Cardiogenic Shock Team
ACTIVATION

Call **888.687.6428** or **505.727.7646** to activate Heart Team

Heart Hospital of New Mexico
@ LOVELACE MEDICAL CENTER

WHY is there a Shock Team?

Early identification and treatment improves survival in Cardiogenic Shock.

WHAT is the Cardiogenic Shock Team?

A **multidisciplinary team** dedicated to optimizing the care of Cardiogenic Shock patients via:

- Rapid identification
- Coordinated consultation
- Early transfer/admission to Cardiac ICU, Cath Lab or Operating Room

WHO is on the Shock Team?

- Interventional Cardiologist
- Cardiac Critical Care

HOW is the Shock Team activated?

Call **888.687.6428** or **505.727.7646**

WHO activates the Shock Team?

- Emergency Department
- Other units in the hospital (eg, Cath Lab or ICUs)
- Other hospitals

WHEN is the Team Activated?

Call the Shock Team as soon as Cardiogenic Shock is suspected

Clinical Criteria: Clinical presentation consistent with acute decompensated heart failure or acute coronary syndrome with either:

- Hypotension. SBP<90 (for 30 min) or use of vasopressors/inotropes or
- Hypoperfusion Lactate>2, evidence of end-organ (eg, renal hepatic, cerebral) hypoperfusion

Hemodynamic Criteria (if known)

- CI < 1.8 (or 2.2 L/min/m² with inotropes or vasopressors)
- CPO < 0.6
- PAPi < 1.0
- PCWP ≥ 15 mmHg

*Contraindications**

- DNAR
- Terminal Illness

>> Note: for STEMI, follow STEMI pathway

**if any questions, contact the Cardiogenic Shock Team*

AFTER the team has been activated

- Obtain ongoing vital signs, ECGs, Labs (eg, BNP, Tn I, Lactate, CBC, CMP)
- Maintain 2 large bore IVs (consider central line as needed)
- Minimize vasopressors/inotropes to maintain MAP of > 60 mmHG
- Preferential use of norepinephrine for vasopressor support
- Preferential use of amiodarone for control of VT or AF
- Avoid negative inotropes (eg, ß-blockers, Ca^{++} channel blockers)
- Consider airway stabilization

Adopted from INOVA Heart and Vascular Institute 25914 Rev. 07/23

Cardiogenic Shock Team
COORDINATION

Heart Hospital of New Mexico
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Heart Team Goals

- Early identification of CS patients
- Early CS phenotyping
- Selective and tailored PMCS
- Optimize hemodynamics
- Native heart recovery

* Clinical Considerations for PMCS

- Shock phenotype (AMI-CS vs HF-CS)
- Shock severity (SCAI Classification)
- Shock profile (LV, RV, Bi-V)
- Lactate level
- Severity of end organ dysfunction
- Amount of vasopressor/inotropic support
- Presence of hypoxia
- Presence of arrhythmias

Relative PCMS Contraindications

- DNAR
- Terminal illness
- Unable to anticoagulate
- Unable to receive blood products
- Cardiac arrest with neurocatastrophe
- Advanced multi-system organ failure
- LA or LV thrombus

*CPO=MAP x CO/451 *PAPi=(sPAP-dPAP)/RA

Heart Team Activation

- Call **888.687.6428** or **505.727.7646** for any patient with criteria for **Cardiogenic Shock**
- Obtain ongoing Vital Signs, ECG, Labs

HF-CS

- Echocardiography
- Right Heart Catheterization

AMI-CS

- Coronary angiography with LVEDP
- Right Heart Catheterization

Are Criteria for Cardiogenic Shock Met?

