Role of Cardiogenic Shock Centres and Regionalised Networks for Acute MI-related Cardiogenic Shock: State-of-the-art Review on Behalf of the British Cardiovascular Interventional Society Focus Group

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Abstract

Cardiogenic shock (CS) complicating acute MI (AMI-CS) remains associated with high mortality and morbidity, despite advances in coronary revascularisation and mechanical circulatory support (MCS). Outcomes are determined not only by individual therapies, but also by the organisation of care, with current guidelines emphasising multidisciplinary, system-level approaches to enable rapid diagnosis, triage and treatment. This review examines how CS centres function within regional networks of care. We review definitions of AMI-CS, including haemodynamic, clinical and biochemical markers and their application to early identification and triage. We summarise key components of centre-based management, including revascularisation, invasive haemodynamic assessment, selective MCS use and post-percutaneous coronary intervention critical care. Contemporary data from dedicated CS centres and regionalised network models are compared, highlighting variations in transfer criteria and hub–spoke organisation alongside limitations in current evidence. The distinct phenotypic nuances of out-of-hospital cardiac arrest-associated CS are discussed and gaps in evidence with future perspectives are highlighted.

Keywords

Acute MI-associated cardiogenic shock (AMI-CS), shock centres, AMI, cardiogenic shock (CS)

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Cardiogenic shock (CS) is characterised by a low cardiac output state resulting in inadequate tissue oxygen delivery and systemic hypoperfusion, representing a global health priority with high rates of mortality and morbidity. Despite advances in physiological understanding, prompt coronary revascularisation and mechanical circulatory support (MCS), in-hospital mortality rates have remained up to 50%, with little improvement over the past few decades.[1–3] This persistent mortality reflects both the complex and heterogeneous pathophysiology of CS, as well as variability in the organisation and delivery of care, including delays in recognition, escalation and access to specialist therapies.[4]

Acute MI-related CS (AMI-CS) is the most common precipitant of CS, accounting for nearly half of all cases and representing the subgroup of patients in whom timely recognition, revascularisation and coordinated systems of care are most critical to improving outcomes.[5,6] International guidelines have proposed multidisciplinary AMI-CS centres with the capability to rapidly recognise, classify and deliver advanced therapies, including revascularisation, MCS and specialised intensive care.[7–9] Hub-and-spoke models have been proposed to facilitate regionalised systems of care to expedite access to high-volume specialist centres.[10,11]

In this review we examine the organisation and delivery of care for AMI-CS within specialised CS centres and regionalised networks. We consider the pathophysiology of AMI-CS and the role of centre-based capabilities in its recognition, classification and management. Evidence for dedicated shock protocols, multidisciplinary team (MDT) care models and interhospital transfer systems is evaluated and current approaches to AMI-CS care structure are outlined, highlighting key areas of consensus, variation and unmet need.

Acute MI-related Cardiogenic Shock, Pathology, Classification and Systems of Care

Pathophysiology of Acute MI-related Cardiogenic Shock

CS represents a state in which cardiac dysfunction causes inadequate tissue perfusion and a failure to meet metabolic demand. In AMI-CS, this process is initiated by acute left ventricular (often with coexisting right ventricular) impairment due to acute coronary occlusion, leading to reduced cardiac output, subsequent hypoperfusion and progressive multiorgan failure. In a minority of patients, AMI-CS may instead result from mechanical complications of infarction, including ventricular septal defect, acute mitral regurgitation due to papillary muscle rupture or free wall rupture. These presentations underline the need for prompt echocardiographic diagnosis and urgent management in high-volume centres with surgical expertise. At a haemodynamic level, reduced cardiac output results in compensatory vasoconstriction leading to impairment of the microcirculation and initiation of a systemic inflammatory response, further contributing to organ dysfunction.[12,13]

Traditionally, patients with AMI-CS formed the largest cohort of patients with CS and, indeed, many studies have previously shown a trend for an increasing number of patients with AMI presenting in CS.[5,14] However, recent observational data have shown that as a proportion of all-comer CS, AMI-CS has declined, making up only 27% of 5,869 patients admitted to a medical cardiac intensive care unit (CICU) with CS.[15] Despite this, it should be noted that those admitted with AMI-CS had the highest in-hospital mortality of any CS aetiology, at 41%, compared with 31% for *de novo* heart failure (HF)-related CS (HF-CS), 25% for acute on chronic HF-CS and 31% for secondary CS.[15]

Unlike several other CS phenotypes, the pathological cascade of AMI-CS may be partially reversed with timely coronary reperfusion and MCS application. Delays in recognition, triage or transfer may result in irreversible MI and worse outcomes.[16–18] In AMI-CS, haemodynamic and metabolic derangements such as sustained hypotension, rising lactate and evidence of end-organ injury reflect different stages of CS. The recognition of these physiological markers can guide triage, transfer decisions and timing of specialist therapies.

CS centres and networks are designed to address a broad spectrum of CS phenotypes including, but not limited to, acute on chronic HF, valvular disease and myocarditis, as well as AMI-CS. These pathophysiological and time-sensitive considerations underpin the need for organisation of care in AMI-CS, which is increasingly delivered within specialised CS centres that centralise expertise, infrastructure and multidisciplinary decision-making. Timely recognition, continuous communication across levels of care and early transfer to higher-level CS centres for specialist care when required are central principles to this approach.

Systems of Care

Centralisation of AMI-CS care within regionalised networks is supported by observational data reporting improved outcomes in high-volume specialist centres.[19–23]

A tiered model of care has been adopted in both US and European guidance, with defined roles for Level 1, Level 2 and Level 3 centres, which are summarised in *Table 1* and illustrated in *Figure 1*.

Level 1 CS centres are high-volume tertiary hospitals functioning as regional CS hubs, described in more detail below. Level 2 centres offer 24/7 primary percutaneous coronary intervention (PCI) with cardiology-led teams and limited MCS capability. These centres can initiate CS pathways and stabilise patients, with early coordination and transfer to Level 1 centres when escalation of care is required. Level 3 centres are typically non-interventional hospitals with diagnostic capability only. Their role is early recognition, initial stabilisation and rapid transfer within a hub-and-spoke network.[9,10]

Level 1 Centres: US Guidelines

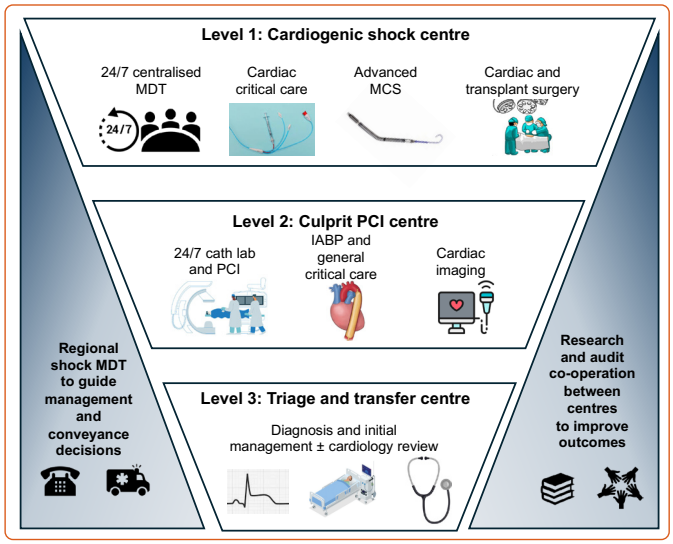
Guidance from the American Heart Association (AHA) and American College of Cardiology (ACC) defines Level 1 CS centres as hospitals capable of providing complete and comprehensive post-resuscitation and

Table 1: Resourcing for a High-volume Cardiovascular Shock Centre as Defined by the American Heart Association

Critical Care Resources	Cardiovascular Resources	Personnel Requirements	Professional Services	Academic Characteristics
• 24/7 on-site medical cover with on-call specialist • 1:1 nurse-to-patient ratio • Invasive cardiac and haemodynamic monitoring • Renal replacement therapy • Temporary pacing	• Cardiac surgery cover • Mechanical support devices such as IABP and microaxial flow pumps • ECMO team and eCPR capabilities • Advanced transthoracic and transoesophageal echocardiography	• Cardiologists with subspecialisation in intervention, electrophysiology, imaging and advanced heart failure • Intensive care physicians • Neurology, nephrology and respiratory support • Palliative care	• Allied health including social work, physiotherapy, occupational therapy and dietician • Pharmacy	• Cardiac shock research • Participation in national shock registries • Regular quality improvement and auditing • Trainee education

ECMO = extracorporeal membrane oxygenation, eCPR = extracorporeal cardiopulmonary resuscitation; IABP = intra-aortic balloon pump.

