEPARTMENT / CARDIAC INTENSIVE CARE UNIT

0 min

EARLY TRIAGE & MONITORING

Start high flow O_2
Establish i.v. access

- Age: 65–74, ≥75
- Heart rate >100 beats per minute
- Systolic blood pressure <100 mmHg
- Proportional pulse pressure ≤25 mmhg (CI <2.2l/min/m²)
- Orthopnea (PCWP >22 mmHg)
- Tachypnea (>20/min), >30/min (!)
- Killip class II-IV
- Clinical symptoms of tissue hypoperfusion/hypoxia:
  - cool extremities,
  - decreased capillary refill or mottling
  - decreased urine output (urine output <40 ml/h)
  - alteration in mental status

5 min

15 min

INITIAL RESUSCITATION

- Arterial and a central venous catheterization with a catheter capable of measuring central venous oxygen saturation
- Standard transthoracic echocardiogram to assess left (and right) ventricular function and for the detection of potential mechanical complications following MI
- Early coronary angiography in specialized myocardial intervention center when signs and/or symptoms of ongoing myocardial ischemia (e.g. ST segment elevation myocardial infarction).

- CORRECT: hypoglycemia & hypocalcemia,
- TREAT: sustained arrhythmias: brady- or tachy-
- Isotonic saline-fluid challenge of 20 to 30 ml per kilogram of body weight over a 30-minute period to achieve a central venous pressure of 8 to 12 mmHg or until perfusion improves (with a maximum of 500 ml)
- CONSIDER NIVmechanical ventilation for comfort (fatigue, distress) or as needed:
  - To correct acidosis
  - To correct hypoxemia
- INOTROPIC SUPPORT (dobutamine and/or vasopressor support)

60 min

TREATMENT GOALS

- a mean arterial pressure of 60 mmHg or above,
- a mean pulmonary artery wedge pressure of 18 mmHg or below,
- a central venous pressure of 8 to 12 mmHg,
- a urinary ouput of 0,5 ml or more per hour per kilogram of body weight
- an arterial pH of 7.3 to 7.5
- a central venous saturation ($ScvO_2$) ≥70% (provided SpO_2 ≥93% and Hb level ≥9 g/dl)

In persistent drug-resistant cardiogenic shock, consider mechanical circulatory support

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Pre-warned is *Pre*-armed

Risk Assessment

- Age
- Heart Rate > 100bpm
- Systolic Blood Pressure < 100mmHg
- Proportional Pulse Pressure ≤25 (CI < 2.2)*
- (if) Orthopnoe (PCWP > 22)
- KILLIP Class II-IV

* Stevenson LW et al. JAMA 1989; 261: 884-8

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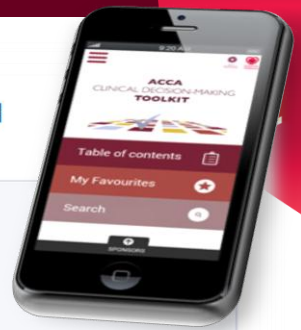

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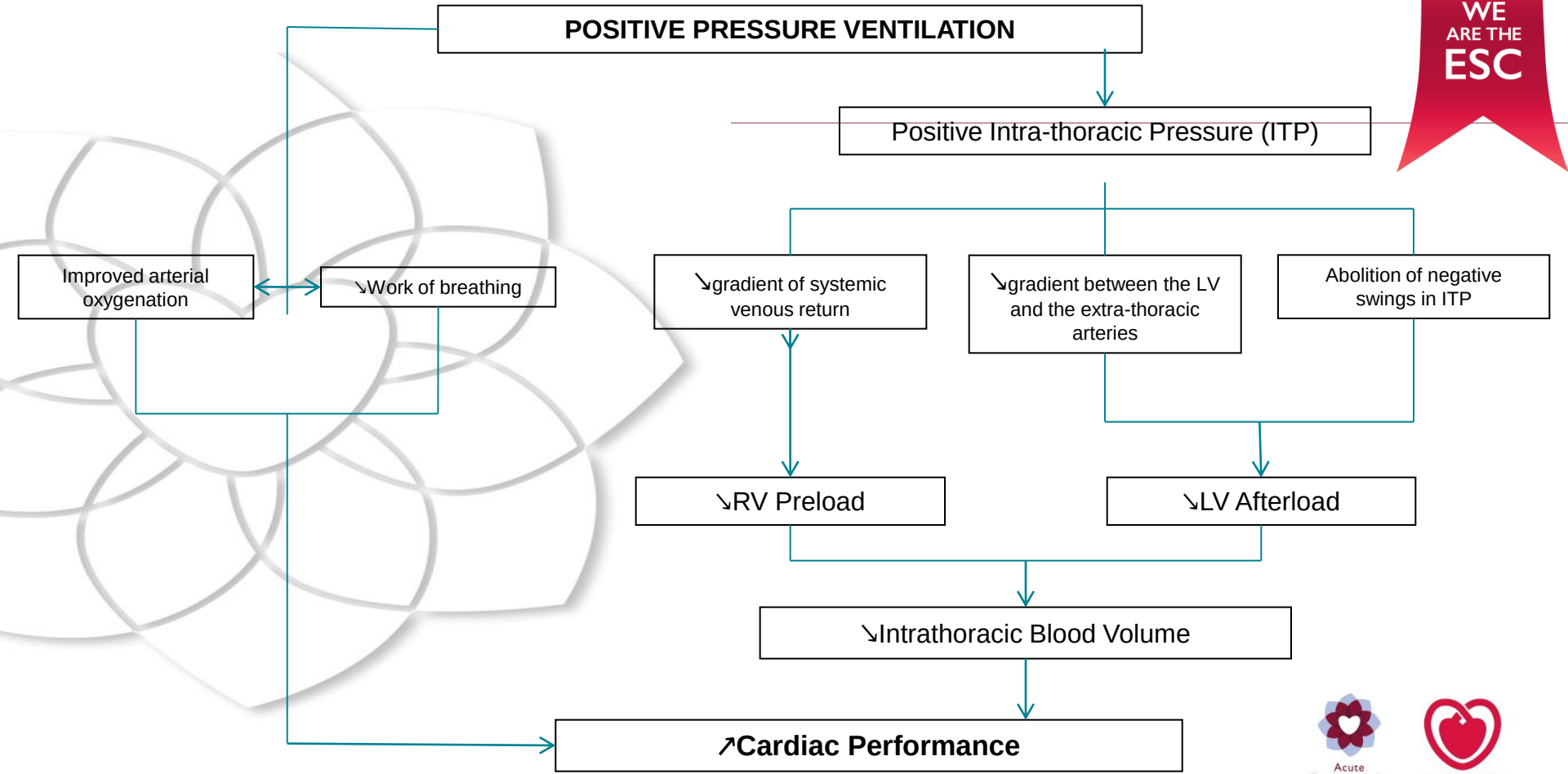
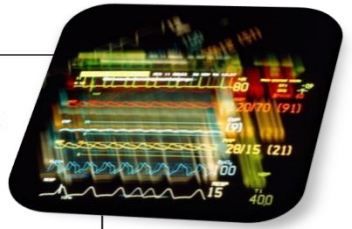

POSITIVE PRESSURE VENTILATION
Positive Intra-thoracic Pressure (ITP)
Improved arterial oxygenation
↘Work of breathing
↘gradient of systemic venous return
↘gradient between the LV and the extra-thoracic arteries
Abolition of negative swings in ITP
↘RV Preload
↘LV Afterload
↘Intrathoracic Blood Volume
↗Cardiac Performance

Assess volume status.
Treat sustained arrhytmias: brady- or tachy.
(Consider) Mechanical Ventilation for comfort (during PCI) and/or as needed:
- to correct accidaemia
- to correct hypoxaemia

Inotropic support (dobutamine and/or vasopressor support)



Signs (ST-segment elevation MI or New LBBB) and/or clinical symptoms of ongoing myocardial ischemia.

No

NSTE ACS, Delayed CS

Emergency echocardiography
± Tissue doppler imaging.
± Color flow imaging.

