Cardiogenic Shock

Courtney A. Shores M.H.S., RPA-C

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Objectives

Understanding the physiology of Cardiogenic Shock

Understand the management of Cardiogenic Shock

Advanced therapies in managing Cardiogenic Shock

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Cardiogenic Shock

- Cardiogenic shock is a state of inadequate tissue perfusion due to cardiac dysfunction
- Manifested with organ dysfunction due to hypoperfusion:
  - oliguria (renal failure)
  - confusion
  - cool extremities
  - lactic acidosis
  - respiratory distress (pulmonary congestion)

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Pathophysiology and hemodynamic profile of shock states

Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion
Clinical measurement	Pulmonary capillary wedge pressure	Cardiac output	Systemic vascular resistance	Mixed venous oxygen saturation
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Distributive	↓ or ↔	↑	↓	↑

UpToDate

Cardiogenic Shock Hemodynamics

- Persistent hypotension (> 30 minutes)
  - SBP <90 mmHg
  - MAP <30 mmHg lower than baseline
- Cardiac Index (CI) <2.2 L/min/m^2
- Left ventricular end-diastolic pressure (LVEDP) >18 mmHg
- Right ventricular end-diastolic pressure (RVEDP) >10 mmHg
- Pulmonary capillary wedge pressure (PCWP) >15 mmHg

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Epidemiology

5-10% of patients with acute ST-elevation Myocardial Infarction (STEMI)

- does not include out of hospital arrests
- associated with a 70-80% mortality rate

2.5% of patients with non-STEMI

RISK FACTORS

- Anterior wall MI
- h/o coronary artery disease (CAD)
- Hypertension
- Diabetes Mellitus

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Etiology

*Acute Myocardial Infarction *

- Pump failure
  - Infarct
    - 55% infarction involved anterior wall
  - Infarct with pre-existing LV dysfunction/Reinfarction
- Mechanical complications
  - Acute MR (papillary muscle rupture)
  - VSD (ventricular septal defect)
  - LV free wall rupture
  - Pericardial Tamponade
- Right ventricular infarct

Etiology

End–stage Cardiomyopathy
Myocarditis
Prolonged cardiopulmonary bypass
Septic shock with myocardial involvement
LV outflow tract (LVOT) obstruction

- Aortic stenosis, Hypertrophic Obstructive Cardiomyopathy (HOCM)

Obstruction to LV filling

- Mitral stenosis, left atrial myxoma

Aortic insufficiency
Tako-tsubo cardiomyopathy (apical ballooning)

Physiology - at time of Myocardial Infarction

INFARCT EVENT - cardiac pump function declines

- initial redistribution of blood flow
  - optimized cardiac and cerebral blood flow
- Sympathetic nervous system activation
  - arterial vasoconstriction
  - increase myocardial contractility
  - tachycardia

Leads to **temporary** increase systemic BP

- increases pressure during diastole minimizes decrease coronary perfusion

Leads to increase *afterload*

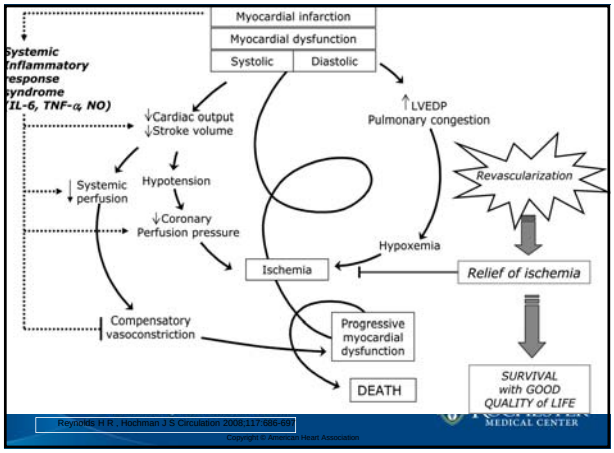
- further impairment of cardiac performance
- increasing myocardial oxygen demand -> ISCHEMIA

Physiology of Cardiogenic Shock

Ischemia (anterior wall)