Figure 1: Levels of Care in Cardiogenic Shock Centres



IABP = intra-aortic balloon pump; MCS = mechanical circulatory support; MDT = multidisciplinary team; PCI = percutaneous coronary intervention.

shock care, including 24/7 coronary angiography and PCI, targeted temperature management, multimodal neuroprognostication, MCS and surgical support.[8,9] High procedural volumes are emphasised, although evidence remains observational.[24] Select centres may also offer durable left ventricular assist device implantation and cardiac transplantation.

Level 1 Centres: European Guidelines

European guidance similarly supports centralisation of AMI-CS care, although the description is currently less prescriptive. European Society of Cardiology (ESC) position statements emphasise management within dedicated CS centres with 24/7 PCI, multiple cath labs, on-site cardiac surgery, advanced haemodynamic monitoring and access to multiple MCS modalities, delivered by an experienced MDT with appropriate staffing ratios.[7,25] Patients with out-of-hospital cardiac arrest (OHCA) complicated by AMI-CS are a particularly high-risk subgroup who may benefit from immediate and direct transfer to centres with advanced MCS capabilities.

These guidelines underpin the delivery of protocolised care and form the basis for contemporary regional AMI-CS networks. Effective triage, escalation and interhospital transfer within these systems require a shared and operational definition of CS, as well as a framework for classifying severity.

Definition and Classification of Acute MI-related Cardiogenic Shock

Despite broad agreement on the underlying pathophysiology, defining the transition from haemodynamic compromise to overt CS remains challenging. Diagnostic criteria for AMI-CS vary across randomised trials, guidelines and registries, contributing to heterogeneity in study populations and reported outcomes. A key example is the threshold for hypotension: although most randomised controlled trials (RCTs) have used a systolic blood pressure (SBP) threshold of 90 mmHg, Burkhoff et al. mandated a mean arterial pressure of <70 mmHg and DanGer Shock used an SBP of 100 mmHg.[26,27] Such variation contributes to heterogeneous study populations representing different stages of AMI-CS. *Table 2* summarises this variability across major trials.

To improve standardisation, the Shock Academic Research Consortium proposed a consensus definition for AMI-CS incorporating sustained hypotension (SBP <90 mmHg for ≥30 minutes, or vasopressors required to maintain an SBP >90 mmHg) alongside markers of hypoperfusion, including reduced cardiac index, raised arterial lactate, systemic vascular resistance index and end-organ injury.[28] The ACC recently proposed the mnemonic SUSPECT-C to support early recognition and triage, integrating Symptoms, Urine output, Sustained hypotension, Perfusion, ECG/Echocardiogram, Congestion and Triage, with shock team activation or transfer where required.[9]

Although these frameworks enhance recognition, they primarily identify established CS and fail to fully capture its dynamic progression. Importantly, a subset of AMI-CS patients may exhibit significant haemodynamic compromise despite preserved blood pressure, reflecting advanced circulatory dysfunction not detected by conventional thresholds.[29]

To address this, classification systems have been developed to stage disease severity (*Table 3*). The Society for Cardiovascular Angiography and Interventions (SCAI) classification, is the most widely adopted framework, describing a continuum from A to E (At risk, Beginning, Classical, Deteriorating and Extremis), each described by a combination of clinical, haemodynamic and biochemical parameters.[30] Observational data, including the National Cardiogenic Shock Initiative, demonstrate that both baseline and dynamic SCAI stage are strongly associated with survival to discharge and 30-day mortality, supporting the role of the SCAI classification in risk stratification and guiding escalation of care within regionalised systems of care.[31,32] However, limitations of the SCAI classification include its partial subjectivity and that it has not yet been prospectively embedded within an RCT design.

Table 2: Definitions of Cardiogenic Shock Used in Landmark Clinical Trials

Trial	Clinical	Haemodynamic	Biochemical
SHOCK 1999[16]	End-organ perfusion • Cool extremities • Oliguria <30 ml/h	SBP <90 mmHg for >30 min and/or need for vasopressors Cardiac Index <2.2 l/min/m^2 and PCWP ≥15 mmHg	NA
Burkhoff et al. 2006[26]	End-organ perfusion • Altered mental status • Oliguria <30 ml/h • Cold/clammy extremities	MAP <70 mmHg, PCWP ≥15 mmHg and cardiac index <2.2 l/min/m^2	NA
IABP-Shock II 2012[51]	End-organ perfusion • Cool extremities • Oliguria <30 ml/h • Cold/clammy extremities	SBP <90 mmHg for >30 min and/or need for vasopressors	Lactate >2 mmol/l
ISAR-SHOCK 2008[52]	End-organ perfusion • Cool extremities • Oliguria <30 ml/h	SBP <90 mmHg for >30 min and/or need for vasopressors Cardiac index <2.2 l/min/m^2 and PCWP ≥15 mmHg	NA
CULPRIT-SHOCK 2017[1]	End-organ perfusion • Cool extremities • Oliguria <30 ml/h • Cold/clammy extremities • Pulmonary congestion	SBP <90 mmHg for >30 min and/or mechanical or inotropic support	Lactate >2 mmol/l
DOREMI 2021[78]	Signs of fluid overload with elevated JVP	SBP <90 mmHg or MAP <60 or >30 mmHg drop from baseline with tachycardia (SCAI B)	NA
ACCOST-HH 2022[79]	End-organ perfusion • Altered mental status • Cold/clammy skin • Oliguria <30 ml/h • Pulmonary congestion	SBP <90 mmHg for >30 min and/or inotropic support	Lactate >2 mmol/l
ECLS-SHOCK 2023[3]	End-organ perfusion • Altered mental status • Cold/clammy skin • Oliguria <30 ml/h	SBP <90 mmHg for >30 min and/or inotropic support	Lactate >3 mmol/l
DanGer Shock 2024[27]	LVEF <45% or SvO_2 <55%	SBP <100 mmHg for >30 min and/or need for vasopressors	Lactate >2.5 mmol/l
EURO SHOCK 2023[80] (terminated early)	End-organ perfusion • Cool extremities • Oliguria <30 ml/h • Cold/clammy extremities	SBP <90 mmHg for >30 min and/or need for vasopressors	Lactate >2 mmol/l

JVP = jugular venous pressure; LVEF = left ventricular ejection fraction; MAP = mean arterial pressure; PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions.

Table 3: Clinical Classification and Risk-stratification Systems Used in Cardiogenic Shock

Prognostic Model	Primary Use	Key Variables	Strengths	Limitations
Diamond–Forrester classification[81]	Simplified assessment of CS	Cardiac index and PCWP	Simple and can be qualitatively assessed clinically, or quantitatively with PAC	Does not incorporate tissue perfusion Confounded by RV shock
INTERMACS[82]	CS for consideration of advanced support and transplantation	Symptoms and need for inotropes +/– MCS	Well validated in the setting of heart failure and transplantation	Not specific to AMI-related CS
APACHE-II[83]	ICU mortality prediction across multiple critical illnesses, including CS	Age, BP, heart rate, PaO_2, pH, GCS, temperature, comorbidities	Well established and validated in all ICU patients	Multiple variables required Not designed specifically for CS
SCAI grade[84]	Classification tool for CS	Haemodynamics, inotropic requirements and hypoperfusion	Well validated in CS and simple to grade clinically	Limited use in normotensive shock and RV failure
CardShock[85]	Early prediction of in-hospital mortality in CS	Age, comorbidities, ejection and fraction biomarkers	Specific to CS	Large scale validation still required

AMI = acute MI; APACHE-II = Acute Physiology and Chronic Health Evaluation II; BP = blood pressure; CS = cardiogenic shock; GCS = Glasgow Coma Scale; ICU = intensive care unit; MCS = mechanical circulatory support; PAC = pulmonary artery catheterisation; PCWP = pulmonary capillary wedge pressure; RV = right ventricular; SCAI = Society for Cardiovascular Angiography and Interventions.