- **SBP < 90mmHg or use of vasopressors/inotropes AND/OR:**
- **CI < 1.8 (or < 2.2 L/min/m² with inotropes/ vasopressors)**
- **PCWP ≥ 15 mmHg and/or LVEDP ≥ 15 mmHg**
- **CPO < 0.6**
- **PAPi < 1.0**
- **Lactate > 2 mmol/L**
- **Evidence of end-organ hypoperfusion**

YES

- **Consider Percutaneous Mechanical Circulatory Support (PMCS) based on Clinical Considerations for PMCS***
- Coronary revascularization PRN (consider IV antiplatelet agent)

NO

- Coronary revascularization as needed
- Swan-Ganz catheter left in place

Cardiac Intensive Care Unit for ongoing CS Management

- **Serial reassessment** of **hemodynamics** & **end-organ perfusion**
- Optimize Preload, Afterload, and Contractility
- Timely, tailored escalation of treatment for **Worsening Shock**
- Assess for ability to wean PMCS

Adopted from INOVA Heart and Vascular Institute 25914 Rev. 07/23

HF-Cardiogenic Shock MANAGEMENT

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CS Management Goals

- ***Serial reassessment (≤ q 6hr)*** of hemodynamics & end-organ perfusion
  - Lactate
  - Renal, hepatic function
  - Continuous hemodynamics
  - CPO & PAPi
- Optimize *Preload, Afterload and Contractility*
  - Volume or diuresis
  - Vasodilators or Vasopressors
  - Inotropes
- Timely, tailored treatment escalation for **Worsening Shock:**
  - Rising Lactate
  - Increasing pressor requirement
  - Worsening end organ function
  - CPO < 0.6 and/or PAPi < 1
  - RA > 15 and/or PCWP > 15
- Assess for LV and RV recovery
  - Wean PMCS, vasopressors and inotropes

CS Hemodynamic Profile

	LV-dominant	RV-dominant	Bi-V
RA	< 15	>15	>15
PCWP	>15	< 15	>15
CPO	< 0.6	< 0.6	< 0.6
PAPi	> 1.0	< 1.0	< 1.0

CPO=MAP x CO/451 *PAPi=(sPAP-dPAP)/RA

Treatment Considerations for Heart Failure-CS

- **Shock severity (SCAI stage)**
- **Shock profile (LV, RV or Bi-V)**
- Anticipated exit strategy (BTT or BTR)
- Presence of hypoxia
- Presence of arrhythmias
- Anticipated duration of support
- Ability to ambulate
- Contraindications to PMCS

SCAI B CS — *Beginning*

Hypoperfusion:
Lactate < 2 mmol/L
Minor renal & hepatic dysfunction

+/-

Hypotension:
SBP < 90 mmHg

Current Treatment:
No drugs or devices

→ **LV, RV or Bi V:**
IABP (and/or trial of vasopressors, inotropes or vasodilators)

SCAI C CS — *Classic*

Hypoperfusion:
Lactate ≥ 2 mmol/L
Major renal & hepatic dysfunction

+

Hypotension:
SBP < 90 mmHg

OR

Current Treatment:
1 drugs OR device

→ **LV-dominant:**
IABP or Impella CP or Trans-septal temporary LVAD

→ **RV-dominant or Bi-V:**
Pro-Tek Duo +/- Impella CP or Trans-septal temporary LVAD

SCAI D CS — *Deteriorating*

Hypoperfusion:
Lactate ≥ 4 mmol/L
Worsening renal & hepatic dysfunction

+

Hypotension:
Escalating pressors

OR

Current Treatment:
2 drugs OR devices

→ **LV-dominant:**
Impella 5.5 or Trans-apical or temporary LVAD

→ **RV-dominant or Bi-V:**
VA-ECMO +/- LV vent

SCAI E CS — *Extremis*

Hypoperfusion:
Lactate ≥ 8 mmol/L
Severe acidosis & end-organ failure

+

Hypotension:
Refractory

OR

Current Treatment:
≥ 3 drugs OR devices

→ **LV, RV or Bi-V:**
VA-ECMO +/- LV vent

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AMI-Cardiogenic Shock MANAGEMENT

