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Class 1b

EUROPEAN
SOCIETY OF
CARDIOLOGY®

Management of acute myocardial infarction in patients presenting with ST-segment elevation

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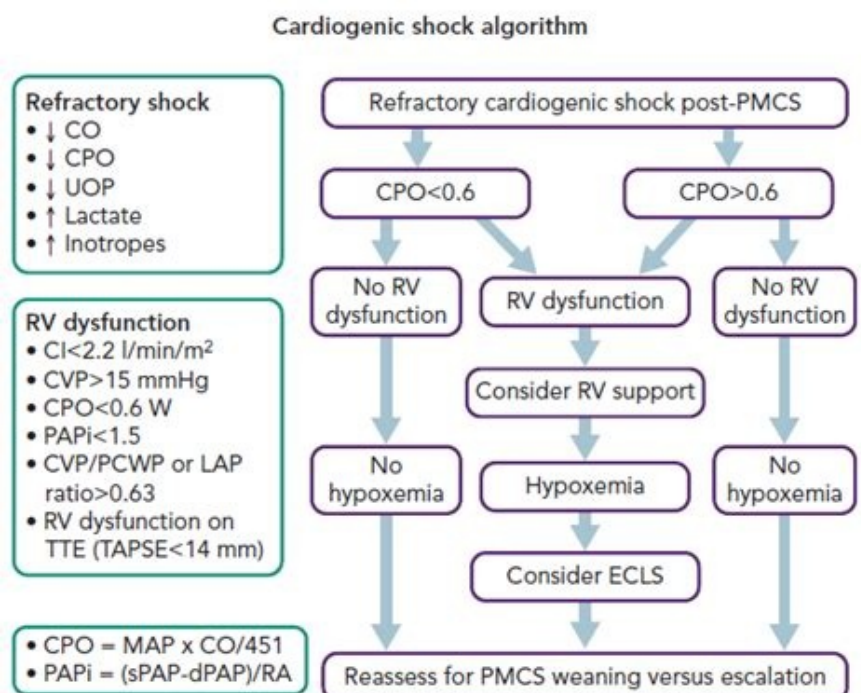
ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction

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 Interventional
& SurgeryAntman, et al,^[2] Van de Werf, et al,^[3] Wijns, et al.^[4]

Figure 3: INOVA Mechanical Circulatory Support Escalation and Weaning Algorithm/Protocol



CI = cardiac index; CO = cardiac output; CPO = cardiac power output; CVP = central venous pressure; dPAP = diastolic pulmonary artery pressure; ECLS = extracorporeal life support; LAP = left atrial pressure; PAPl = pulmonary artery pulsatility index; PCWP = pulmonary capillary wedge pressure; PMCS = percutaneous mechanical circulatory support; RV = right ventricle; sPAP = systolic pulmonary artery pressure; TAPSE = tricuspid annular plane systolic excursion; TTE = transthoracic echocardiogram; UOP = urine output.

Recommendations	Class ^a	Level ^a	Ref ^b
All patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.	I	C	
All patients with cardiogenic shock should be rapidly transferred to a tertiary care center which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term mechanical circulatory support.	I	C	
In patients with cardiogenic shock complicating ACS an immediate coronary angiography is recommended (within 2 hours from hospital admission) with an intent to perform a primary percutaneous revascularization.	I	C	
Continuous ECG and blood pressure monitoring are recommended.	I	C	
Invasive monitoring with an arterial line is recommended.	I	C	
Fluid challenge (saline or Ringer's lactate, *200 ml/15–30 min) is recommended as the first-line treatment if there is no sign of overt fluid overload.	I	C	
Intravenous inotropic agents (dobutamine) may be considered to increase cardiac output.	IIb	C	
Vasopressors (norepinephrine preferable over dopamine) may be considered if there is a need to maintain SBP in the presence of persistent hypotension.	III	B	558
IA2P is not routinely recommended in cardiogenic shock.	III	B	585, 586
Short-term mechanical circulatory support may be considered in refractory cardiogenic shock depending on patient age, comorbidities and neurological function.	IIb	C	

ACS = acute coronary syndrome; CCU = coronary care unit; ECG = electrocardiogram; IABP = intra-aortic balloon pump; ICU = intensive care unit; SBP = systolic blood pressure.

^aClass of recommendation.
^bLevel of evidence.^aReference(s) supporting recommendations.