Table 4: Summary of Guidelines for Cardiogenic Shock

	AHA and/or ACC				ESC			
	2017 AHA[8]	2022 AHA Heart Failure[45]	2025 ACS ACC/ AHA/SCAI[37]	2025 ACC Concise CS[9]	2017 STEMI[44]	2020 ACCA ESC[7]	2021 Heart Failure[36]	2022 Shock to Survival[86]
Monitoring and Imaging								
Echocardiography	Advised	IC	IC	Advised	IC	Advised	IC	Advised
PAC	Suggested in uncertainty	IIa*	None	Suggested	IIb B	Suggested	IIb C	None
Arterial line	Advised	None	None	Suggested	IC	Suggested	IC	None
Revascularisation								
Early PCI	Advised	None	IB	Advised	IA	Advised	IC	Advised
Culprit PCI	Advised	None	IB	None	IB	Advised	None	None
Thrombolysis	Advised if PCI delayed	None	IA (if not for PCI)	None	IIa C (if PCI >120 min)	Suggested if PCI unavailable	None	None
CABG	Suggested†	None	IIa (if not suitable for PCI)	None	IB (if not for PCI)	Suggested if PCI unsuccessful	None	None
Mechanical Support								
IABP	IIA‡	None	IIA‡	Suggested§	IIA‡	IIA‡	III B	In select cases
MFP	Suggested (after MDT)	IIb	IIA	Suggested§	IIb C	Insufficient data	IIC	In select cases
ECMO	Suggested (after MDT)	IIb	IIIB	Suggested§	IIb C	Not routine	IIC	In select cases
Systems of Care								
Need for MDT	None	IIa	Advised	Advised	None	None	Advised	Advised
PCI transfer	Advised	None	IB	Advised	IC	Advised	IC	Advised
Transfer to specialist CS centre	Suggested	IIb	Suggested	Advised	None	None	Suggested	Advised

*Colour coding indicates overall direction of each recommendation: green, recommended or advised; yellow, may be considered or suggested in select circumstances; red, not recommended or potentially harmful.*Worsening or uncertain haemodynamics.†Discretion for multivessel or left main disease. ‡Acute mitral regurgitation or ventricular septal defect only, otherwise not recommended. §In select patient populations based on shock severity and phenotype. ACC = American College of Cardiology; ACCA = Acute Cardiovascular Care Association; ACS = acute coronary syndrome; AHA = American Heart Association; CABG = coronary artery bypass grafting; CS = cardiogenic shock; ECMO = extracorporeal membrane oxygenation; ESC = European Society of Cardiology; IABP = intra-aortic balloon pump; MDT = multidisciplinary team; MFP = microaxial flow pump (e.g. Impella); PAC = pulmonary artery catheterisation; PCI = percutaneous coronary intervention; STEMI = ST-elevation MI.*

Beyond staging, phenotyping may further refine risk assessment. Earlier approaches relied on single parameters such as lactate, but registry data and machine learning have enabled the identification of distinct CS phenotypes.[33] In 1,959 patients, Zweck et al. identified three phenotypes: non-congested (I), cardiorenal shock (II) and cardiometabolic shock (III).[33] Compared with phenotype I, patients with phenotype II (OR 3; 95% CI [1.8–5.1]) and phenotype III (OR 4.6; 95% CI [2.7–5.1]) had higher rates of mortality. Within the 410-patient Cardiogenic Shock Working Group AMI cohort, in-hospital mortality was 21%, 45% and 55% across phenotypes I, II and III, respectively.[34] Integration of phenotyping with SCAI staging further improves risk stratification and may support more individualised treatment strategies.[35]

Ultimately, the clinical utility of classification and phenotyping lies in supporting timely decision-making. The SCAI classification provides a dynamic framework for reassessment, where progression or failure to improve, such as transition to stage D, may identify a window for escalation before refractory shock develops.[30] In practice, rising lactate, low cardiac power output (CPO), worsening end-organ function or progression in SCAI stage should prompt reassessment of revascularisation, haemodynamic support, MCS suitability and transfer to a specialist centre. When integrated into structured systems of care, these frameworks support early triage and more consistent escalation decisions.

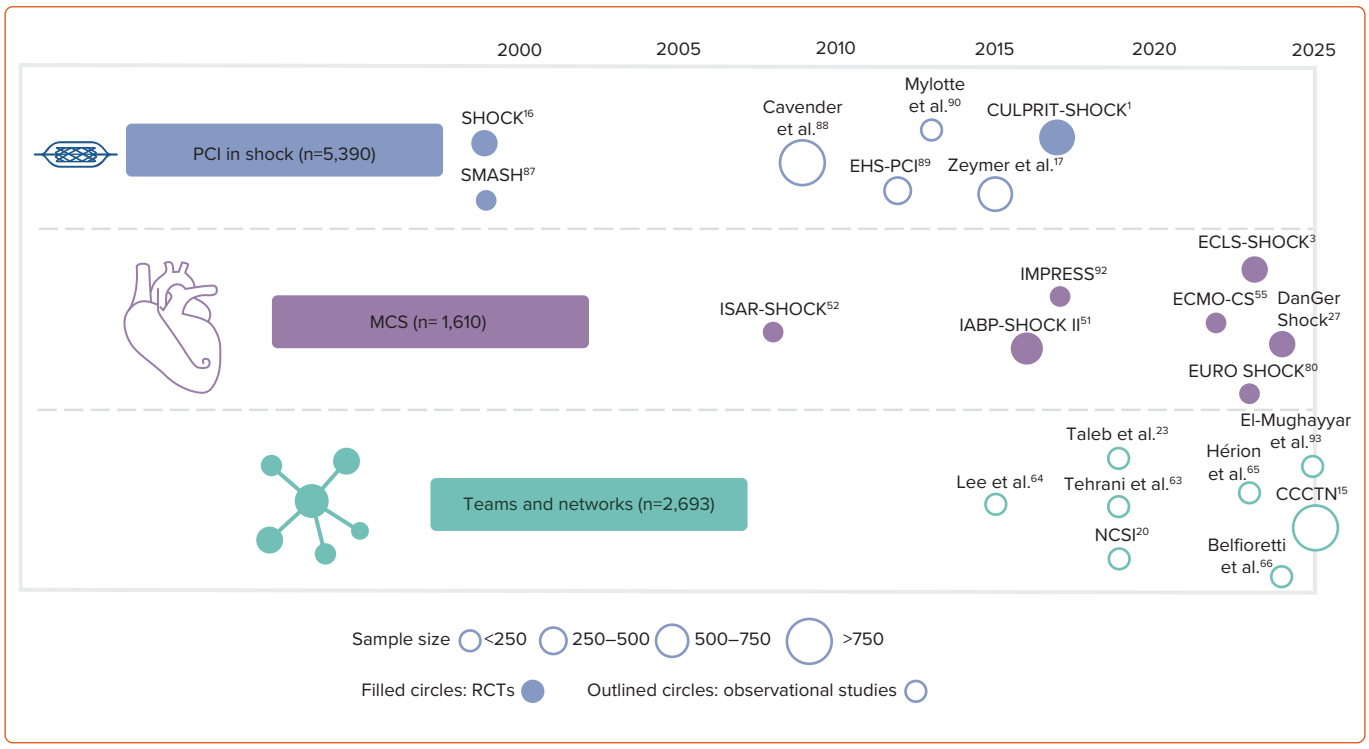
Management of Acute MI-related Cardiogenic Shock within Shock Centres

Treatment of AMI-CS is typically protocolised and delivered through multidisciplinary CS teams, integrating rapid diagnostic assessment, early revascularisation, invasive haemodynamic monitoring, selective MCS use and structured critical care. Trial outcomes should be interpreted within systems of care, because timing, patient selection and centre expertise may influence treatment effect. *Table 4* summarises levels of evidence for CS assessment and management across guidelines, while *Figure 2* highlights the timeline of published evidence.