LV myocardial ischemia -> pump failure (systolic and diastolic)
Systolic:
decreased stroke volume (SV) -> decreased Cardiac output (CO) -> hypotension -> decrease coronary perfusion -> **ISCHEMIA**
-> dec systemic perfusion->vasoconstriction -> **ISCHEMIA**
Diastolic:
increased LVEDP -> pulmonary congestion/edema -> hypoxia -> increased oxygen demand -> **ISCHEMIA**

further **ISCHEMIA** -> decrease myocardial function -> **DEATH**



Reynolds H R , Hochman J S Circulation 2008;117:686-697

Systolic Dysfunction

Loss of intrinsic inotropy (contractility)

Alterations in signal transduction mechanism for regulating inotropy

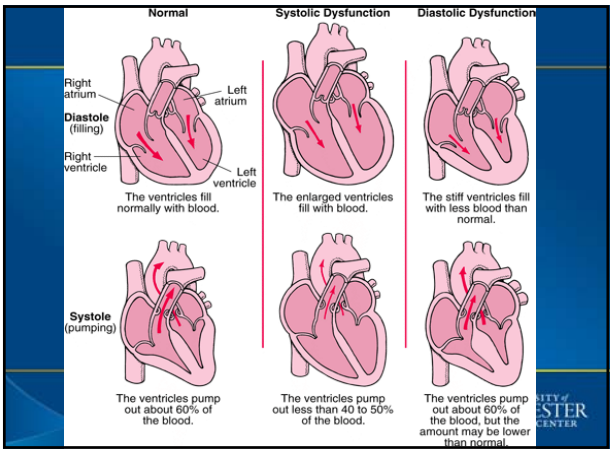
Loss of viable myocardium

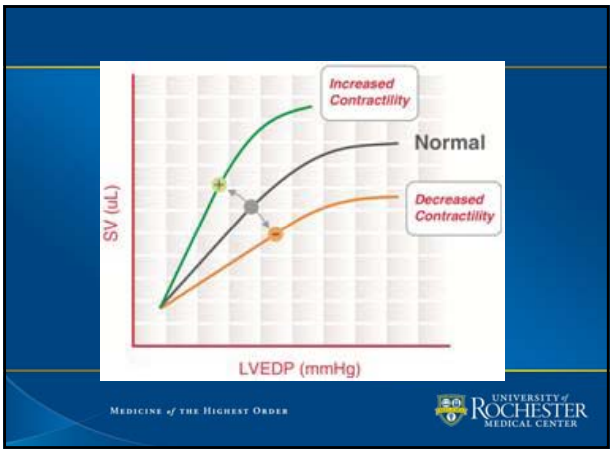
Diastolic dysfunction

Loss of compliance

(stiff ventricle)

Leads to impaired ventricular filling (high filling pressure to achieve normal filling volume)





Case 1

CC: chest pain

53 yo female presented to OSH with SSCP (7/10) beginning 1 day ago. Progressive chest pain, now at rest and associated with SOB. Radiates to neck. Aggravated by any activity not relieved with anything. Pain 9/10 upon transfer.

EKG: sinus tachycardia 114, anterior wall STE

Meds given at OSH: tPA, ASA, heparin, oxygen, insulin

Transferred for emergent cardiac catheterization.

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Case 1

PMH
- HTN
- DM II - non-insulin dependent
- Hypercholesterolemia

Medications (Home) Allergies: NKDA
- HCTZ 25mg, Metformin 1000mg BID, Pravachol 40mg

FHx
- Father: + CAD, Deseased: age 55, STEMI

SHx
- Married, three children, Emplorer: teacher
- + Tobacco: 50 PackYr (2 PPDx25 years)

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Case 1

Physical Exam

BP: 80/50 HR 110 regular

General: Moderate distress

Heart: Rapid rate, reg rhythm, no murmor, S3 gallop

Lungs: few rales

Ext: cool, clammy

EKG

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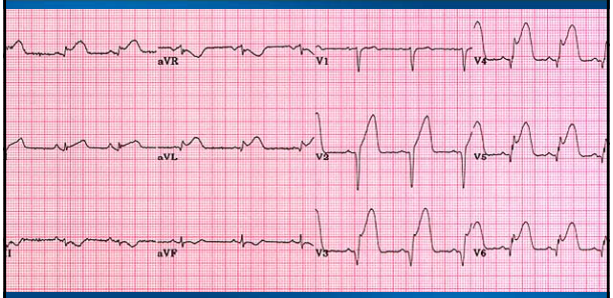
Case 1