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CS Management Goals

- ***Serial reassessment (≤ q 6hr)*** of hemodynamics & end-organ perfusion
  - Lactate
  - Renal, hepatic function
  - Continuous hemodynamics
  - CPO & PAPi
- Optimize *Preload, Afterload and Contractility*
  - Volume or diuresis
  - Vasodilators or Vasopressors
  - Inotropes
- Timely, tailored treatment escalation for Worsening Shock:
  - Rising Lactate
  - Increasing pressor requirement
  - Worsening end organ function
  - CPO < 0.6 and/or PAPi < 1
  - RA > 15 and/or PCWP > 15
- Assess for LV and RV recovery
  - Wean PMCS, vasopressors and inotropes

CS Hemodynamic Profile

	LV-dominant	RV-dominant	Bi-V
RA	< 15	>15	>15
PCWP	>15	< 15	>15
CPO	< 0.6	< 0.6	< 0.6
PAPi	> 1.0	< 1.0	< 1.0

CPO=MAP x CO/451 *PAPi=(sPAP-dPAP)/RA

Treatment Considerations for AMI-CS

- **Shock severity (SCAI stage)**
- **Shock profile (LV, RV or Bi-V)**
- Revascularization status (mode and completeness)
- Presence of mechanical complications (eg, VSD, MR)
- Presence of hypoxia
- Presence of arrhythmias
- Contraindications to PMCS
- Use of IV antiplatelet agent

SCAI B CS *Beginning*

Hypoperfusion:
Lactate < 2 mmol/L
Minor renal & hepatic dysfunction

+/-

Hypotension:
SBP < 90 mmHg

Current Treatment:
No drugs or devices

→ **LV, RV or Bi V:**
Impella CP

SCAI C CS *Classic*

Hypoperfusion:
Lactate ≥ 2 mmol/L
Alteration of renal & hepatic dysfunction

+

Hypotension:
SBP < 90 mmHg

OR

Current Treatment:
1 drugs OR device

→ **LV-dominant:**
Impella CP
or
Transeptal temporary LVAD

→ **RV-dominant or Bi-V:**
Pro-Tek Duo
+/-
Impella CP
or
Transeptal temporary LVAD

SCAI D CS *Deteriorating*

Hypoperfusion:
Lactate ≥ 4 mmol/L
Worsening renal & hepatic dysfunction

+

Hypotension:
Escalating pressors

OR

Current Treatment:
2 drugs OR devices

→ **LV-dominant:**
Impella 5.5
or
VA-ECMO
or
Transeptal temporary LVAD

→ **RV-dominant or Bi-V:**
VA-ECMO
+/-
LV vent

SCAI E CS *Extremis*

Hypoperfusion:
Lactate ≥ 8 mmol/L
Severe acidosis & end-organ failure

+

Hypotension:
Refractory

OR

Current Treatment:
≥ 3 drugs OR devices

→ **LV, RV or Bi-V:**
VA-ECMO
+/-
LV vent

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New Mexico Heart Hospital E-CPR Guidelines

Age < 70 years

Witnessed arrest Arrest to first CPR (“no-flow interval”) < 5 minutes (i.e., bystander CPR)

Initial cardiac rhythm of VF/pVT/PEA Arrest to ECMO flow < 60 minutes

“Signs of life” during conventional CPR may be a positive predictive factor for survival

The absence of previously known life limiting comorbidities (e.g. end stage heart failure/chronic obstructive pulmonary disease/endstage renal failure/liver failure/terminal illness) and consistent with patient’s goals of care

No known aortic valve incompetence (>mild aortic valve incompetence should be excluded)

*Unless other favorable prognostic features are present: e.g., periods of intermittent ROSC/hypothermia prearrest/ young age/signs of life during CPR. CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation; ECPR, extracorporeal cardiopulmonary resuscitation; ROSC, return of spontaneous circulation.

Protocol adopted from ELSO guidelines, 2021.

25914 Rev. 07/23