Echocardiography

International guidelines emphasise the importance of echocardiographic assessment for CS, where this is considered mandatory for several aspects of AMI-CS care (ESC Class 1, Level C).[8,9,36,37] Immediate echocardiography can identify the underlying pathophysiology of AMI-CS in >95% of cases, including left ventricle and right ventricle impairment (in 70% and 30%, respectively), valvular function (mitral regurgitation present in 5–10% of patients) and pericardial effusion or severe mechanical complications in 5%.[38] Echocardiography also provides non-invasive estimates of haemodynamic parameters, including cardiac index, pulmonary artery pulsatility index (PAPi) and CPO, and plays a key role in serial assessment, as well as in guiding MCS device implantation and

Figure 2: History of Evidence in Cardiogenic Shock Complicating Acute MI



MCS = mechanical circulatory support; PCI = percutaneous coronary intervention.

weaning. Despite this, real-world uptake remains variable, with incomplete usage within the first 72 hours of critical care admission and substantial heterogeneity across centres.[39] This likely reflects differences in infrastructure, the availability of advanced imaging modalities and access to experienced operators, which may be more challenging in Level 2 or Level 3 centres.

These disparities have important implications for the design of regionalised systems of care, where consistent access to early echocardiographic imaging, potentially across all levels of care, is essential to standardise assessment, identify reversible pathology and guide escalation decisions.

Pulmonary Artery Catheterisation

Pulmonary artery catheterisation (PAC) is currently used in approximately 30% of AMI-CS patients, with substantial international variability. Although specific parameters, such as PAPi and CPO, may have a role in guiding aspects of care, the routine use of PAC for AMI-CS is controversial according to current evidence. Randomised data from a number of trials, including ESCAPE, have not shown benefit for PAC in either mortality or rehospitalisation in CS compared with clinical assessment.[40,41] Importantly, PAC placement was associated with complications (e.g. infection, pulmonary artery rupture, arrhythmias) in approximately 3–5% of patients.[42] However, these RCTs did not specifically evaluate PAC use in AMI-CS and do not reflect contemporary practice. Nevertheless, recent observational data suggest that PAC placement within dedicated CS initiatives for select patients with diagnostic uncertainty or non-responsiveness to initial treatment may be of benefit.[43] Clinical guidelines for PAC use in AMI-CS are consequently discrepant, with the ESC proposing a Class IIb recommendation, whereas the ACC/AHA guidelines reserve the use of PAC for refractory cases only.[44,45] Ongoing RCTs, such as the PACCS trial (NCT05485376), may address these evidence gaps.

Percutaneous Coronary Intervention

Coronary revascularisation remains a cornerstone of AMI-CS care and may be delivered across Level 1 and Level 2 centres. Several RCTs have assessed its role in AMI-CS care (summarised in *Table 5*). The SHOCK trial randomised 302 ST-elevation MI patients with shock to medical therapy (thrombolysis plus intra-aortic balloon pump [IABP]) or early coronary revascularisation (PCI/coronary artery bypass grafting and IABP) and was the first to show benefit with early revascularisation.[16] However, system-level factors, including timing of presentation, interhospital transfer and centre expertise, were not standardised or explicitly described within the trial design. Although underpowered to meet its primary outcome of 30-day all-cause mortality, early revascularisation was found to confer a 6-month survival benefit (50.3% versus 63.1%; RR 0.80; 95% CI [0.65–0.98]; p=0.027), which persisted at both 1 and 6 years.[46]

Following the SHOCK trial findings, the question of revascularisation extent (complete or culprit-only revascularisation) was addressed by CULPRIT-SHOCK, which randomised 706 AMI-CS patients to either culprit-only PCI (with optional staged intervention) or immediate multivessel PCI.[1] Culprit-only intervention was associated with a significantly lower 30-day composite outcome of all-cause mortality or renal replacement therapy (RR 0.83; 95% CI [0.71–0.96]; p=0.01), driven primarily by lower mortality. These findings should be interpreted in the context of trial design, where MCS use was not protocolised and broader system-of-care factors were not the primary focus.

Consequently, current ESC and AHA guidelines recommend culprit-only PCI in AMI-CS (ESC: Class I, Level B; AHA: Class I, Level B–R), with both assigning a Class III (harm) recommendation against multivessel PCI during the index procedure.[47,48] As practice evolves, the role and timing of non-culprit revascularisation in the context of dynamic CS deterioration warrants re-examination.

Table 5: Trials Exploring Percutaneous Coronary Intervention in Acute MI-related Cardiogenic Shock

Study	Study Design and Sample Size	Population	Interventions	Outcomes	Key Findings
Urban et al. SMASH 1999[87]	International multicentre RCT, n=55	Patients with acute MI and CS (SBP <90 mmHg despite fluids +/– inotropes) <48 h of admission	Early angiography +/– PCI/CABG versus no early revascularisation MCS permitted in both groups	Primary: all-cause mortality at 30 days	Trial halted early due to low patient recruitment Non-significant survival benefit in early revascularisation (69% versus 78%; RR 0.88; 95% CI [0.6–1.2]; p=0.68)
Hochman et al. SHOCK 1999[16]	International multicentre RCT, n=302	Patients with acute MI and CS within 36 h of onset	Early revascularisation (PCI or CABG) (n=152) versus initial medical therapy (n=150)	Primary: 30-day mortality Secondary: 6-month mortality	Early revascularisation significantly reduced 6-month mortality (50.3% versus 63.1%; RR 0.80; 95% CI [0.65–0.98]; p=0.027) compared with medical therapy; there was no significant difference in 30-day mortality
Cavender et al. 2009[88]	Retrospective observational study National cardiovascular data registry (US, 638 centres), total entries n=708,401	Analysis of patients with STEMI and multivessel disease, n=28,936 Subgroup analysis of those with CS, n=3,087	Comparison of outcomes in immediate multivessel PCI versus IRA-only PCI	Primary: in-hospital mortality	In patients with STEMI and CS, those treated with immediate multivessel PCI had increased in-hospital mortality (36.5% versus 27.8%; p = 0.01; adjusted OR 1.54; 95% CI [1.22–1.95])
Bauer et al. EHS-PCI registry 2012[89]	Prospective international multicentre (176) observational study Total PCIs, n=47,407	Patients with acute MI complicated by CS and with multivessel disease, n=336	Multivessel PCI, n=82 (24%) IRA-only PCI, n=254 (76%)	Primary: in-hospital mortality	Hospital mortality was greater in the multivessel PCI group, but this was not statistically significant (adjusted OR 1.28; 95% CI [0.72–2.28])
Mylotte et al. 2013[90]	Multicentre prospective observational study (1998–2010) at 5 French centres, total patients n=11,530	Sub-analysis of patients with STEMI, CS and cardiac arrest with multivessel disease, n=169	IRA-only PCI, n=103 Multivessel PCI (during index procedure), n=66	Primary: 6-month survival	6-month survival was significantly greater in the multivessel PCI group (43.9% versus 20.4%; p=0.0017)
Zeymer et al. 2015[17]	German multicentre prospective registry, n=735	Patients with acute MI and CS undergoing multivessel PCI (n=173) versus IRA-only PCI (n=562)	Multivariate regression analysis for in-hospital mortality	Primary: in-hospital mortality	Multivariate analysis showed multivessel PCI in CS associated with increased mortality (OR 1.5; 95% CI [1.15–1.84])
Thiele et al. CULPRIT-SHOCK 2017[1]	European open-label multicentre RCT, n=706	Patients with acute MI, CS (SBP <90 mmHg for >30 min or requiring catecholamines to maintain SBP >90 mmHg) and multivessel coronary artery disease	Immediate multivessel PCI for stenosis >70% (n=355) versus culprit-only PCI with an option for a staged procedure (n=351)	Primary: 30-day mortality or renal failure	Primary endpoint was lower in IRA-only PCI and associated with a 9.5% absolute risk reduction compared to multivessel PCI (RR 0.83; 95% CI [0.71–0.96]; p=0.01)

CABG = coronary artery bypass grafting; CS = cardiogenic shock; MCS = mechanical circulatory support; IRA = infarct-related artery; PCI = percutaneous coronary intervention; RCT = randomised control trial; SBP = systolic blood pressure; STEMI = ST-elevation MI.