	All patients (n = 68)	PCD ≤ median* (n = 35)	PCD > median* (n = 33)	P-value
SOFA score [median (IQR)]				
Total	5 (4–7)	5 (3–8)	6 (4–7)	0.96
Cardiac subscore	2 (2–3)	2 (2–3)	3 (2–3)	0.65
Renal subscore	1 (0–1)	1 (0–1)	0 (0–2)	0.7
Respiratory subscore	1 (1–2)	2 (1–2)	1 (1–2)	0.59
Coagulation subscore	0 (0–1)	0 (0–1)	0 (0–1)	0.86
Liver subscore	0 (0–0)	0 (0–0)	0 (0–0)	0.62
Timing of baseline measurements [median (IQR)]				
Time from AMI (h)	16 (6–20)	16 (9–20)	12 (4–22)	0.36
Time from shock onset (h)	5 (3–10)	5 (4–8)	4 (2–11)	0.39

ND, standard deviation; NS, non-significant; AMI, acute myocardial infarction; CV, cardiovascular; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; NT-proBNP, N-terminal pro-B-type natriuretic peptide; IQR, interquartile range; WBC, white blood cell; CRP, C-reactive protein; GFR, glomerular filtration rate; MR, mitral valve regurgitation; LM, left main coronary artery; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft surgery; SOFA, sequential organ failure assessment.

^bSystolic blood pressure < 90 mmHg and clinical signs of hypoperfusion.

^aDosages in $\mu\text{g kg}^{-1} \text{min}^{-1}$

European society of cardiology guidelines for cardiogenic shock. Cardiogenic shock nice guidelines. Guidelines for cardiogenic shock.

split-ventricle, fluid, intraaortic balloon pumping (IABP), and also active assist devices are widely used for CS management. However, there is only limited evidence for any of the above treatments except for early revascularization and the relative ineffectiveness of IABP. This updated review will therefore outline the management of CS complicating acute myocardial infarction with major focus on evidence-based revascularization techniques, intensive care unit treatment including ventilation, transfusion regimens, adjunctive medication, and mechanical support devices. Cardiogenic shock (CS) is defined as a state of critical endorgan hypoperfusion due to reduced cardiac output. The pathophysiology of CS is complex and has been reviewed previously.1,6 The clinical picture of CS can be summarized by the following criteria: (a) altered mental status; (b) cold, clammy skin; (c) oliguria; (d) increased serum lactate. The diagnosis of CS can usually be made on the basis of easy-to-assess clinical criteria without advanced haemodynamic monitoring although it has previously been recommended to assess cardiac index and pulmonary capillary pressure.1 Acute myocardial infarction (AMI) with subsequent ventricular dysfunction is the most frequent cause of CS accounting for ~80% of cases. Mechanical complications such as ventricular septal (4%) or free wall rupture (2%), and acute severe mitral regurgitation (7%) are less frequent causes of CS after AMI.2 Non-AMI-related CS may be caused by compensated valvular heart disease, acute myocarditis, arrhythmias, etc. with heterogeneous treatment options. Cardiogenic shock complicating AMI occurs in the range from 5 to 15%–35. This translates in ~40 000 to 50 000 patients per year in the USA and ~60 000 to 70 000 in Europe.6 Despite advances in treatment mainly by early revascularization with subsequent mortality reduction, CS remains the leading cause of death in AMI with mortality rates still approaching 40–50% according to recent registries and randomized trials.3–5 The underlying causes, the pathophysiology, and treatment of CS complicating AMI have been reviewed previously.1,6 This update will therefore focus on the management of CS complicating AMI. The pathophysiology of CS is complex and has been reviewed previously.1,6 In brief, ischaemia induces profound depression of myocardial contractility, which initiates a vicious spiral of reduced cardiac index and low blood pressure which in combination impair cardiac power index and further promote coronary ischaemia. The reduction in cardiac index causes severe tissue hypoperfusion which is most sensitively measured by serum lactate and may finally lead to death if the circle is not successfully interrupted by adequate treatment measures. It has been recognized that CS cannot only be attributed to the loss of left-ventricular function but is rather the result of derangements in the entire circulatory system. Initial compensatory vasoconstriction is subsequently counteracted by pathological vasodilation. Among others, development of systemic inflammation with capillary leakage, impairment of microcirculation, and vasodilation contribute to the vicious circle of CS. Bleeding and transfusion further contribute to inflammatory derangements in the shock spiral. By multivariable modelling from the major CS trials (SHOCK, TRIUMPH, IABP-SHOCK II), typical factors associated with higher mortality were older age, anoxic brain damage, lower left-ventricular ejection fraction, lower cardiac power index, lower systolic blood pressure, need for vasopressor support, worse renal function, and higher serum lactate.8–10 Multiple biomarkers have been investigated for prognostic value in CS, but only the pro-BNP and the procalcitonin can easily be measured sublingually by bedside rapid darkfield imaging. A diminished perfused capillary density at baseline and a lack of improvement derived from serial measurements was associated with dismal prognosis.13 However, the clinical value of these new biomarkers and imaging methods has not yet been finally defined. Management

Revascularization Early revascularization as shown in the SHOCK we emergently revascularize Occluded Coronaries for cardiogenic shock (SHOCK) trial is the most important treatment strategy in CS complicating AMI.14 Although the trial failed to meet the primary endpoint (superiority of early revascularization over medical therapy on 30-day mortality) there was a significant mortality reduction at longer follow-up of 1/2, 1, and 6 years.14,15 The number needed to save one life by early revascularization in comparison to initial medical stabilization is

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