Yes

Short term Circulatory Support

Early coronary angiography
± Pulmonary Artery Catheter
± IABP *selected patients*
In a specialized Myocardial Intervention Center.

Pump failure
RV, LV, both.

- Acute severe mitral valve regurgitation
- Ventricle Septum Rupture
- Criticle Aortic/Mitral Valve Stenosis

Aortic dissection
Pericardial Tamponade

Operating theater ± coronary angiography

PCI ± stenting
of the culprit lesion

CABG
+ correct mechanical complications

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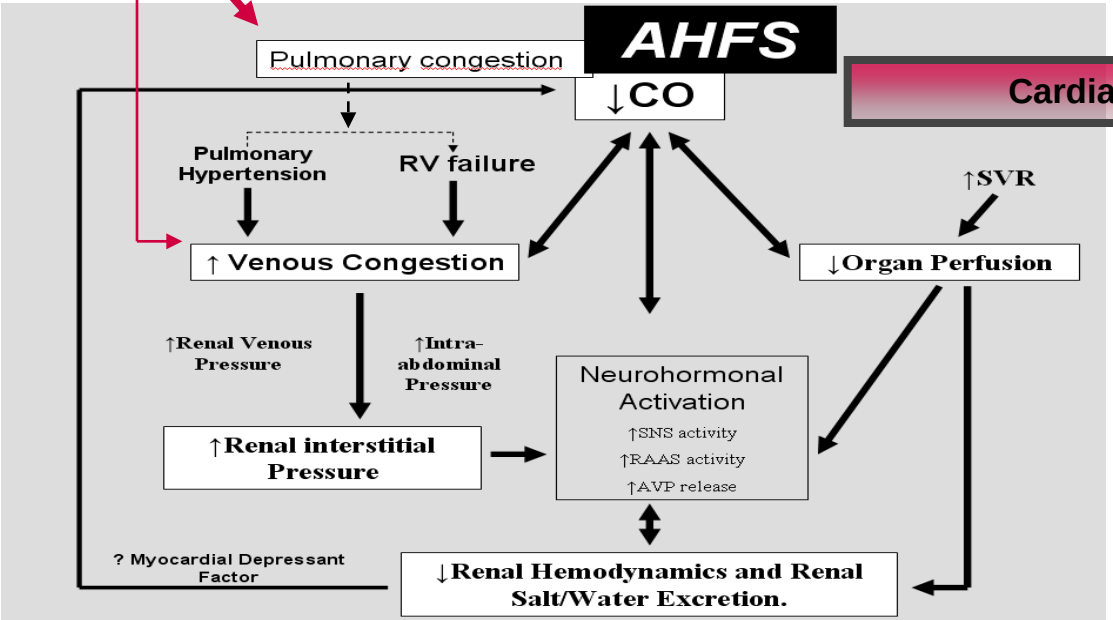
Mechanical ventilation

TV 6-8ml/kg

PAW <30mmHg

PEEP 3-5(-7) cmH_2O

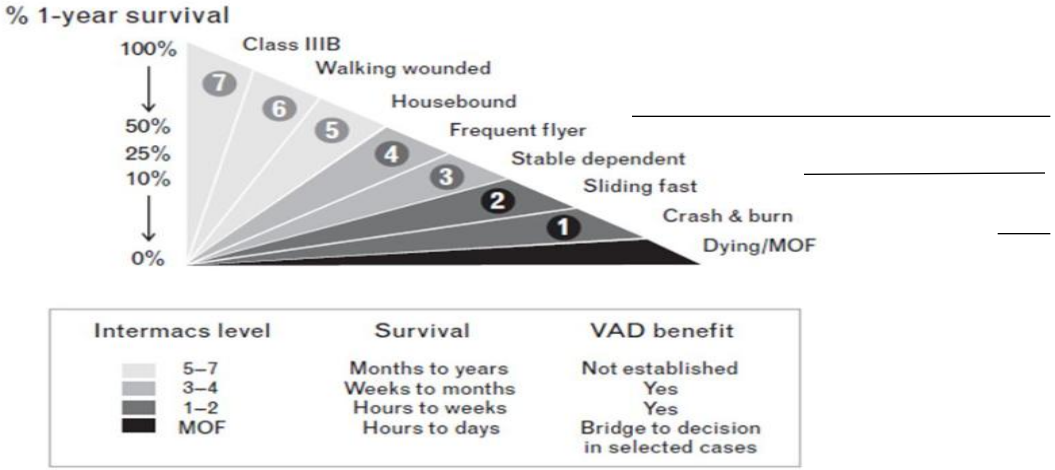
20 cpm, I:E 1/2



Cardiac Assist

Ultrafiltration ± dialysis

INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support) stages for classifying patients with advanced heart failure



Long Term VAD

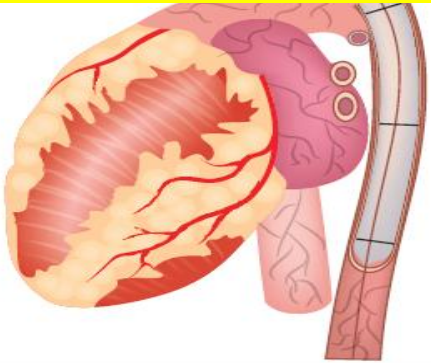
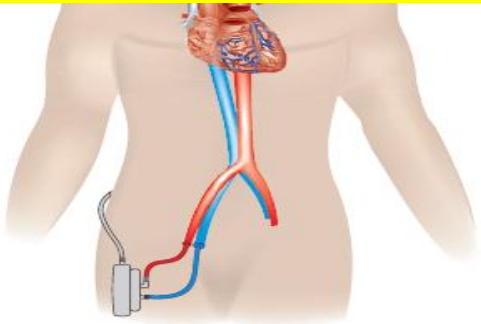
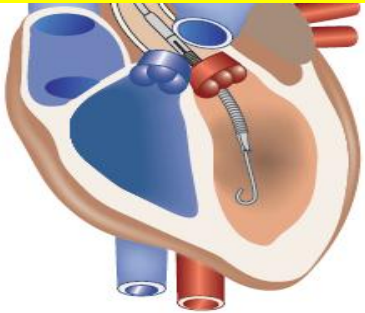
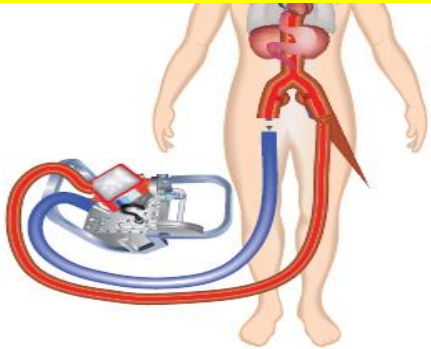
Short Term VAD

No VAD candidate: INTERMACS 1 or multi-system organ failure

INTERMACS level	NYHA Class	Description	Device	1y survival with LVAD therapy
1. Cardiogenic shock "Crash and burn"	IV	Haemodynamic instability in spite of increasing doses of catecholamines and/or mechanical circulatory support with critical hypoperfusion of target organs (severe cardiogenic shock).	ECLS, ECMO, percutaneous support devices	52.6±5.6%
2. Progressive decline despite inotropic support "Sliding on inotropes"	IV	Intravenous inotropic support with acceptable blood pressure but rapid deterioration of renal function, nutritional state, or signs of congestion.	ECLS, ECMO, LVAD	63.1±3.1%

Circulatory support systems for cardiogenic shock after ACS can be distinguished by:

- the method of placement (i.e. percutaneous vs. surgical),
- the type of circulatory support (i.e. left ventricular, right ventricular, or biventricular pressure and/or volume unloading), whether they are combined with gas exchange.