Mechanical Circulatory Support in Acute MI-related Cardiogenic Shock

MCS is increasingly used in select AMI-CS patients across CS centres, with device choice and timing guided by haemodynamic profile and response to therapy, as well as institutional protocols. MCS has historically been used on the basis of physiological efficacy and observational data suggesting benefit.[49] Primarily, this includes venoarterial extracorporeal membrane oxygenation (VA-ECMO), offering full cardiopulmonary support, and unloading devices (including IABP and microaxial flow pumps, most commonly Impella [Abiomed]). There has been no clear benefit demonstrated when MCS is delivered to all-comers in CS.[50] However, specific devices targeted to specific CS subpopulations, particularly AMI-CS, have suggested potential mortality benefit (*Table 6*).

Initial evidence for MCS use in AMI-CS came from the IABP-SHOCK II trial, which randomised 600 patients with AMI-CS to IABP or no IABP. The trial demonstrated no reduction in 30-day mortality (HR 0.96; 95% CI [0.79–1.17]).[51] These findings informed Class III recommendations in international guidelines and contributed to a global decline in routine IABP use in AMI-CS.[37,44]

The ISAR-SHOCK study demonstrated the haemodynamic efficacy of Impella support, increasing cardiac output by around 1.0–3.5 l/min.[52] This precipitated the DanGer Shock trial, a 360-patient RCT that showed that the Impella group had a reduced 180-day mortality (45.8% versus 58.5%; HR 0.74; 95% CI [0.55–0.99]) in AMI-CS, primarily with ST-elevation MI.[27] Importantly, that study excluded patients with comatose OHCA (Glasgow Coma Scale <8), established multiorgan failure and with severe right ventricular failure, thereby reflecting a highly selected population of AMI-CS. Subsequent subgroup analysis showed improved lactate clearance and reduced vasoactive drug requirement in the Impella group.[53] Although there was increased use of renal replacement therapy in the Impella group, none of the patients required long-term haemodialysis.[53]

IABP and Impella offload the left ventricle to varying degrees, whereas VA-ECMO provides high-flow circulatory support (approximately 4–6 l/min) but at the expense of increasing left ventricular afterload and

Table 6: Randomised Trials of Mechanical Circulatory Support in Acute MI-related Cardiogenic Shock

Trial	Year	Cohort	Cardiac Arrest	Comparison	Key Inclusion Criteria	Primary Endpoint	Findings
Intra-aortic Balloon Pump							
IABP-SHOCK II[51]	2012	Multicentre RCT of 600 patients	45%	IABP versus no IABP	CS complicating AMI	30-day all-cause mortality	IABP did not reduce 30-day mortality
Microaxial Flow Pumps (Impella)							
DanGer Shock[27]	2024	International multicentre RCT of 360 patients	20%	Upfront Impella versus standard care	Patients with STEMI and CS	Death from any cause at 180 days	Lower risk of death from any cause at 180 days versus standard care
IMPRESS[92]	2017	Multicentre RCT	92%	Impella CP versus IABP	Severe shock complicating AMI	30-day all-cause mortality	Routine Impella not superior to IABP
ISAR-SHOCK[52]	2008	Two-centre RCT of 25 patients	77%	Impella versus IABP	All patients with AMI and compromised haemodynamic state receiving inotropic drugs	Change in the cardiac index from baseline to 30 min after implantation	Microaxial flow pump feasible and safe with superior haemodynamics versus IABP
VA-ECMO							
ECLS-SHOCK[3]	2023	Multicentre RCT	77%	Early ECLS versus standard care	AMI complicated by CS planned for early revascularisation	30-day all-cause mortality	ECLS did not lower risk of death at 30 days
EURO SHOCK[80]	2023	Multicentre pan-European RCT	49%	Upfront VA-ECMO versus standard care	Persistent CS 30 min after primary PCI	30-day all-cause mortality	COVID-19 impacted, but feasibility of such trials demonstrated
ECMO-CS[55]	2022	Multicentre RCT	77%	Immediate VA-ECMO versus no immediate VA-ECMO	Either rapidly deteriorating or severe CS	Composite of death from any cause, resuscitated cardiac arrest, implementation of another MCS device at 30 days	No improvement in clinical outcomes with immediate VA-ECMO

AMI = acute MI; CS = cardiogenic shock; ECLS = extracorporeal life support; IABP = intra-aortic balloon pump; MCS = mechanical circulatory support; PCI = percutaneous coronary intervention; RCT = randomised controlled trial; STEMI = ST-elevation MI; VA-ECMO = venous arterial extracorporeal membrane oxygenation.

mechanical work of the heart (in approximately 20%), particularly in the absence of concurrent mechanical unloading.[54] Recent data from high-volume US centres alongside two VA-ECMO RCTs have not shown benefit in AMI-CS.[3,55,56] High OHCA rates, cross-over and heterogeneity in the timing and use of adjunctive strategies, including left ventricular unloading, highlight important system-level factors not fully captured in the ECLS-SHOCK trial and should be considered when interpreting its findings.[3]

In summary, there is no evidence to suggest VA-ECMO as first-line treatment in AMI-CS. Its role in refractory cardiac arrest remains promising, but with significant logistical and implementation challenges.

Current international guidelines assign Impella/ECMO a Class IIb (Level C) recommendation (*Table 4*) for refractory CS based on limited evidence. However, guidelines for VA-ECMO are likely to be iterated to a more negative recommendation in light of recent trials.[44,45] A consensus statement prior to DanGer Shock publication emphasised that MCS application should be based on haemodynamic parameters including PAC, biochemical markers and overall clinical scenario.[57] Dedicated AMI-CS centres may possess the volume and expertise to more efficiently decide on the suitability, timing and device selection of MCS, although definitive evidence supporting this approach is lacking. More broadly, interpretation of randomised trial data in AMI-CS should be considered within the context of systems of care. Trial populations are often highly selected and do not reflect real-world practice. The effectiveness of therapies, particularly MCS, is influenced by timing, patient selection and centre expertise. Variations in these factors may contribute to neutral or negative trial results.

Care Beyond the Cardiac Cath Lab

The management of AMI-CS patients following revascularisation is highly heterogeneous, despite being a critical determinant of outcome. Although guideline recommendations exist for components of haemodynamic monitoring and management, there is a lack of randomised evidence or protocolisation in this setting.

In broader critical care populations, closed models (led by dedicated critical care teams rather than the admitting team) have been associated with improved outcomes, and emerging data suggest similar in the CICU setting.[58] Observational data also support cardiology intensivist comanagement in the coronary care unit.[59] Respiratory support is often needed, ranging from non-invasive strategies, such as high-flow nasal oxygen and continuous positive airway pressure or bilevel positive airway pressure, to invasive mechanical ventilation in severe cases, and may reduce the work of breathing and improve gas exchange, although AMI-CS-specific data are limited. Renal support, antimicrobials and nutrition also require intensivist-led MDT care.[25]

Inotropic and Multiorgan Support

Vasopressors, most commonly norepinephrine, are used to maintain perfusion pressure, whereas inotropes are used to augment cardiac output. Inotropes are used in up to 90% of AMI-CS patients after PCI despite risks of worsening ischaemia through increased myocardial work.[51] Although the SOAP II and OPTIMA-CC trials have suggested the superiority of norepinephrine over dopamine and epinephrine, generalisability to AMI-CS is limited.[60,61] There is no clear evidence to support the superiority of one vasoactive strategy over another in AMI-CS. Renal dysfunction complicates approximately 30% of cases of AMI-CS,

with 20% requiring renal replacement, although early initiation in the absence of classical indications confers uncertain benefit.[8,27,62]

Dedicated Centre Protocols for Acute MI-related Cardiogenic Shock Treatment

Contemporary AMI-CS management has evolved towards centre-based protocols integrating early recognition, multidisciplinary activation, invasive haemodynamic assessment and selective MCS use. Evidence supporting these approaches is largely observational, with important limitations including selection bias, incomplete cohort data capture and limited external validity. Nevertheless, although derived from observational data, several dedicated CS protocols delivered at Level 1 centres have reported improved outcomes compared with historical care (*Table 7*), despite marked heterogeneity in physiology-driven strategies and device usage.

