Acute Heart Failure

In simple words, it is an acute state of decreased cardiac output resulting in inadequate tissue perfusion despite adequate or excessive circulating volume.

- Cardiogenic shock: It is characterised by systemic hypoperfusion due to severe depression of the cardiac index < 2.2 L/min/m2 and sustained systolic arterial hypotension (<90 mmHg) despite an elevated filling pressure (PCWP>18mmHg).

Clinical Features

- Dyspnea
- Pulmonary edema
- Narrow pulse pressure
- Cool extremities
- Increased heart rate
- SBP <90mmHg
- Decreased urine output
- Altered mental status

Hypertensive heart failure is the most common acute presentation, representing 50% of patients.

Precipitating Factors

Non compliance
- Excess salt
- Medication compliance

Cardiac cause
- New arrhythmia
- Rapid atrial fibrillation
- ACS
- Uncontrolled hypertension

Iatrogenic
- Use of calcium channel blocker, beta blocker or NSAID's
- Inappropriate therapy reduction
- Antiarrythmic agents within 48h

Non cardiac cause
- Infections
- Anemia
- Exacerbations of comorbidity
- Pulmonary embolus

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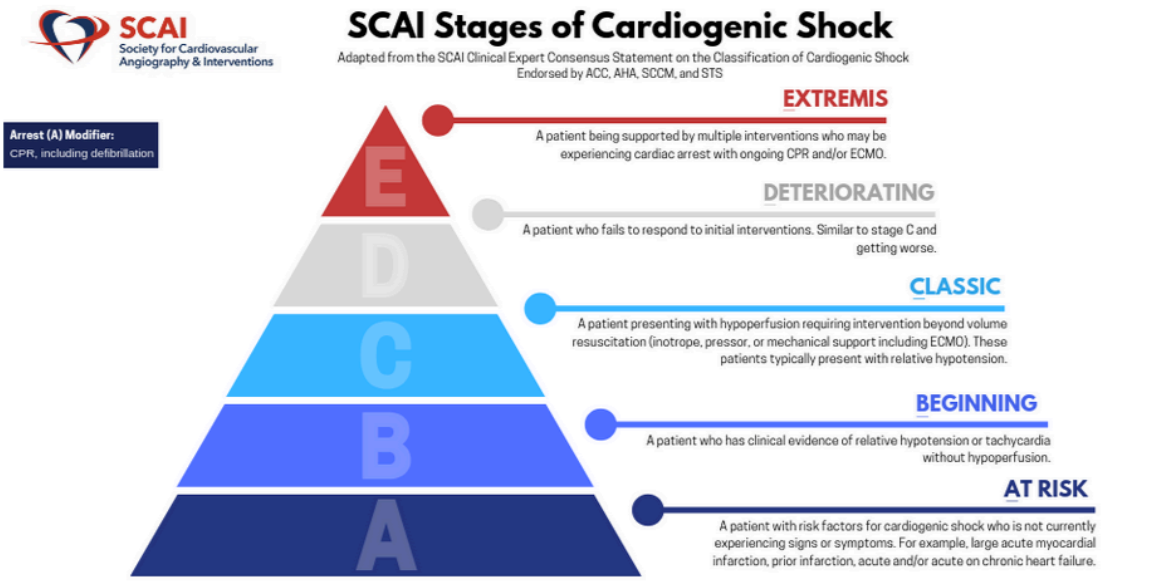
Volume overload

- Renal failure

SCAI Classification



Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock. Catheter Cardiovasc Interv. 2019;1-9. https://doi.org/10.1002/ccd.28329
For more information, please visit: www.scai.org/shockdefinition

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Stage	Description	Physical examination/bedside findings		Biochemical markers		Hemodynamics	
		Typically includes	May include	Typically includes	May include	Typically includes	May include
A At risk	A patient who is **not currently experiencing signs or symptoms of CS, but is at risk for its development**. These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Clear lung sounds	**Normal lactate**	Normal labs • Normal (or at baseline) renal function	**Normotensive** (SBP ≥100 mmHg or at baseline)	If invasive hemodynamics are assessed: • Cardiac Index ≥2.5 L/min/m^2 (if acute) • CVP ≤10 mmHg • PCWP ≤15 mmHg • PA saturation ≥65%
B Beginning CS	A patient who has **clinical evidence of hemodynamic instability** (including relative hypotension or tachycardia) **without hypoperfusion**.	**Elevated JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Rales in lung fields	**Normal lactate**	Minimal acute renal function impairment Elevated BNP	**Hypotension** • SBP <90 mmHg • MAP <60 mmHg • > 30 mmHg drop from baseline **Tachycardia** • Heart rate ≥100 bpm If invasive hemodynamics assessed **(strongly recommended)** • Cardiac index <2.2 L/min/m^2 • PCWP >15 mmHg	
C Classic CS	A patient who manifests with **hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation**. These patients typically present with relative hypotension (but hypotension is not required).	**Volume overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output <30 mL/h	**Lactate ≥2 mmol/L**	Creatinine increase to 1.5 x baseline (or 0.3 mg/dL) or > 50% drop in GFR Increased LFTs Elevated BNP		
D Deteriorating	A patient who is similar to category C but is getting worse. **Failure of initial support strategy to restore perfusion** as evidenced by worsening hemodynamics or rising lactate.	**Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion despite the initial therapy.**		**Any of stage C and lactate rising and persistently >2 mmol/L**	Deteriorating renal function Worsening LFTs Rising BNP	**Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion**	
E Extremis	**Actual or impending circulatory collapse**	**Typically unconscious**	Near pulselessness Cardiac collapse Multiple defibrillations	**Lactate ≥8 mmol/L**[a]	CPR (A-modifier) Severe acidosis • pH < 7.2 • Base deficit >10 mEq/L	**Profound hypotension despite maximal hemodynamic support**	Need for bolus doses of vasopressors