Mechanical LV support by LV pressure unloading	Mechanical LV support by LV volume unloading		Mechanical biventricular support with membrane oxygenation
			

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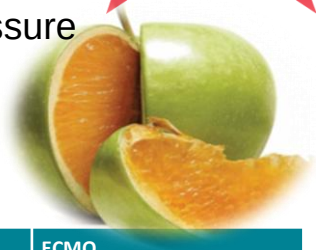
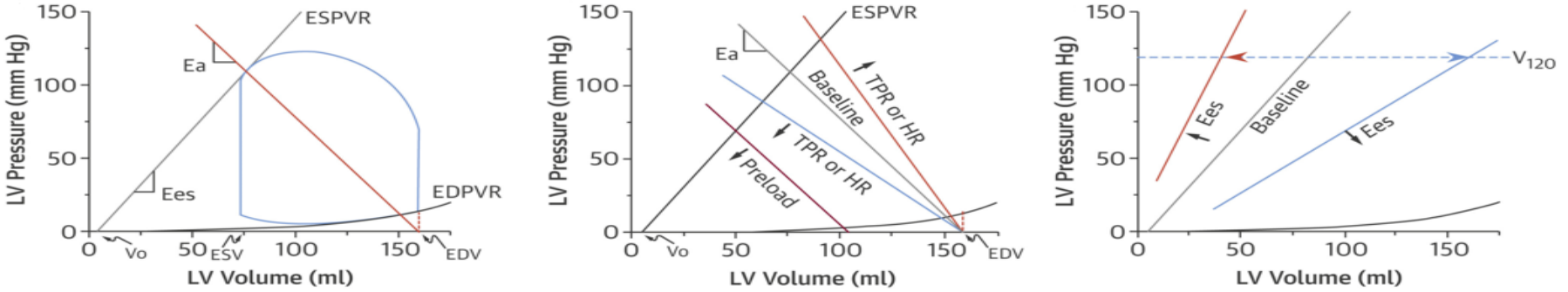


	TandemHeart™	Impella Recover® LP 5.0	Impella Recover® LP 2.5	Impella CP®	HeartMate PHP	ECMO
Catheter size (French)	–	9	9	9	-	–
Cannula size (French)	21 venous 12–19 arterial	21	12	14	14	17–21 venous 16–18 arterial
Flow (L/min)	Max 4.0	Max 5.0	Max 2.5	3.7–4.0	≈ 4,0	Max 7.0
Pump speed (rpm)	Max 7500	Max 33 000	Max 51 000	Max 51 000		Max 5000
Insertion/placement	Percutaneous (femoral artery plus LA after trans-septal puncture)	Peripheral surgical cut-down (femoral artery)	Percutaneous (femoral artery)	Percutaneous (femoral artery)	Percutaneous (femoral artery)	Percutaneous (femoral artery plus vein)
Recommended duration of use	–14 days	10 days	10 days	10 days		–7 days

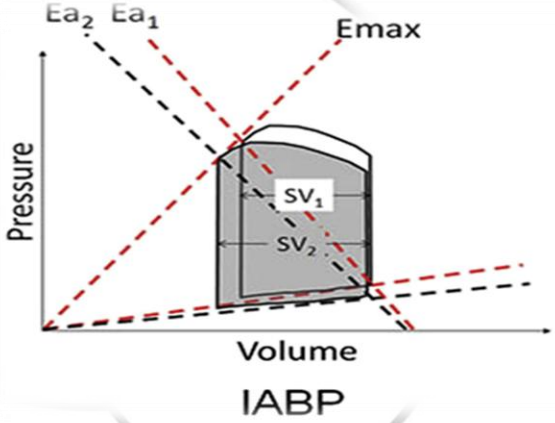
Fundamentals of Left Ventricular Mechanics



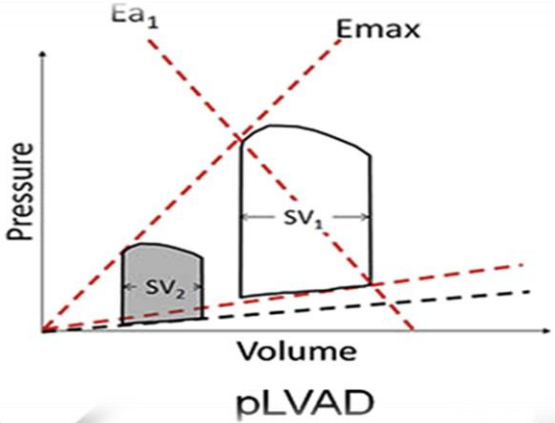
Normal pressure–volume loop (PVL), is bounded by the end-systolic pressure–volume relationship (ESPVR) and end-diastolic pressure–volume relationship (EDPVR).

J Am Coll Cardiol. 2015;66(23):2663-2674. doi:10.1016/j.jacc.2015.10.017

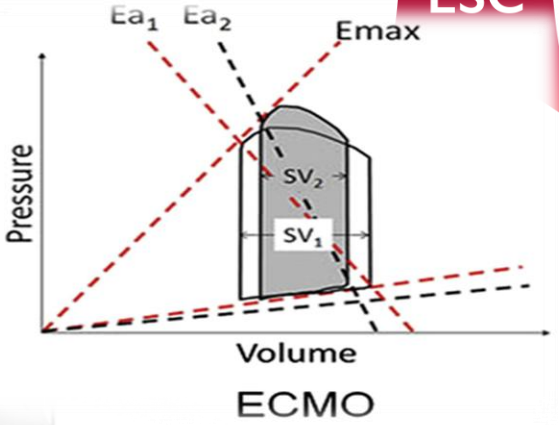
Potential benefits of Mechanical Circulatory Support Systems.