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Case 1

Cardiac catheterization:
- focal dissection of Left Main, 100% occlusion of pLAD, 100% dRCA
- EF: ~8, global hypokinesis

Acute Pulmonary Edema
- Oxygen saturayion 80%
- Emergent intubation

VF arrest
- CRP, Defibrillation x2, full ACLS protocol

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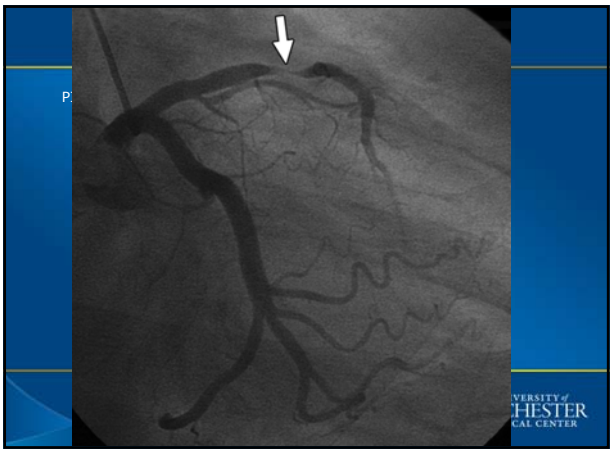

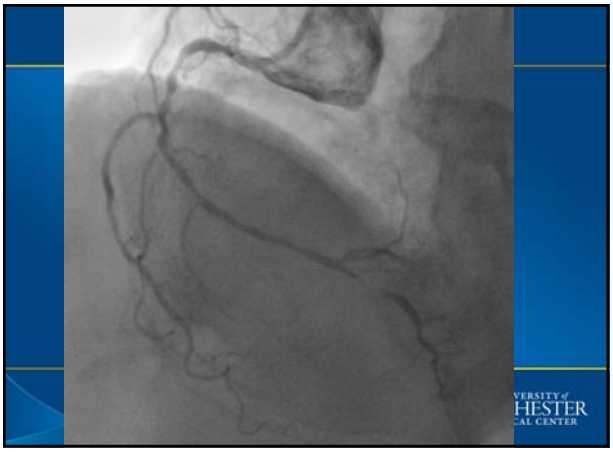

Echocardiogram

Transthoracic (TTE)/Transesophageal (TEE)
- Assess LV and RV fcn
- Tamponade
- Assess valvular function
  - MR
  - AS/AI
  - Rupture chordae/papillary muscle
  - VSD

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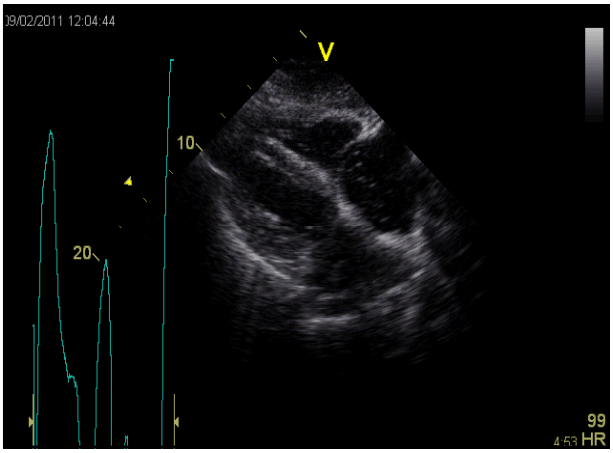
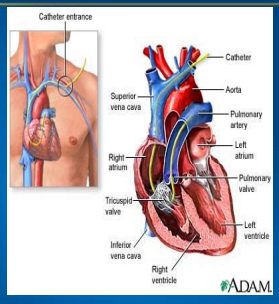


Pulmonary artery catheter

Monitor Hemodynamics
- Filling pressures
- RVEDP
- PCWP
- CO

Indications:
Progressive hypotension unresponsive to fluid
STEMI with suspect of mechanical complication



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Case 1 Hemodynamics

- Radial arterial line BP: 75/48
- HR: ST 120 (after epinepherine)

- PAC
  - CVP 15 mmHg
  - PAP 50/13 mmHg
  - PCWP 20 mmHg
  - CO/CI 3.4/1.3 L/min.m2
  - SVR 1250
  - SVO2 54%

Case 1 Labs

- CBC: WBC 11, Hgb 13, Hct 39, Plt 213
- BMP: Na 143, K 3.5, Cl 99, Co2 21, BUN 13, Cr 1.3, Mag 1.7
- BG: 156
- LFT: nl
- Lactate: 5.4
- CK: 8342
- CK-MB: 13
- Troponin I: 7.3

- Abg: 7.29/45/79/19

Chest radiograph following the onset of shock demonstrates interval development of bilateral alveolar infiltrates.