The Detroit Cardiogenic Shock Initiative used standardised early MCS approaches. In a pilot cohort of 41 consecutive patients with AMI-CS in a Level 1 centre, 19.5% of patients were transferred from another site.[21] This protocol included a single-call activation with rapid invasive haemodynamic assessment and near-universal Impella use, with pre-PCI MCS application in 66% of patients. Escalation and weaning were guided by serial physiological metrics, including CPO <0.6 W and PAPi <0.9. In this small, select observational cohort, survival to discharge was 76%, exceeding historical benchmarks.[21]

The INOVA/Virginia programme evaluated outcomes before and after implementation of a multidisciplinary shock team, activated through a dedicated call, in 82 patients treated at a Level 1 centre.[63] The protocol incorporated early transthoracic echocardiography, coronary angiography and invasive haemodynamics in most patients. Shock was defined using haemodynamic criteria (cardiac index ≤1.8 l/min/m^2 [≤2.2 on inotropes] and pulmonary capillary wedge pressure ≥15 mmHg), with management and MCS selection guided by serial reassessment including CPO <0.6 W and PAPi. Daily joint critical care and shock team meetings facilitated management plans, including MCS escalation. Following protocol implementation, 30-day survival improved from 47% to 77% over two consecutive years.[63]

In Utah, an HF-led CS team managed a cohort of 123 patients with refractory CS requiring MCS, compared with 121 historical controls.[23] Management incorporated early invasive haemodynamic assessment and revascularisation where appropriate. Escalation to MCS was guided by haemodynamic criteria (cardiac index <2.2 l/min/m^2 or pulmonary capillary wedge pressure >15 mmHg), alongside regular shock team consultation. Implementation of the shock team was associated with higher in-hospital survival (61% versus 47.9%) and reduced 30-day mortality (HR 0.61; 95% CI [0.41–0.93]).[23]

The Ottawa programme reported outcomes in 100 patients with CS (64 managed with a code-shock protocol and 36 historical controls), representing a heterogeneous cohort with predominantly non-ischaemic aetiologies.[64] Approximately three-quarters of patients were transferred from external institutions, although this differed between groups (63% in the code-shock group versus 94% in the historical controls). CS was defined by reduced cardiac output (hypotension or low cardiac index) with evidence of end-organ hypoperfusion. Management was coordinated through a digitally enabled activation pathway with daily shock team review and included the selective use of MCS (45%). Although short-term outcomes were unchanged, with similar 30-day survival (~70%), programme implementation was associated with improved longer-term survival (67% versus 42%; HR 0.53).[64]

The Bordeaux experience evaluated 250 patients with refractory CS before and after implementation of a multidisciplinary shock team.[65] Selection for advanced support was guided by validated risk scores (including CARDSHOCK ≥7) alongside markers of hypoperfusion and end-organ dysfunction. One-year survival improved from 45% to 59%, with shock team management, which was independently associated with improved survival (HR 0.59; 95% CI [0.40–0.88]). A substantial proportion of patients received short-term MCS (n = 166), reflecting a highly selected population and introducing important selection bias.[65]

The Ancona programme evaluated protocolised CS care in 167 patients within a CICU using the Shock Academic Research Consortium definition of CS.[66] Ninety per cent of patients received MCS, primarily IABP. There were daily shock team consultations, without protocolised escalation pathways. This organisational model was associated with improvement in in-hospital survival from 43% to 71%, although the findings are limited by the observational study design and high device use.[66]

Finally, the ACT-SHOCK pathway (Bristol, UK) was a pragmatic, physiology-triggered shock team model embedded within a high-volume UK Level 1 centre.[67] Activation of a CS team was based on predefined markers of hypoperfusion (e.g. SBP <90 mmHg, heart rate <50 or >150 BPM, SpO_2 <90%, lactate >4 mmol/l, base excess <–5 mmol/l or left ventricular end-diastolic pressure >25 mmHg), and could be triggered prehospital, prior to PCI or during PCI (19.6%, 17.6% and 64.8% of patients, respectively). The revascularisation strategy was not protocolised and left to operator discretion. Unlike device-driven protocols, ACT-SHOCK prioritised rapid multispecialty mobilisation during PCI without mandated routine invasive haemodynamic monitoring or MCS. Critical care management was not protocolised, including decisions regarding MCS escalation. Overall, MCS use was low (14% Impella, 6% ECMO). In 165 patients, ACT-SHOCK was associated with lower 1-year mortality (35% versus 48.1%) and an independent reduction in adjusted 1-year death (HR 0.53; 95% CI [0.30–0.92]) compared with a historical control cohort with AMI-CS in the preceding 12 months.[67]

Collectively, these centre-based experiences demonstrate two important observations. First, that bespoke patient care pathways are associated with higher observed survival compared with conventional care. Second, that improved outcomes in contemporary AMI-CS may be driven less by any single device and more by structured systems of care that integrate early activation, haemodynamic assessment and protocolised escalation aligned with institutional expertise. Nevertheless, RCTs are now required to assess the value of the components of this specialised approach before they can be recommended in routine clinical care. Notably, many of these programmes included patients transferred from referring centres, highlighting the importance of coordinated regional systems to enable timely access to specialist care.

These findings should be interpreted with caution because most of the available evidence is observational and prone to bias. Differences in patient selection, survival to treatment and underlying illness severity, particularly in relation to interhospital transfer and the use of MCS, may influence outcomes. In addition, comparisons with historical cohorts and the lack of data on patients not selected for transfer or advanced therapies may further distort estimates of benefit.