IABP



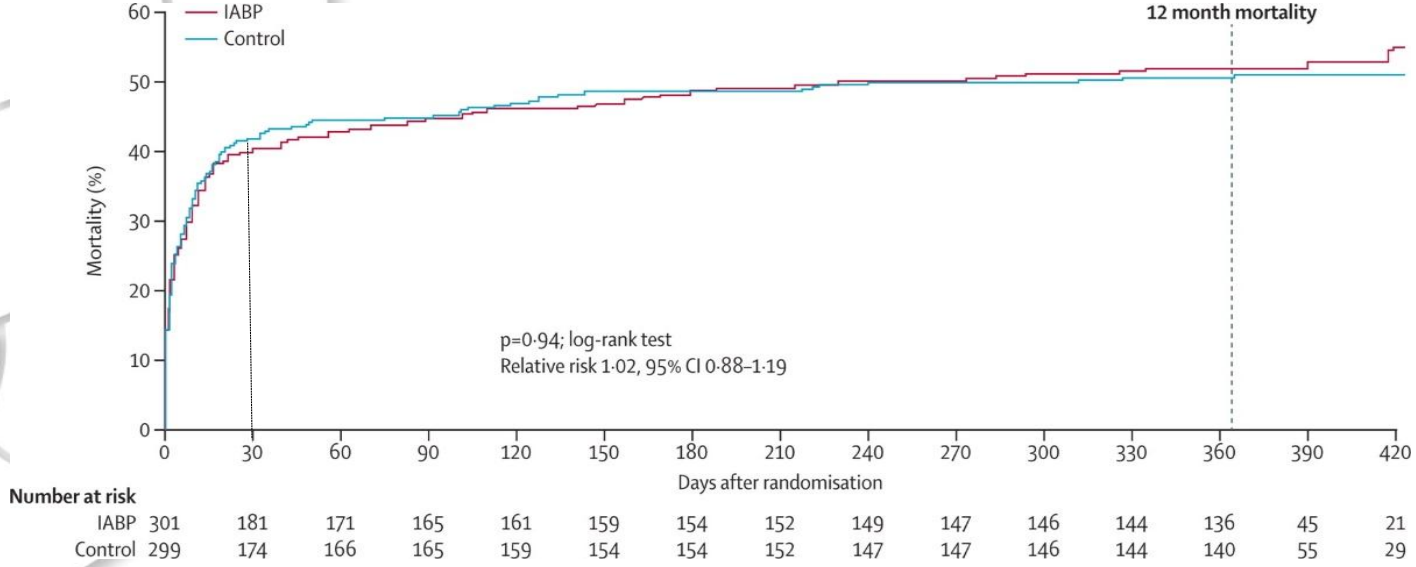
pLVAD



ECMO

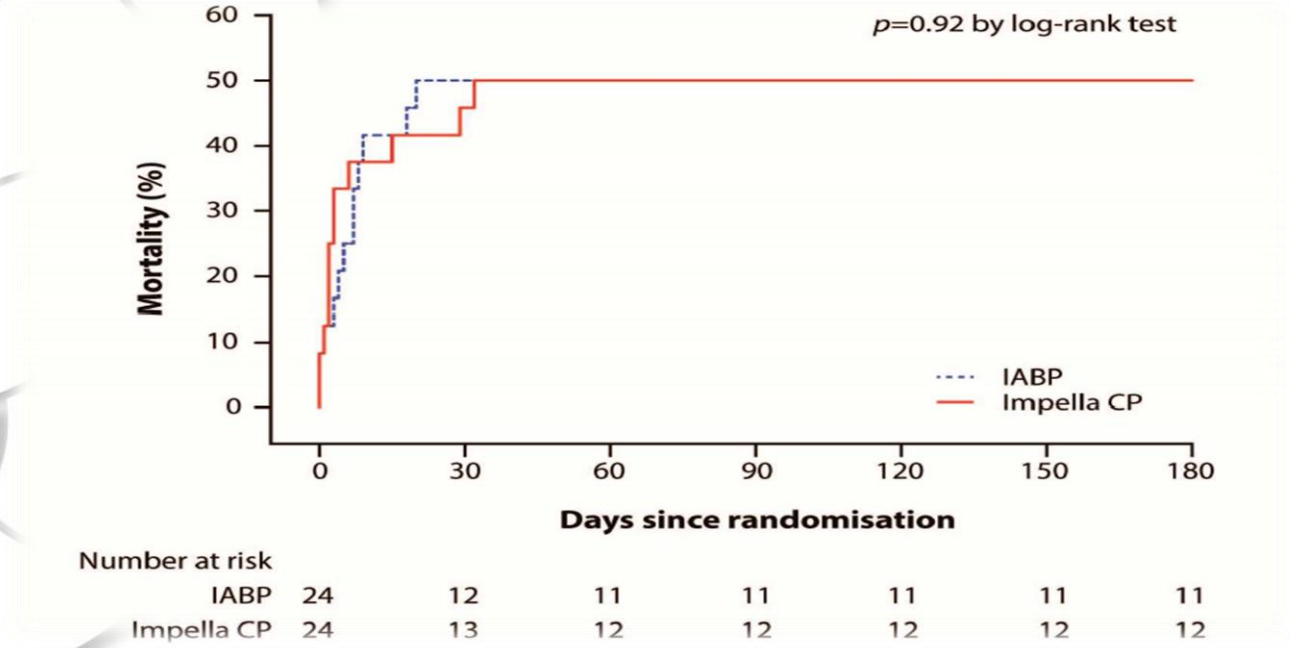
- **maintain vital organ perfusion**, thereby preventing systemic shock syndrome,
- reduce intra-cardiac filling pressures, thereby reducing congestion and/or pulmonary edema,
- reduce left ventricular volumes, wall stress, and myocardial oxygen consumption.

The use of intra-aortic balloon counterpulsation did not significantly reduce 30-day or 1 year mortality in patients with cardiogenic shock complicating acute myocardial infarction for whom an early revascularization strategy was planned.



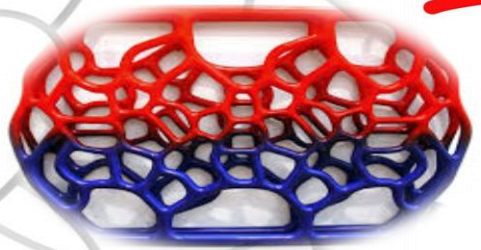
Thiele H. et al. N Engl J Med 2012; 367:1287-1296 / Lancet 2013 382, 1638-1645

In the IMPRESS-trial, a small (n=48) explorative randomized controlled involving mechanically ventilated cardiogenic shock patients after acute myocardial infarction, routine treatment with Impella CP was not associated with reduced 30-day mortality compared with IABP.



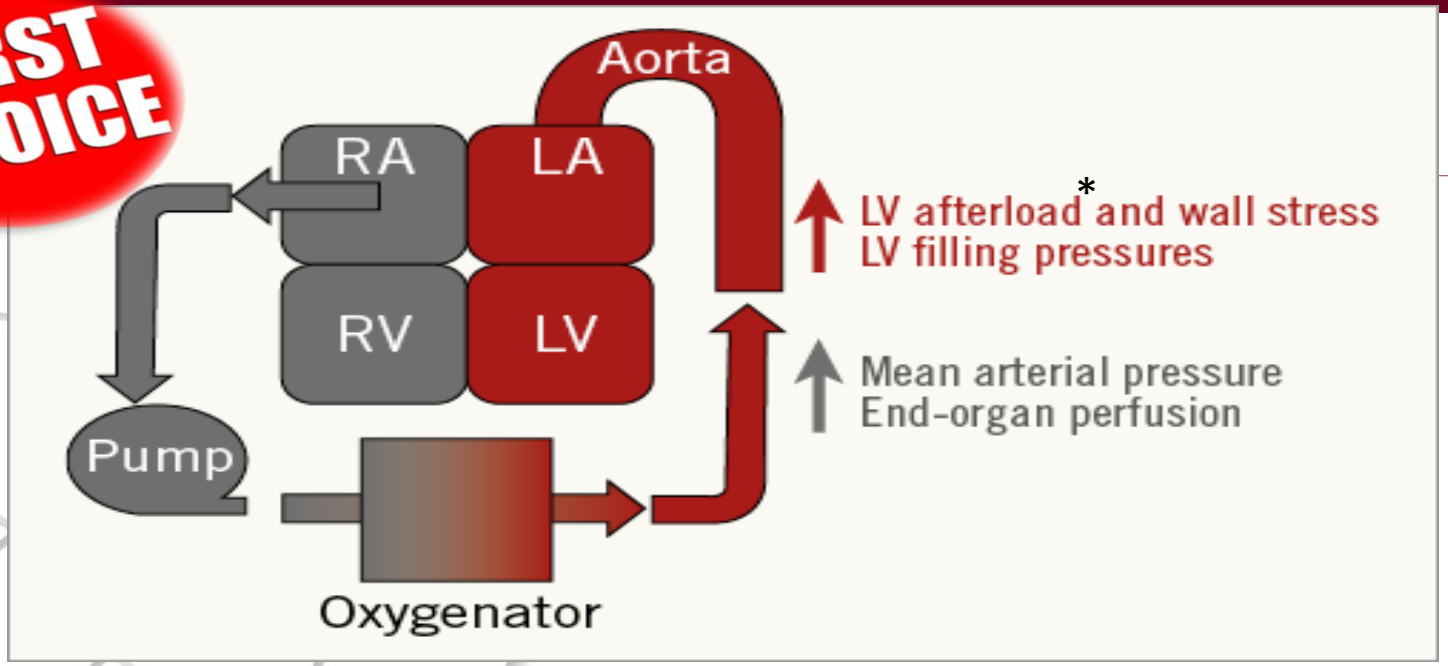
J Am Coll Cardiol. 2016;():. doi:10.1016/j.jacc.2016.10.022

KNOW THE RULES!