Guerrero J et al. Chest 2009;135:217-220

Management

Pharmacologic

Hypothermic protocol

Revascularization (PCI/Surgical)

Mechanical
- IABP
- ECMO
- Ventricular Assist Device
  - Impella
  - VAD

Cardiac Transplant

Pharmacologic

Goal:

optimize coronary and systemic perfusion

- Treat both hypotension and hypoperfusion
- Maintain/improve organ perfusion

Pharmacologic

Blood pressure GOAL: MAP >60 tissue perfusion

Vasopressor agents:
- ***Dopamine*** (first line)
  - <5 mcg/kg/min – vasodilation renal, mesentary, coronary
  - 5-10mcg/kg/min – ß1-agonist – inotrope/chronotrope
  - >10 mcg/kg/min – α-agonist - arterial vasoconstriction, inc BP
- Norepinepherine potent α-agonist, minimal ß1-agonist
  - potent vasoconstriction, little inotrope
- Epinepherine increase MAP by increasing SV, SVR and HR
  - Increase oxygen delivery
- Vasopressin

Adrenergic receptors

α receptors (α_1 α_2)
- Vasoconstriction of arteries and veins
- Decrease motility of smooth muscle in GI tract

ß receptors (β_1 β_2 β_3)

β_1
- increase CO, by raising heart rate (positive chronotropic), increasing conduction, and increasing contraction, increases volume expelled with each beat (increased ejection fraction).
- Increase renin secretion

β_2
- Smooth muscle relaxation

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Pharmacologic

Inotropic agents
- Dobutamine ß1-agonist (some ß2, minimal α)
  - Increase CO and HR
  - vasodilation -> decrease afterload
  - * avoid in pts with severe hypotension
- Phosphodiesterase inhibitors (milrinone)
  - Increase CO
  - Vasodilation -> decrease afterload
  - Reduces PVR -> decreased preload
  - *optimize patient fill, greater incidence of arrhythmias [32]

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Revascularization

Percutaneous Coronary intervention (PCI)
- Timing: door-to-balloon <90min
- Stent (bare metal, drug eluding)
- Glycoprotein IIb/IIIa inhibition
- Antiplatelet therapy (aspirin, clopidogrel)

Surgical
- Coronary artery bypass grafting (CABG)

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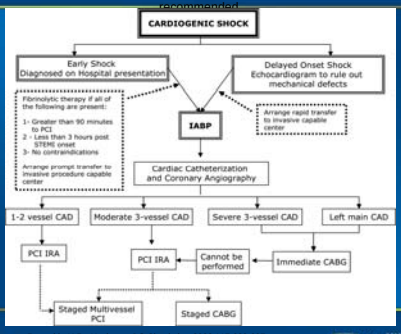
Figure 4. Algorithm for revascularization strategy in cardiogenic shock, from ACC/AHA guidelines.42,44
Whether shock onset occurs early or late after MI, rapid IABP placement and angiography are



Reynolds H R , Hochman J S Circulation 2008;117:686-697

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Case 1

IABP urgently placed in cardiac catheterization lab

Gtt: Dopamine, dobutamine, heparin, insulin, amiodarone

transferred to Cardiac ICU
- Hemodynamic monitoring
- Medication optimization/stabilization

2 days later

CABG x3 (LIMA-LAD, SVG-OM1, SVG-RCA)

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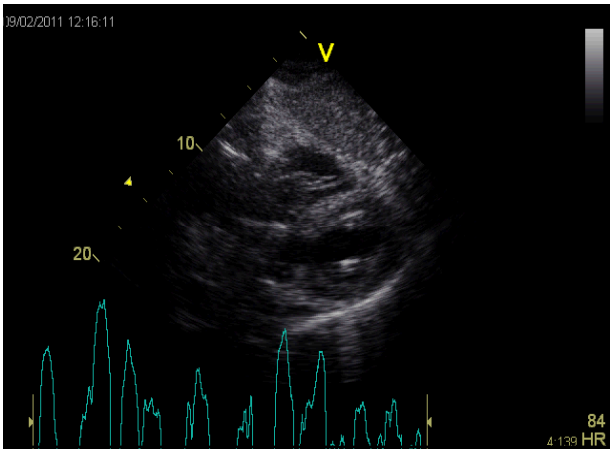