BNP, B-type natriuretic peptide; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function tests; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; SVP, systolic ventricular pressure.

[a] Stage E prospectively is a patient with cardiovascular collapse or ongoing CPR.

Investigations

Chest Xray

- Dilated upper lobe vessels
- Cardiomegaly
- Interstitial edema
- Enlarged pulmonary artery
- Pleural effusion
- Alveolar edema
- Prominent superior venacava
- Kerley lines

ECHO

BNP

- Natiuretic peptides are the endogenous counterregulatory arm to the neurohormonal activation of heart failure.
- Three types are recognised: Atrial natriuretic peptide secreted from atria, type B type natriuretic peptide secreted from ventricles & C natriuretic peptide localised in the endothelium.
- BNP is the only natriuretic peptide currently available for assay.
- BNP is synthesised as N-terminal pre-pro-BNP which itself is cleaved into N-terminal pro-BNP with a half life of approx 2 hrs and physiologically active BNP, with a half life of about 20 minutes.

- A BNP < 100 picograms/ml rules out heart failure with sensitivity of 90% & BNP >500 picograms/ml is suggestive of heart failure with a positive predictive value of 90%.

- A N-Terminal pro-BNP <300 picograms/ml rules out heart failure with a sensitivity of 99% and BNP > 900-1000 picograms is suggestive of heart failure with a specificity of 85% and PPV of 76%.

- Drawbacks:
  - Despite rapid release BNP levels may lag by 1hr or more in a setting of acute presentation.
  - Obesity results in low natriuretic peptide concentration.
  - BNP will be elevated in a patient with chronic heart failure, COPD, Pneumonia, hypertension,AF, Advanced age.

	Sensitivity	**Sepcificity**
NT pro BNP > 300	90.4%	38.2%
NT pro BNP > 1500	75.5%	72.9%
NT pro BNP > 450, Age <50	85.7%	93.9%

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NT pro BNP > 900, Age 50-75	79.3%	84%
NT pro BNP > 1800,Age>75	75.9%	75%

MANAGEMENT

Airway & Breathing

- Supplemental oxygen (SpO2 > 90%) as an initial measure for mild to moderate heart failure.
- Non invasive ventilation : Moderate to marked respiratory distress.
- Endotracheal intubation : Failure of NIV.

Diuretics

- Most patients with dyspnoea caused by pulmonary oedema obtain rapid symptomatic relief from administration of IV diuretics.
- The aim is to produce an urine output > 100ml/hr during the first 2hrs.

  Frusemide :
  - <0.5mg/kg for new onset acute pulmonary edema without hypervolemia.
  - 1mg/kg for acute on chronic volume overload, renal insufficiency.

Opiates

- Opiates such as morphine may be useful as they reduce anxiety and relieve distress associated with dyspnoea.
- DOSE : 4-8 mg
- Adverse effects: Respiratory depression.

Vasodilators

- Nitroglycerin
  - They act by stimulating guanylyl cyclase within smooth muscle cells, nitroglycerin, nitroprusside and nesiritide exerts dialting effects on arterial resistance and venous capacitance vessels, which results in a lowering of LV filling pressure, a reduction in mitral regurgitation and improved forward cardiac output.
  - NTG Dose : 10 - 20mcg/min titrated according to BP without reducing SBP < 80mmHg. A common side effect is headache.
  - Nitroprusside dose : Started at 0.3 mcg/kg/min and increased by 5 mcg/kg/min every 10-20 min as tolerated.

- Nesritide

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- It is a brain type natriuretic peptide which is an endogenous peptide secreted primarily from the LV in response to an increase in wall stress. It causes dilatation of arteries and veins.
- Dose : 2mcg/kg bolus followed by fixed infusion of 0.01 – 0.03 mcg/kg/min.

Inotropic agents :

These agents are not recommended unless the patient is hypotensive or evidence of organ perfusion is present.

- Dobutamine
  - It is the most commonly used inotropic agent for the treatment of acute HF.
  - It is given as a continuous infusion at an infusion 1- 2mcg/kg/min. May need doses > 5mcg/kg/min. Doses >10 mcg/kg/min has little added benefit.

- Milrinone

  - It is a phosphodiesterase III inhibitor that leads to increased cyclic AMP by inhibiting its breakdown.
  - Milrinone may act synergistically with beta adrenergic agonists to achieve a greater increase in cardiac output than dobutamine.
  - Dose: 25 – 75 mcg/kg over 10 -20 min followed by continuous infusion of 0.375 – 0.75 mcg/kg/min.

