TABLE 24 Society for Cardiovascular Angiography and Interventions (SCAI) Cardiogenic Shock Criteria (29)

Stage	Bedside Findings	Selected Laboratory Markers	Hemodynamics
A: At risk ■ Normotensive ■ Normal perfusion ■ Cause for risk for shock such as large myocardial infarction or HF	■ Normal venous pressure ■ Clear lungs ■ Warm extremities ■ Strong palpable pulses ■ Normal mentation	■ Normal renal function ■ Normal lactate	■ SBP >100 mm Hg ■ Hemodynamics: Normal
B: Beginning shock ("pre-shock") ■ Hypotension ■ Normal perfusion	■ Elevated venous pressure ■ Rales present ■ Warm extremities ■ Strong pulses ■ Normal mentation	■ Preserved renal function ■ Normal lactate ■ Elevated BNP	■ SBP <90 mm Hg, MAP <60 mm Hg, or >30 mm Hg decrease from baseline SBP ■ HR >100 bpm ■ Hemodynamics: CI ≥2.2 L/min/m^2
C: Classic cardiogenic shock ■ Hypotension ■ Hypoperfusion	■ Elevated venous pressure ■ Rales present ■ Cold, ashen, livedo ■ Weak or nonpalpable pulses ■ Altered mentation ■ Decreased urine output ■ Respiratory distress	■ Impaired renal function ■ Increased lactate ■ Elevated BNP ■ Increased LFTs ■ Acidosis	■ SBP <90 mm Hg; MAP <60 mm Hg; >30 mm Hg from baseline SBP despite drugs and temporary MCS ■ HR >100 bpm ■ Hemodynamics: CI ≤2.2 L/min/m^2; PCW >15 mm Hg; CPO <0.6 W; PAPi <2.0; CVP-PCW >1.0
D: Deteriorating ■ Worsening hypotension ■ Worsening hypoperfusion	■ Same as stage C	■ Persistent or worsening values of stage C	■ Escalating use of pressors or MCS to maintain SBP and end-organ perfusion in setting of stage C hemodynamics
E: Extremis ■ Refractory hypotension ■ Refractory hypoperfusion	■ Cardiac arrest ■ CPR	■ Worsening values of stage C laboratories	■ SBP only with resuscitation ■ PEA ■ Recurrent VT/VF

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BNP indicates brain natriuretic peptide; CI, cardiac index; CPO, cardiac power output; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; HR, heart rate; LFT, liver function test; MAP, mean arterial blood pressure; MCS, mechanical circulatory support; PAPi, pulmonary artery pulsatility index; PCW, pulmonary capillary wedge pressures; PEA, pulseless electrical activity; SBP, systolic blood pressure; VF, ventricular fibrillation; and VT, ventricular tachycardia.

specific inotropic agent is guided by blood pressure, concurrent arrhythmias, and availability of drug.

2. Despite the lack of direct comparative data, the use of short-term MCS has dramatically increased (9-16,31,32). The hemodynamic benefits of the specific devices vary, and few head-to-head randomized comparisons exist (33-39). Randomized clinical trials are underway that will address the risks and benefits of one modality over another. Vascular, bleeding, and neurologic complications are common to MCS devices, and the risk of such complications should generally be considered in the calculation to proceed with such support (40). As much as possible, an understanding of a patient's wishes, overall prognosis and trajectory, and assessment of therapeutic risk should precede the use of invasive temporary MCS.
3. Team-based cardiogenic shock management provides the opportunity for various clinicians to provide their perspective and input to the patient's management (17-22). The escalation of either pharmacological and mechanical therapies should be considered in the context of multidisciplinary teams of HF and critical care specialists, interventional cardiologists, and cardiac surgeons. Such teams should also be capable of providing appropriate palliative care. Most documented experiences have suggested outcomes improve after shock teams are instituted (17-22). In 1 such experience, the use of a shock team was associated with improved 30-