Case 1

POD 0-1
- neurologically intact
- wean pressors

POD #2
- IABP weaned and removed
- weaned inotropic support
- Extubated

POD #7

d/c home

Meds: ASA, Beta Blocker, ACEi, diuretic, statin, insulin

SHOCK Trial

30 days

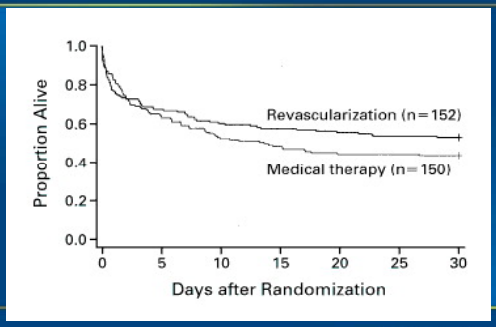
No significant overall benefit of early revascularization for patients with STEMI new left bundle-branch block who had cardiogenic shock due to LV dysfunction.

Early revascularization resulted in lower mortality from all causes at six months.

Recommend that early revascularization be strongly considered for patients with acute myocardial infarction complicated by cardiogenic shock

Hochman JS et al. N Engl J Med 1999;341:625-634

Overall 30-Day Survival in the Study.



Hochman JS et al. N Engl J Med 1999;341:625-634.

SHOCK trial

TABLE 4. MORTALITY AMONG STUDY PATIENTS.*

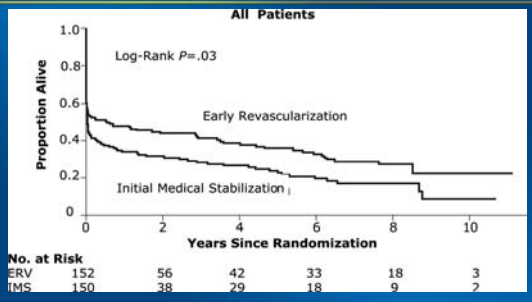
OUTCOME AND SUBGROUP	REVASCULARIZATION	MEDICAL THERAPY	DIFFERENCE BETWEEN GROUPS (95% CI)	RELATIVE RISK (95% CI)	P VALUE
	percent (number in subgroup)		percent		
30-day mortality					
Total	46.7 (152)	56.0 (150)	−9.3 (−20.5 to 1.9)	0.83 (0.67 to 1.04)	0.11
Age <75 yr	41.4 (128)	56.8 (118)	−15.4 (−27.8 to −3.0)	0.73 (0.56 to 0.95)	0.01†
Age ≥75 yr	75.0 (24)	53.1 (32)	+21.9 (−2.6 to 46.4)	1.41 (0.95 to 2.11)	
6-mo mortality‡					
Total	50.3 (151)	63.1 (149)	−12.8 (−23.2 to −0.9)	0.80 (0.65 to 0.98)	0.027
Age <75 yr	44.9 (127)	65.0 (117)	−20.1 (−31.6 to −7.1)	0.70 (0.56 to 0.89)	0.003†
Age ≥75 yr	79.2 (24)	56.3 (32)	+22.9 (0.7 to 46.6)	1.41 (0.97 to 2.03)	

*CI denotes confidence interval.

†Appropriate subgroup-analysis P values (for the interaction between treatment and the subgroup variable) are shown. Univariate P values for the comparison between treatments within subgroups were as follows: for 30-day mortality, P=0.02 for patients <75 years of age and P=0.16 for those ≥75 years of age; and for 6-month mortality, P=0.002 for patients <75 years of age and P=0.09 for those ≥75 years of age.

Hochman J et al. N Engl J Med 1999;341:625-634

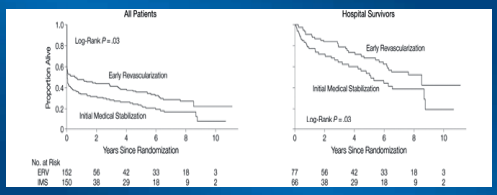
Figure 5. Long-term follow-up of the SHOCK trial cohort.55 Early revascularization (ERV) is associated with sustained benefit.