- Levosimendan
  - It enhances calcium sensitivity of contractile proteins by binding to cardiac troponin -C in dose dependent ,manner.
  - Dose: Loading dose 6 -24 mcg/kg IV bolus followed by infusion.

Vasopressors

- Vasopressors like dopamine , noradrenaline, phenylephrine and vasopressin are used to support systemic blood pressure in heart failure.
- Nordrenaline is considered as the first line vasopressor.

- If pharmacological intervention fails to stabilize a patient with refractory heart failure, mechanical and surgical intervention like intrathoracic aortic counter pulsation, percutaneous and surgically implanted LV assist devices and cardiac transplantation may be needed.

- SOAP II Trial showed lower risk of tachyarrhythmia for noradrenaline as compared with dopamine in a general shock patient and nominally lower 28 day mortality in patients with cardiogenic shock.
- DOREMI Trial showed no significant difference between milirone and dobutamine with regard to in hospital death, resuscitated cardiac arrest, TIA, heart transplant,

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renal replacement therapy.
- OPTIMA-CC Trial investigating efficacy and safety of epinephrine compared with norepinephrine in AMI related cardiogenic shock showed significantly higher incidence of refractory shock in epinephrine group (37% v/s 7%) and trial was terminated early.
- SURVIVE Trial - levosimendan v/s dobutamine did not show any significant difference in 1 month and 6 months.

Mechanical Circulatory Support Devices

- IABP
    - IABP placed in the descending aorta enhances coronary perfusion during systole.
    - It is not routinely recommended for infarct related cardiogenic shock but european guidelines recommend it for infarct related mechanical complication.
    - Altshock-2 Trial has shown that IABP did not improve survival or successful bridging to heart replacement therapy in heart failure related cardiogenic shock did not show a survival benefit as compared with medical therapy.

- VA ECMO
    - VA ECMO delivers flow support upto 6l/min can provide full respiratory and circulatory assistance for the right and left ventricles.
    - ECLS Shock trial showed no difference in death from any cause at 30 days between patients assigned to the group that received venoarterial ECMO (47.8%) and those in the control group (49.0%) (relative risk, 0.98; 95% confidence interval [CI], 0.80 to 1.19; P=0.81),with similar mortality in the two groups at 1-year follow-up.
    - Routine use of VA ECMO is not recommeded currently.

- Miroaxial Flow Pumps
    - It provides a peak flow of 4.3 l/min with a percutaneously placed catheter and are used to treat cardiogenic shock with LV dysfunction.

    - DanGer Shock Trial enrolled 360 patients with ST-segment elevation myocardial infarction who were not at risk for hypoxic brain injury, evaluated outcomes among patients treated with the microaxial flow pump as compared with those among patients who received standard (i.e., no microaxial flow pump but mechanical circulatory support for specific situations); in that trial, the pump led to lower all-cause mortality at 180 days (hazard ratio, 0.74; 95% CI, 0.55 to 0.99; P=0.04).
    - The survival benefit was sustained for up to 10 years, with proportional-hazards ratios over time suggesting a lasting effect. This trial also reported a greater incidence of bleeding and limb ischemia as well as renal replacement therapy in the microaxial flow pump group than in the

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control group.
    - Even though this trial showed a benefit of the microaxial flow pump among selected patients with left ventricular dominant cardiogenic shock,further discussion regarding appropriate patient selection and complication avoidance is ongoing.

- Left Atrial to Femoral Arterial Devices
    - Tandem heart mechanical circulatory support device which directs flow from left atrium to femoral artery is rarely used can produce a flow rate of upto 4l/min.

Non pharmacological
- Salt restriction <2.0 gm/d
- Fluid restriction to 1.5 – 2 L/d.

What are acute heart failure syndromes ?

Acute heart failure syndromes are classified by clinical presentation.

1. Hypertensive acute heart failure : Signs and symptoms of acute heart failure with relatively preserved left ventricular dysfunction, systolic blood pressure >140 mmHg, typically with chest radiograph compatible with pulmonary edema and symptoms onset over 48hr or less.

2. Pulmonary edema: Respiratory distress, reduced oxygen saturation from baseline, verified by chest radiograph findings.

3. Cardiogenic shock: It is characterised by systemic hypoperfusion due to severe depression of the cardiac index < 2.2 L/min/m2 and sustained systolic arterial hypotension (<90 mmHg) despite an elevated filling pressure (PCWP>18mmHg)

4. Acute decompensated heart failure: Signs and symptoms of acute heart failure that are mild to moderate and do not meet criteria for hypertensive heart failure, pulmonary edema or cardiogenic shock, systolic blood pressure <140mmHg and >90 mmHg, typically associated with increased peripheral edema and symptom onset over days.

5. High output failure : High cardiac output, typically with tachycardia, warm extremities & pulmonary congestion.

6. Right heart failure: Low output syndrome with jugular venous distension, hepatomegaly and may have hypotension.

Updated on 11/11/2026

Reference

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