Regionalised Cardiogenic Shock Pathways of Care

Early recognition, rapid triage and MDT-supported transfer between

Table 7: Heart Attack Centre Advanced Cardiogenic Shock Team Protocols

Study	Cohort	Activation Model	Shock Definition	Mandatory Investigations/Access	MCS Protocol and Usage	Post-procedural	Communication Model	30-day, Hospital or ICU Mortality
Basir et al. 2019[20] (National Cardiogenic Shock Initiative, US)	• 171 AMI-CS • Mean age 63 years • 77% male • 78% STEMI • 20% OHCA • 29% in-hospital arrest	• Early cath lab activation for AMI-CS • Shared institutional shock protocol • Impella prior to PCI and before escalation of inotropes	AMI plus ≥2 of: • SBP ≤90 mmHg or vasopressor/inotrope requirement • Evidence of hypoperfusion • Cardiac index <2.2 l/min/m^2 or CPO ≤0.6 W	• Invasive haemodynamics (RHC 92%) • Femoral large-bore access • CPO-guided haemodynamic management	• Impella-first strategy (100% Impella; 74% pre-PCI) • Haemodynamic-guided escalation • Persistent CPO <0.6 W + elevated lactate ➔ consider advanced MCS or durable LVAD evaluation	• Serial invasive haemodynamics • CPO + lactate at 12–24 h for risk stratification/escalation decisions • Monitor limb/access-site complications	• Multidisciplinary institutional shock protocol embedded within cath lab workflow • Shared team-based haemodynamic decision-making	• Survival to discharge 72%
Tehrani et al. 2019[63] (INOVA; Virginia, US)	• AMI-CS n=82 (within 204 total CS cohort) • Mean age 64 years • 71% male • 52% transferred from outside institutions	• Single-call 'shock line' activation • Immediate MDT mobilisation (interventional cardiology, cardiac surgery, advanced HF, critical care) • ED activated team for out-of-hospital AMI-CS	• SHOCK trial criteria: SBP <90 mmHg ≥30 min (or vasopressors) + end-organ hypoperfusion • Cardiac index ≤1.8 l/min/m^2 (≤2.2 on vasopressors) and PCWP ≥15 mmHg • CPO <0.6 W and PAPi <1.0 incorporated into 24-h reassessment	• Protocolised invasive haemodynamic assessment (RHC performed in 82%) • Coronary angiography and revascularisation for AMI-CS • Transthoracic echocardiography • Peripheral vascular assessment prior to large-bore access	• Algorithm-driven MDT selection • Early percutaneous MCS consideration • Escalation guided by CPO <0.6 W and PAPi <1.0 • Impella CP most common; VA-ECMO for severe/ biventricular failure; escalation from IABP where required	• Daily bedside echocardiography if MCS used • Serial lactate, CPO and PAPi monitoring • Structured VA-ECMO management and weaning algorithms • Referral for LVAD/transplant in refractory CS	• Daily CICU–shock team review regarding escalation/weaning • Centralised MDT oversight throughout admission	• 30-day survival improved from 47% pre-team to 58% (2017) and 77% (2018)
Taleb et al. 2019[23] (Utah, US)	• Refractory CS treated with short-term MCS: shock team (n=123) versus control (n=121) • Mean age 57 years • 78% male • ACS 61% • 67.5% transferred from referring hospital	• Single-page activation; HF cardiologist triage as central hub • Shock team: HF cardiologist, cardiothoracic surgeon, interventional cardiologist, CICU attending • After-hours coordinated discussion among on-call members	• SBP <90 mmHg or MAP <50 mmHg for >30 min (or IV vasoactive support) • Plus ≥1 of: PCWP or LVEDP >15 mmHg with cardiac index <2.2 l/min/m^2, pulmonary oedema or end-organ hypoperfusion	• Arterial line placement • RHC • Coronary angiography/ revascularisation when indicated • Haemodynamic assessment before escalation to MCS	• Short-term MCS for persistent shock despite optimal medical therapy (cardiac index <2.2 l/min/m^2 with PCWP/ LVEDP >15 mmHg and/or impaired end-organ perfusion) • Device selection by full team consensus • IABP, Impella and VA-ECMO used • Device type not independently associated with survival	• Team continues management until shock resolution or decision to de-escalate • Weaning with clinical improvement • Escalation to durable LVAD in 34 patients among survivors	• Centralised multidisciplinary oversight throughout admission • HF cardiologist coordinates decisions	• 30-day mortality reduced (HR 0.61; 95% CI [0.41–0.93]) • In-hospital survival 61.0% (shock team) versus 47.9% (control)

Table 7 (cont)

Study	Cohort	Activation Model	Shock Definition	Mandatory Investigations/Access	MCS Protocol and Usage	Post-procedural	Communication Model	30-day, Hospital or ICU Mortality
Lee et al. 2020[64] (Ottawa, Canada)	• Code Shock (n=64) versus control (n=36) • Median age 55 (IQR 43–64) versus 64 years • 78% male (treatment) • New HF 61% versus 36% • AMI 13% • Cardiac arrest 20%	• Stepwise 4-stage protocol: identification ➡ CCU senior resident review ➡ advanced HF review ➡ Code Shock activation • Smartphone-based application enabling virtual MDT discussion	CS defined as: • Physiology: SBP <90 mmHg or MAP <60 mmHg (≥30 min), single moderate inotrope or ≥2 inotropes or cardiac index <1.8 l/min/m^2 • Plus hypoperfusion: oliguria, AKI, ALI or lactate ≥2 mmol/l • Exclusion criteria applied prior to activation	• CCU confirmation of shock prior to activation • PAC used in 66% (treatment) versus 50% (control) • Invasive haemodynamic profiling common but not mandated	• Shock team determined candidacy, timing and device choice • Temporary MCS used in 45% (treatment) versus 28% (control) • IABP most common; Impella and VA-ECMO used (including combination strategies) • Durable LVAD/ transplant evaluation incorporated	• Daily shock team rounds • Serial end-organ perfusion assessment • Ongoing reassessment for escalation/de-escalation and durable therapy pathways • Higher HF specialist follow-up after discharge	• Smartphone-enabled virtual MDT activation (advanced HF, interventional cardiology, cardiac surgery, intensive care) • Continuous digital updates between reviews	• No significant difference in 30-day survival (72% versus 69%) or survival to discharge (69% versus 61%) • Improved long-term survival 67% versus 42% (HR 0.53; 95% CI [0.28–0.99])
Hérion et al. 2023[65] (Bordeaux, France)	• n=250 (166 shock team, 84 control) • Median age 53 (IQR 45–61) years • 55% transferred	• In-person shock team meeting • Team: cardiac anaesthetist–intensivist, interventional cardiologist, cardiac surgeon • Most commonly activated by cardiac ICU physician • 24/7 availability	• SBP <90 mmHg and cardiac index <2.0 l/min/m^2 • ± pulmonary congestion (PCWP >15 mmHg or CXR) • Plus ≥1 refractory criterion (including high-dose inotropes and SvO_2 <55% as per paper)	• Transoesophageal echocardiography • PAC • Arterial blood gas and SvO_2 assessment	Algorithm-driven device selection: • Impella CP/5.0 if no hypoxaemia and no RV dysfunction • VA-ECMO if RV dysfunction without hypoxaemia • VAV-ECMO if hypoxaemia with RV dysfunction • LV unloading (IABP or Impella) added to ECMO when LV distension/sludge or persistent PCWP >15 mmHg	• Not specified	• In-person multidisciplinary decision-making at activation • Ongoing specialist input with HF specialist involvement (as described)	• 1-year survival 59% versus 45% (p=0.043) • Shock team independently associated with improved 1-year survival (HR 0.592; 95% CI [0.398–0.880]; p=0.010)
Belfioretti et al. 2024[66] (Ancona, Italy)	• AMI-CS admitted to CICU (n=167) • 67% male • Median age 71 (IQR 61–80) years	• Shock team: cardiac intensivist, interventional cardiologist, anaesthetist, cardiac surgeon • Role includes identifying patients who may benefit from MCS	• Hypotension with peripheral hypoperfusion • SBP <90 mmHg for >30 min or catecholamines to maintain SBP >90 mmHg • Lactate >2 mmol/l	• Coronary angiography 100% • Primary PCI 81%	MCS used in 91.1%: • 65% IABP • 23% Impella CP • 4% VA-ECMO	• Not described	• Not explicitly specified as a single-call/pager model • Team-based approach described after shock team implementation	• In-hospital mortality decreased from 57% to 29% over study period (per-year OR 0.90; p=0.0015)

Table 7 (cont)

Study	Cohort	Activation Model	Shock Definition	Mandatory Investigations/Access	MCS Protocol and Usage	Post-procedural	Communication Model	30-day, Hospital or ICU Mortality
El-Mughayyar et al. 2025 [93] (St John Canada)	• CS n=80 (6.9% of screened admissions) • Mean age 64.6 ± 12.3 years • 34% female • Ischaemic 79% • Cardiac arrest 45%	• Direct communication between CCU attending, interventional cardiologist and cardiac surgeon (virtual or in-person) • Activation in 35/80 (44%)	• Hypotension (SBP <90 mmHg/MAP <60 mmHg, cardiac index < 1.8 l/min/m^2, inotrope support) plus hypoperfusion • SCAI C–E	• No mandatory stipulations • Activation associated with more invasive monitoring: • arterial line 91% versus 53% • central line 94% versus 51% • PAC 49% versus 7%	• IABP and ECMO available • MCS use 51% (activated) versus 2% (not activated)	• Not protocolised; process-focused implementation study	• Three-physician MDT model (CCU, interventional cardiology, cardiac surgery) • Rapid virtual-capable discussion	• In-hospital mortality 35% • No difference with activation (34% versus 36%)
ACT-SHOCK, 2024[67] (Bristol, UK)	• AMI-CS n=165 • ACT input (n=82) versus standard care (n=83) • Age 71 ± 1.3 versus 67 ± 1.3 years • Male 68% versus 71%	• Advanced CS team (interventional cardiology, intensive care, anaesthesia, cardiac physiology) • Activation before, during or after PCI	• Physiology-triggered criteria including: • SBP <90 mmHg • Heart rate <50 or >150 BPM • SpO_2 <90% • Lactate >4 mmol/l • Base excess <–5 mmol/l • LVEDP >25 mmHg	• No routine invasive haemodynamic profiling; pragmatic physiology-guided model	• Advanced MCS (Impella, ECMO) available • Non-mandated, clinician-directed use	• Physiology-triggered escalation criteria (e.g. SBP <90 mmHg, lactate >4 mmol/l, base excess <–5 mmol/l, urine output <0.3 ml/kg/h, SpO_2 <90%)	• Structured multidisciplinary coordination during PCI and early post-PCI care	• 30-day mortality 34% versus 37% • 1-year mortality 35% versus 48.1%