Reynolds H R , Hochman J S Circulation 2008;117:686-697

Kaplan-Meier Long-term Survival of All Patients and Those Discharged Alive Following Hospitalization

SHOCK – 6 years



Hochman, J. S. et al. JAMA 2006;295:2511-2515.

Mechanical

Goal:

optimize coronary and systemic perfusion

Intra-aortic Balloon Pump (IABP)

Ventricular Assist Device (VAD)

Extracorporeal membrane oxygenation (ECMO)

IABP

Diastolic balloon inflation
- Counterpulsation
  - Augments coronary artery blood flow

Systolic balloon deflation
- Decreased afterload
  - Augments LV performance

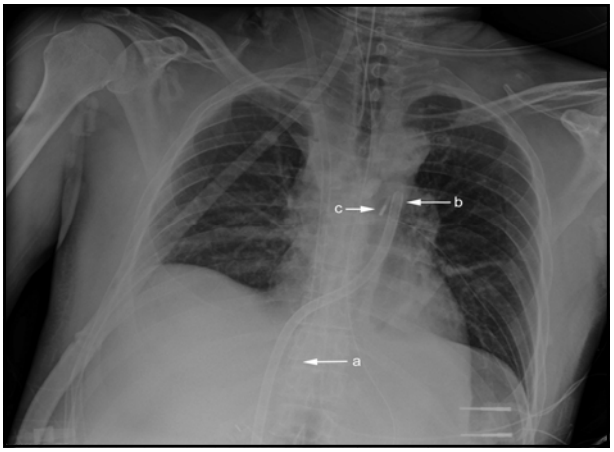
Decreases myocardial oxygen demand

VAD

blood pump that is as extracorporeal circulatory support device providing hemodynamic stabilization in patients in need of cardiopulmonary support

Temporary (~0-30 days)
- Abiomed
- Centrimag LVAD/RVAD

Destination/Bridge to transplant
- Heartmate II (LVAD)



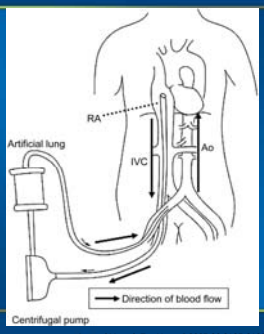
ECMO

Continuous pump to optimize gas exchange (O2/CO2) and perfusion

VV (Venovenous) – Oxygenation

VA (venoarterial) –Oxygenation and perfusion

Figure 1 Illustration of ECMO system.



Asaumi Y et al. Eur Heart J 2005;26:2185-2192

European Heart Journal

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Impella

circulatory support by unloading the left ventricle in addition to maintaining adequate forward blood flow

Case 2

29 yo male with severe familial dilated cardiomyopathy. EF 8%

maintained on IV milrinone, listed for cardiac transplant.

VT with AICD cardioversion.

loaded with amiodarone and started on an amio gtt

Hypotensive (SBP 50)

Take to cardiac cath lab for RHC and IABP placement

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Case 2 RHC

BP	72/58 mmHg
HR	90 SR
CVP	15 mmHg
PA Pressure	42/31 mmHg
PCW Pressure	25 mmHg mmHg
CO/CI	3.63 / 1.82 l/m/m2
SVR	882 dynes/sec/cm-5
PVR	1.65 Woods U
RVSWI	324 mmHg ml/m2

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Case 2

Impella placed in cath lab

Labs

CBC: WBC 15, HgB 12.9, Hct 35, Plt 116

Plasma Hbg: 36

BMP: Na 136, **K 5.8**, Cl 102, CO2 25, BUN 23, Cr 1.4

LFT: T.bili 1.9, AST 1193, ALT 795, **LDH > 392**

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Case 2

OR for BiVAD

- Heartmate II LVAD and Centrimag RVAD
- Impella removed intraop

Postop (CVICU)

POD #0

- Coagulopathy reversed
- IABP weaned and removed

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Case 2

POD #2 - wean Flolan, Pressure support

POD #3 - Extubate

POD #4-10

- Optimize nutrition, strength
- RVAD wean

POD #11 – RVAD explant

POD #12-17

- wean inotropic support, transition to PO medication
- Patient and Family education
- optimize nutrition, strength and motility

POD #18 - d/c on home on LVAD, on Transplant list

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