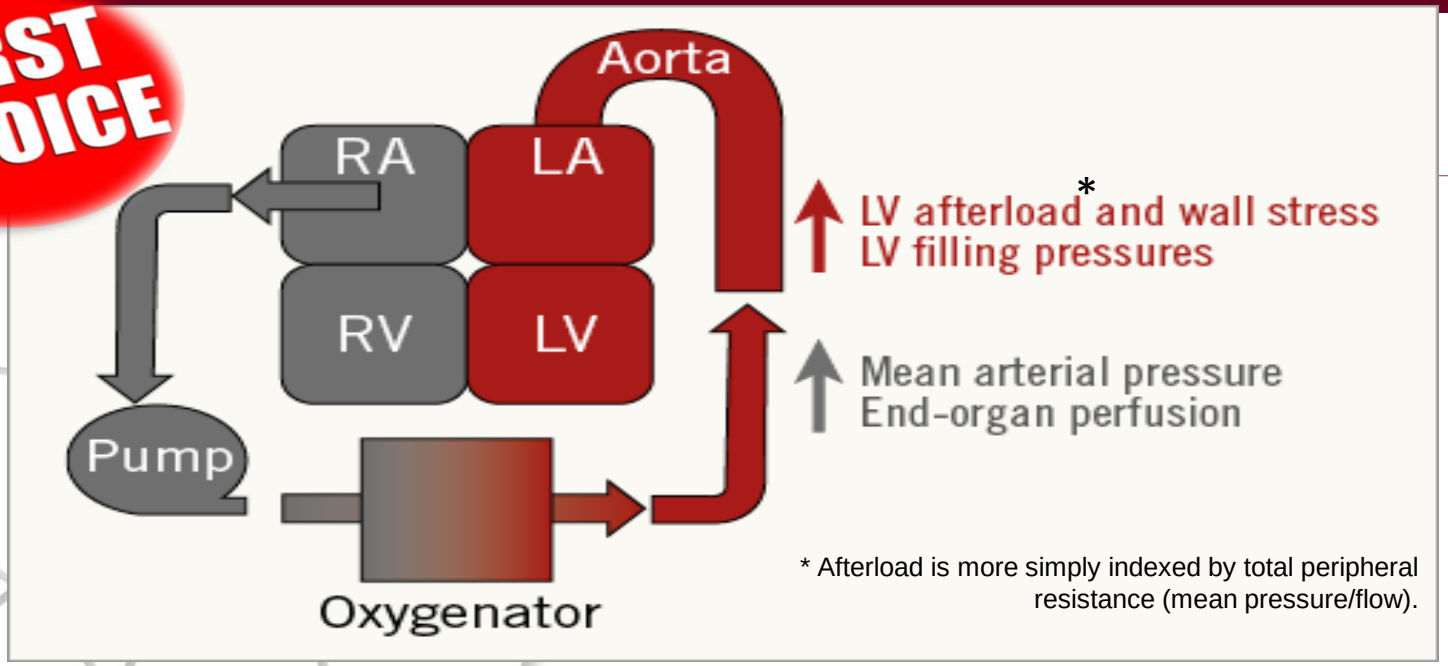


70

ml/kg body weight *to* achieve S_aO_2>80%

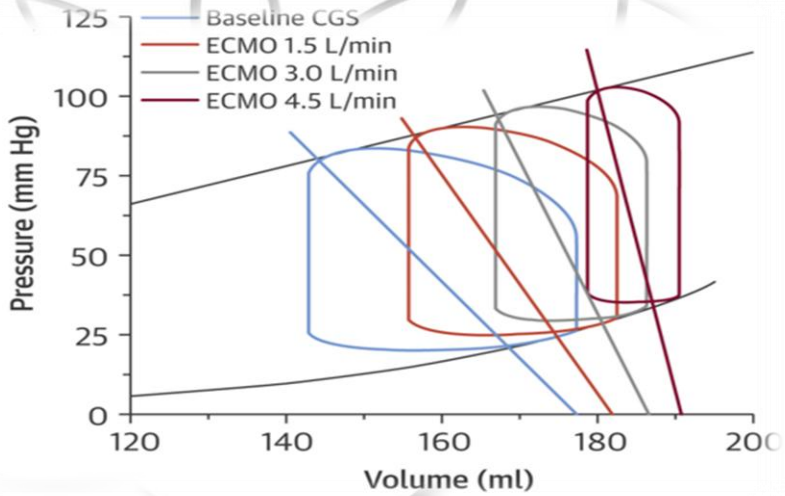


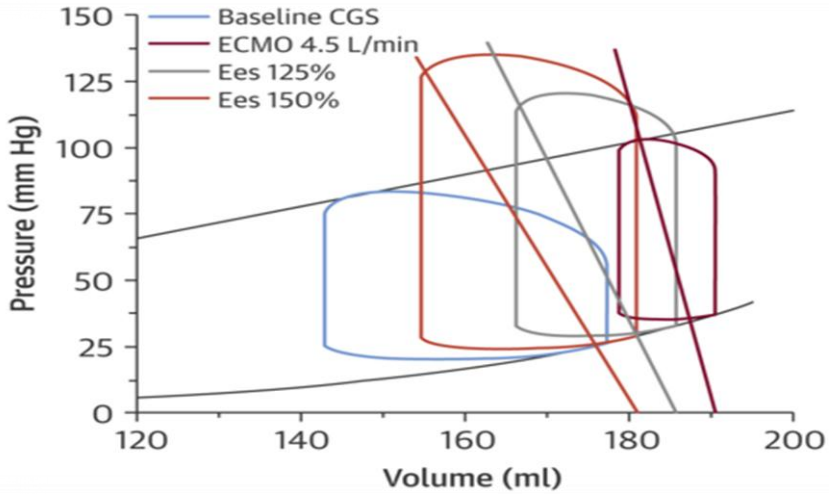
* Afterload is more simply indexed by total peripheral resistance,the ratio mean pressure/flow.



However, strictly on a hemodynamic basis, the use of this circuit configuration can cause **significant increases in LV pre-load** and, in some cases, pulmonary edema.

Impact of extracorporeal membrane oxygenation (ECMO) on pressure–volume loops in patients with cardiogenic shock.





Short-term improvements in LV function can also modulate the rise in PCWP.

J Am Coll Cardiol. 2015;66(23):2663-2674. doi:10.1016/j.jacc.2015.10.017

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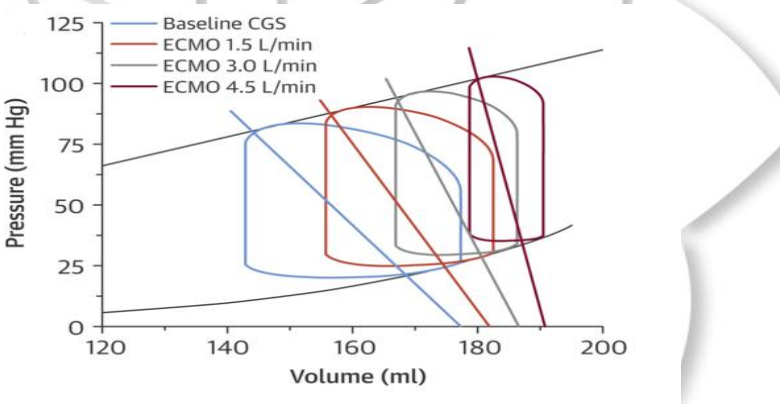

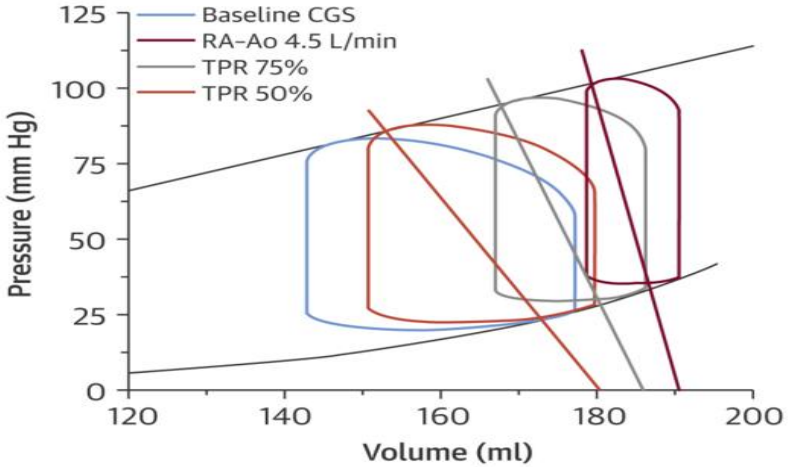

TPR can be reduced naturally by the baroreceptors, pharmacologically (e.g., nitroprusside), or mechanically (e.g., by IABP).

J Am Coll Cardiol. 2015;66(23):2663-2674. doi:10.1016/j.jacc.2015.10.017

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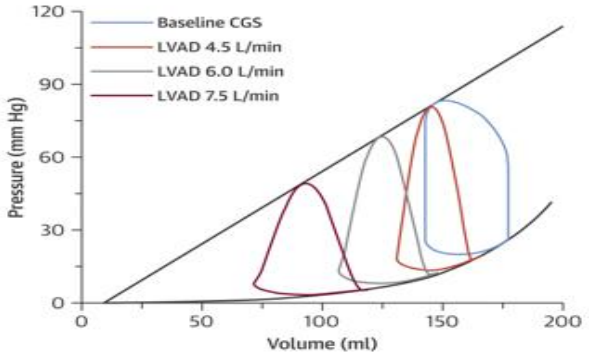
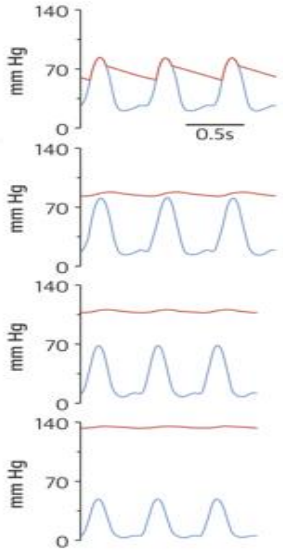
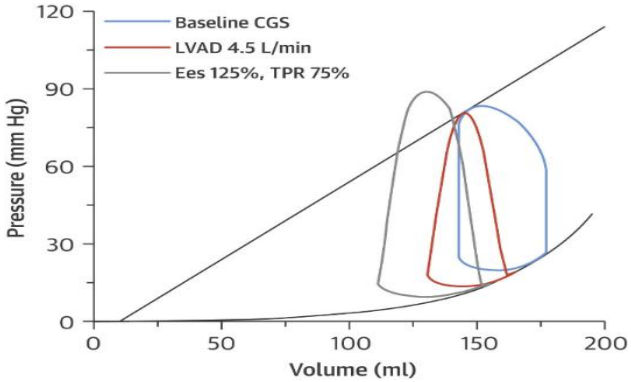
When secondary factors are insufficient to self-mitigate a rise in LV EDP, other strategies may be utilized to reduce possible increases in afterload pressure and allow for LV decompression. These include:

- atrial septostomy (to allow left-to-right shunting),
- a surgically placed LV vent,
- an intra-aortic balloon pump, or use of a percutaneous LV-to-aorta ventricular-assist device (**i.e. axial flow device**)

Flow-dependent changes of the pressure-volume loop (triangular) with LV-to-aortic pumping







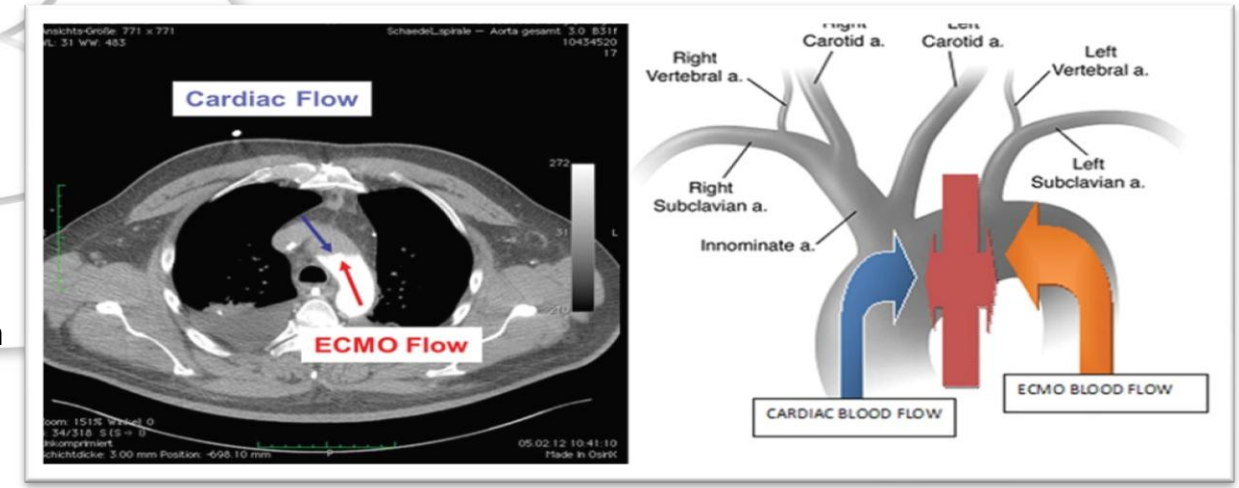
With increased flow, there are greater degrees of LV unloading and uncoupling between aortic and peak LV pressure generation

J Am Coll Cardiol. 2015;66(23):2663-2674. doi:10.1016/j.jacc.2015.10.017

The Harlequin (north-south) syndrome.

Femoral veno-arterial extracorporeal membrane oxygenation may cause differential hypoxia (lower P_aO_2 in the upper body than in the lower body, i.e., two-circulation syndrome) because of **normal cardiac output** with **severe impairment of pulmonary function**

Hypoxic arterial blood gas when saturations in the right radial artery are measured.



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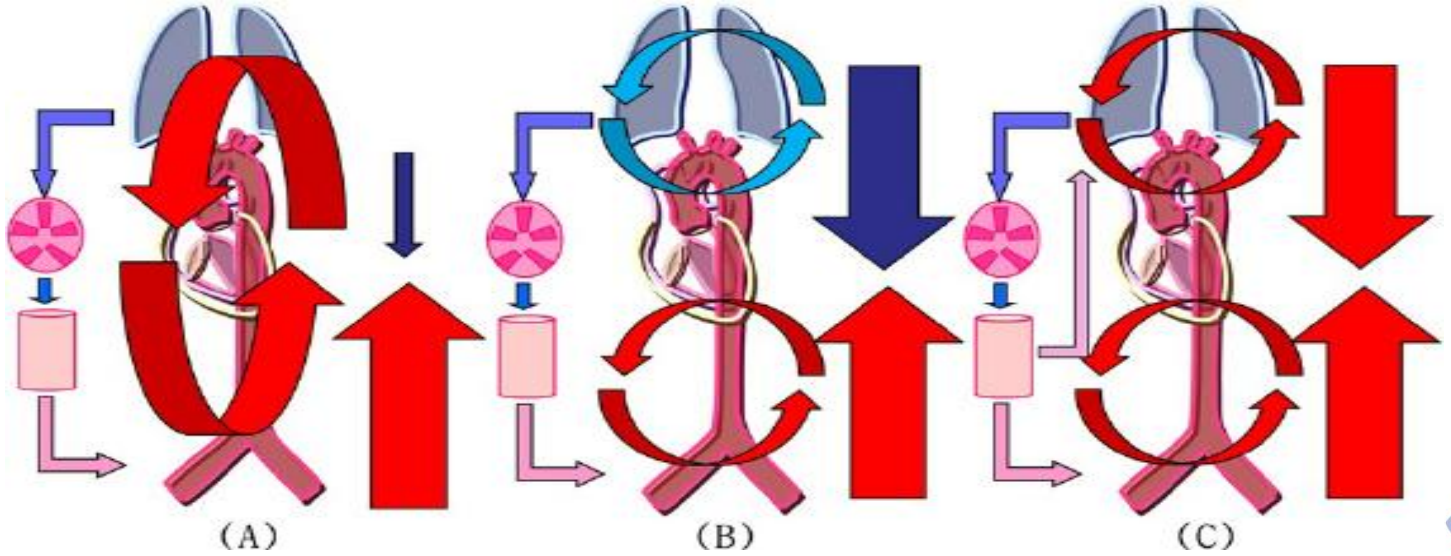
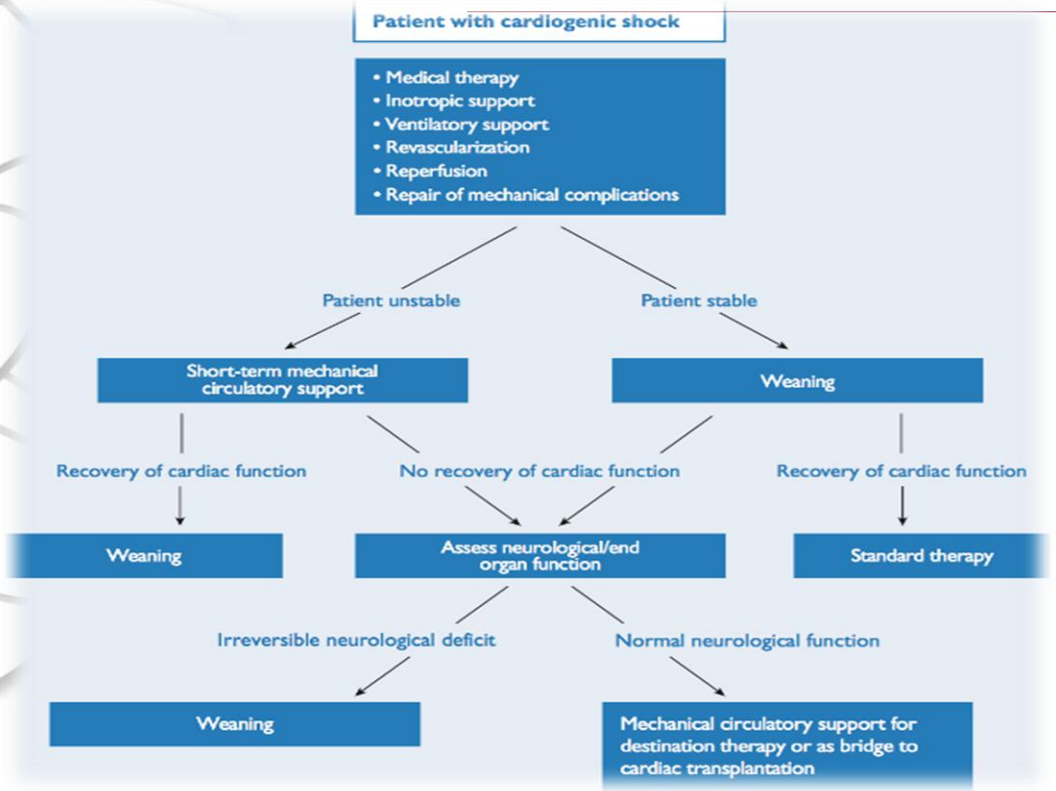
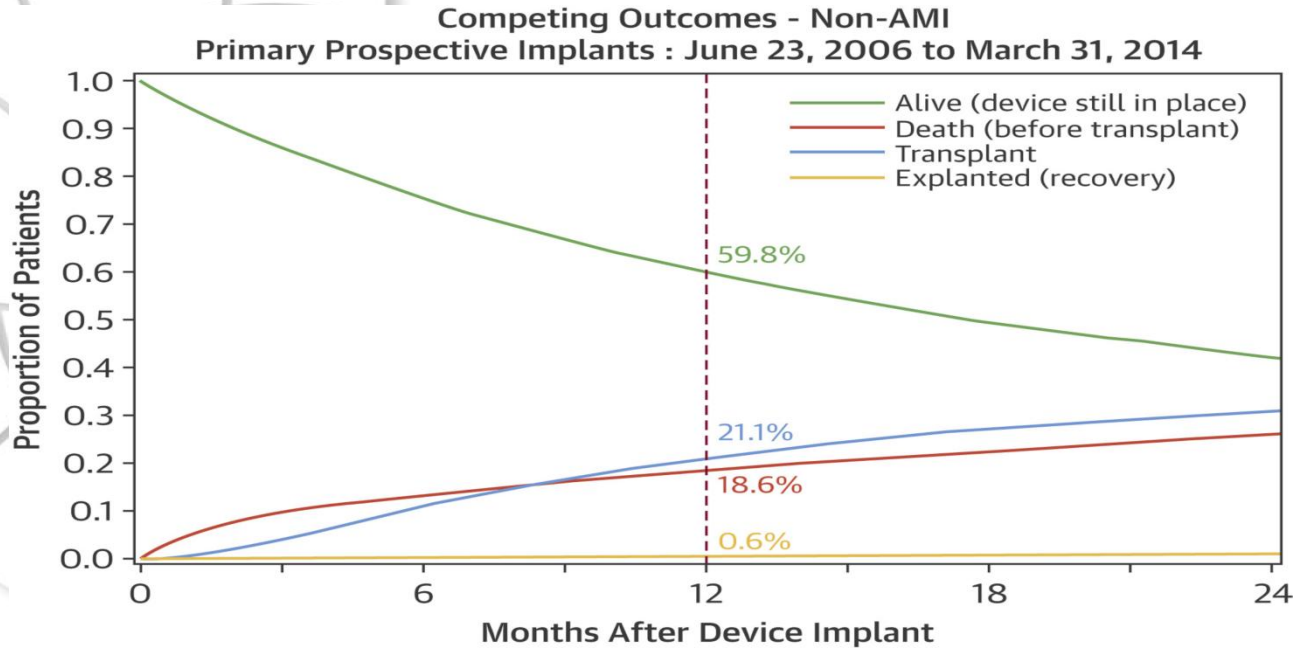


Circulatory support systems for cardiogenic shock after ACS are never used in a vacuum.



SHORTER DURATION OF HEART FAILURE AT LVAD IMPLANTATION MITIGATES SOME OF THE RISKS OF HIGH ACUITY.



Intermacs
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J Am Coll Cardiol. 2016;67(16):1871-1880. doi:10.1016/j.jacc.2016.02.025

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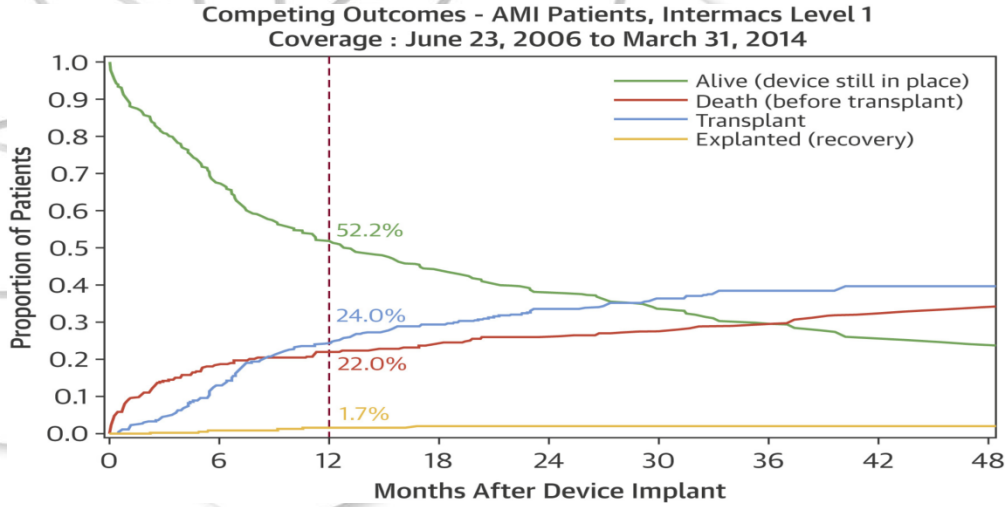
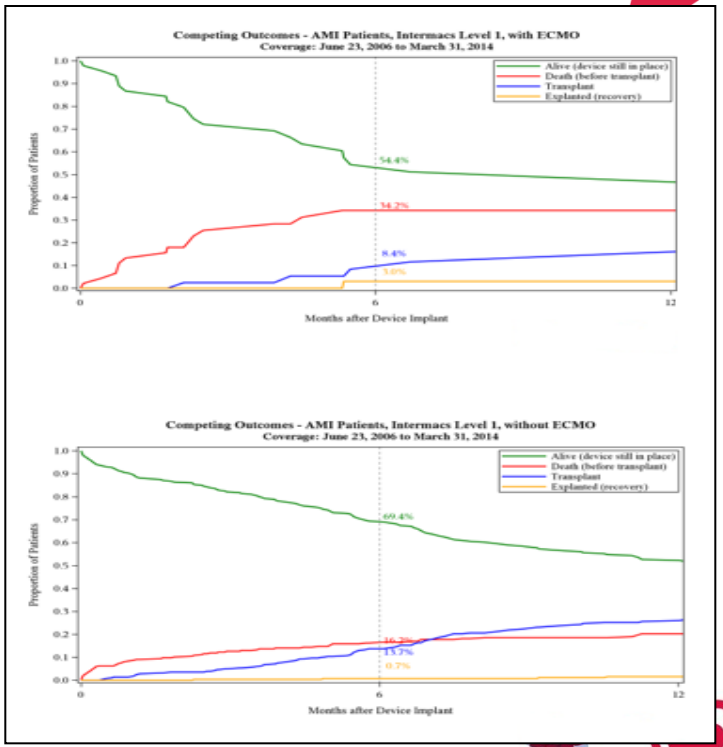
AMI PATIENTS HAVE THE SAME 1YR-OUTCOME, DESPITE BEING MORE CRITICALLY ILL PRE-IMPLANTATION.





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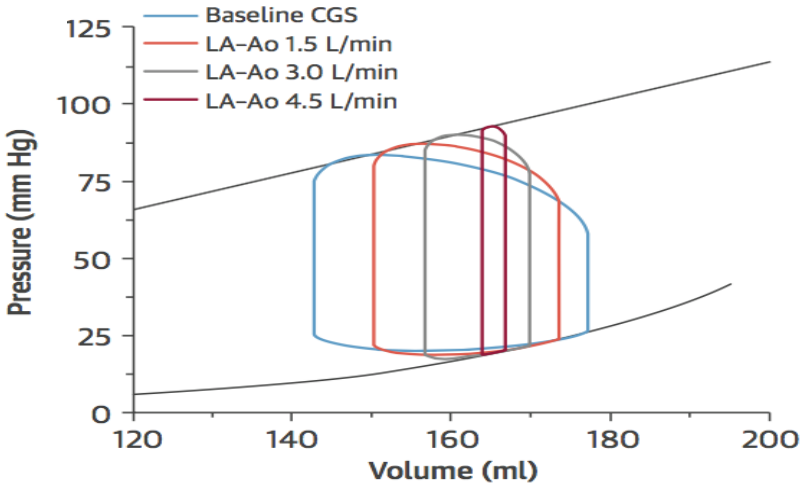
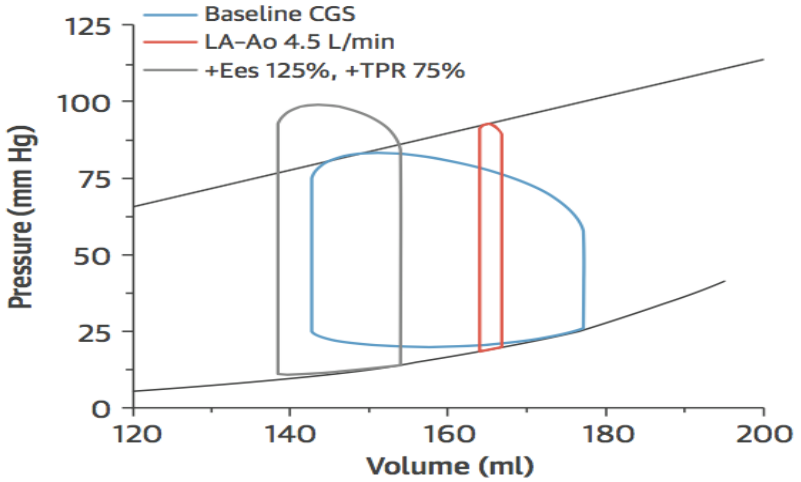
ADVERSE EVENTS

	Early Period				Late Period			
	AMI Rate (n = 502) (Per 100 Patient-Months)	Non-AMI Rate (n = 9,727) (Per 100 Patient-Months)	Rate Ratio	p Value	AMI Rate (n = 502) (Per 100 Patient-Months)	Non-AMI Rate (n = 9,727) (Per 100 Patient-Months)	Rate Ratio	p Value
Bleeding	30.30	19.46	1.6	<0.01	3.02	3.43	0.9	0.10
Cardiac arrhythmia	16.20	10.96	1.5	<0.01	0.47	1.09	0.4	<0.01
Hemolysis	3.47	2.12	1.6	<0.01	0.92	0.69	1.3	0.04
Infection	24.12	16.50	1.5	<0.01	4.84	5.10	0.9	0.39
Myocardial infarction	0.38	0.12	3.2	0.01	0.09	0.03	3.0	0.02
Neurological dysfunction	6.48	4.05	1.6	<0.01	1.49	1.24	1.2	0.09
Other SAE	20.27	12.95	1.6	<0.01	1.94	1.95	1.0	0.99
Rehospitalization	16.88	20.46	0.8	<0.01	14.18	16.28	0.9	<0.01
Renal dysfunction	5.58	4.02	1.4	0.01	0.35	0.51	0.7	0.09
Respiratory failure	12.81	7.44	1.7	<0.01	0.42	0.52	0.8	0.3
Right heart failure	5.50	6.27	0.9	0.275	0.21	0.53	0.4	<0.01
Venous thromboembolism	3.32	1.75	1.9	<0.01	0.05	0.08	0.7	0.48

J Am Coll Cardiol. 2016;67(16):1871-1880. doi:10.1016/j.jacc.2016.02.025

Ventricular Effects of LA-to-Arterial MCS on pressure–volume loops in patients with cardiogenic shock.





J Am Coll Cardiol. 2015;66(23):2663-2674. doi:10.1016/j.jacc.2015.10.017

When you don't have full access to these tools…?

BELIEVE IN

GOD

NEVER PANIC

JUST

PRAY

Conclusions

- The number of patients with advanced heart failure that has become unresponsive to conventional medical therapy is increasing rapidly.
- No other field in cardiology is experiencing such explosive growth as mechanical circulatory support for advanced heart failure (HF).
- To date, there are no guidelines for appropriate selection for use of these devices that are approved by national societies in the field.

Conclusions

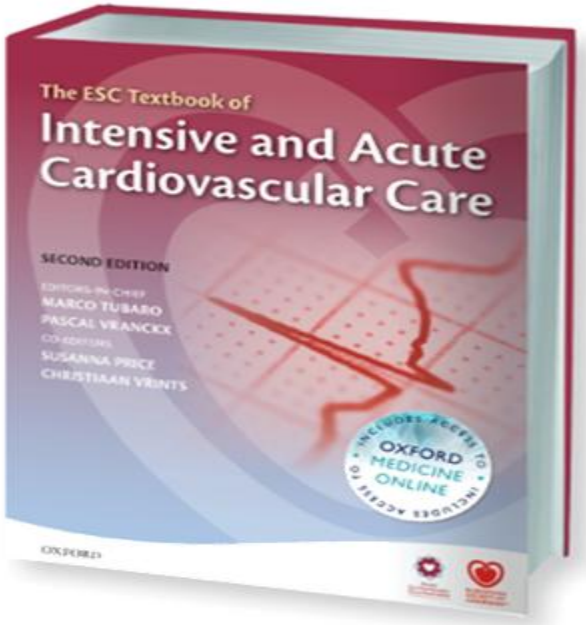
- Treatment options for mechanical circulatory support must be tailored to each patient in order to maximize the potential benefits and minimize the risk of detrimental effects.
- Flow rates and circuit configurations both have a major impact on their overall cardiac and systemic effects.

Conclusions

- **Other factors also affect the response to MCS, include:**
  - 1) the cardiovascular substrate;
  - 2) the degree of acute LV recovery following initiation of MCS (;
  - 3) right-sided factors, such as RV systolic and diastolic function and pulmonary vascular resistance;
  - 4) the degree to which baroreflexes are intact and can modulate vascular and ventricular properties;
  - 5) concomitant medications;
  - 6) metabolic factors, such as pH and pO_2, which, if corrected, could result in improved ventricular and vascular function.

Discover!

Tools for practice



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