ACS = acute coronary syndrome; AKI = acute kidney injury; ALI = acute liver injury; AMI = acute MI; CCU = coronary care unit; CICU = cardiac intensive care unit; CPO = cardiac power output; CS = cardiogenic shock; CXR = chest X-ray; ECMO = extracorporeal membrane oxygenation; ED = emergency department; HF = heart failure; IABP = intra-aortic balloon pump; ICU = intensive care unit; IQR = interquartile range; LV = left ventricle; LVAD = left ventricular assist device; LVEDP = left ventricular end-diastolic pressure; MAP = mean arterial pressure; MCS = mechanical circulatory support; MDT = multidisciplinary team; OHCA = out-of-hospital cardiac arrest; PAC = pulmonary artery catheterisation; PAPi = pulmonary artery pulsatility index; PCI = percutaneous coronary intervention; PCWP = pulmonary capillary wedge pressure; RHC = right heart catheterisation; RV = right ventricle; SCAI = Society for Cardiovascular Angiography and Interventions; STEMI = ST-elevation MI; VA-ECMO = veno-arterial extracorporeal membrane oxygenation.

centres of differing capability may be associated with improved outcomes, with decisions guided by expert teams and clear protocols.

In a large observational analysis of 412,000 CS hospitalisations between 2010 and 2014 in the US, in-hospital mortality was lower at hub centres than in spoke hospitals both for direct admissions (39.3% versus 47.8%) and for patients transferred to hubs (33.4% versus 47.8%).[19] On multivariable analysis, both direct admission to a hub (OR 0.86) and interhospital transfer (OR 0.72) were associated with lower mortality after adjustment. These findings suggest that transfer to a hub centre may be associated with improved survival, although this likely reflects the selection of patients sufficiently stable for transfer and causality cannot be inferred.

Operational regionalisation is particularly important for AMI-CS, where timely coronary reperfusion and access to advanced therapies require coordinated delivery across specialised centres. However, high-quality evidence defining optimal transfer criteria, the timing of transfer and pretransfer management remains limited. Existing data are derived predominantly from observational studies focused on Level 1 centres and are subject to important biases, including incomplete capture of network activity and lack of outcome data for patients not selected for transfer, which may overestimate the apparent benefit of centralised care.

Multidisciplinary Cardiogenic Shock Hub Multidisciplinary Teams

A central component of these systems is the provision of a cardiogenic shock MDT, typically a team of specialists coordinating care from Level 1 centres to referring Level 2 and Level 3 hospitals. Activation may occur prehospital, upon emergency department arrival or during PCI, enabling rapid MDT decisions on local management, transfer or escalation.

The composition of MDTs, transfer criteria and protocols vary between systems, particularly for AMI-CS. Although MDT-led care may support more coordinated decision-making across a network, the absence of transparent evidence-based transfer criteria may introduce variability and potential delays. Regionalisation within the three principal reported North American programmes is summarised in *Table 8*.

The National Cardiogenic Shock Initiative represents a multicentre standardisation model of care centred around Level 1 centres rather than a tightly governed regionalised network.[20] Although no emergency medical services bypass or formal PCI prior to transfer mandate was reported, centres used a 24/7 single-call activation with cath lab preactivation. Transfer decisions were guided by conventional CS criteria, including hypotension or inotrope/vasopressor requirement, hypoperfusion and a cardiac index <2.2 l/min/m^2 or cardiac power output ≤0.6 W. Overall, 27% of patients were transferred from external institutions (including 16% receiving MCS prior to transfer). On multivariable analysis, interhospital transfer was independently associated with higher in-hospital survival, although this may reflect the selection of patients suitably stable for transfer. MDT activation included interventional cardiology, critical care and perfusion teams. However, no formal spoke–hub triage algorithm or screening log of non-transferred patients was used.

The University of Utah Cardiac Recovery Program reflects a quaternary hub-and-spoke model centred on interhospital transfer. In the model, shock identification occurs at the spoke hospital, with subsequent transfer to a dedicated HF-led cardiac recovery team integrating interventional cardiology, cardiac surgery and MCS expertise.[23] Activation operates 24/7, and CS was defined using conventional criteria, including hypotension or vasopressor requirement, cardiac index <2.2 l/min/m^2, elevated filling pressures and evidence of end-organ hypoperfusion. PCI before transfer was not mandated but recommended if at a PCI-capable spoke hospital. Earlier transfer and shorter spoke dwell time were associated with improved survival.[23] Although over 700 patients were transferred, no comprehensive screening log of non-transferred patients was reported.

The Inova Heart and Vascular Institute network provides the most explicit regional governance structure, evolving from earlier single-centre shock team experience.[22,63] Regionalisation begins at first hospital presentation, without a formal emergency medical services bypass pathway. Thus, Level 2 and Level 3 centres transfer AMI-related shock patients directly to a Level 1 hub via a structured single-call pathway, generally without prior PCI, although initial stabilisation, including with MCS, may be undertaken at the spoke centre. Conventional CS criteria are used, with most patients presenting to Level 1 centres having an SCAI Grade C–E. Invasive haemodynamic phenotyping at the hub guides escalation, with decisions taken by an MDT integrating HF, interventional cardiology, critical care and cardiothoracic surgery specialists. A uniform algorithm for transfer and activation is applied across all centres with biweekly hub–spoke quality review. Clinical and administrative champions at spoke centres reinforce network fidelity, although a screening log of non-transferred patients does not exist.

Across reported regional models, several common features emerge, including single-call activation, predefined shock criteria and hub-based MDT review. However, approaches differ in key areas, including whether PCI is undertaken before transfer, whether MCS is initiated at the spoke centre and how transfer decisions are operationalised. In particular, no model provides robust comparative evidence to determine whether AMI-CS patients should undergo culprit PCI locally before transfer or be transferred directly to a Level 1 hub. The absence of screening logs and outcomes for non-transferred patients further limits comparison between systems and prevents firm conclusions regarding optimal transfer strategy.

Collectively, these models demonstrate that single-call activation, predefined shock criteria and rapid MDT mobilisation underpin effective regional systems.

Out-of-hospital Cardiac Arrest and Cardiogenic Shock

OHCA represents abrupt circulatory collapse in the community, with CS complicating up to half these presentations and contributing to high mortality.[32] Recent observational data highlight the impact of cardiac arrest on outcomes in AMI-CS beyond clinical trial populations, with both OHCA and in-hospital cardiac arrest associated with significantly worse survival.[68] In a simulated cohort study of 2,331 resuscitated OHCA patients from EUCAR and GLOBAL-MIRACLE, eligibility for landmark AMI-CS trials was low and varied substantially by trial criteria, with ECLS-SHOCK-like criteria selecting a higher-risk phenotype and DanGer Shock-like criteria selecting a more favourable cardiac phenotype.[69]

Postarrest CS exhibits a distinct pathophysiology characterised by global ischaemia–reperfusion injury, myocardial stunning and systemic inflammation, resulting in profound haemodynamic instability. Within

Table 8: Regionalised Shock Initiative Protocols

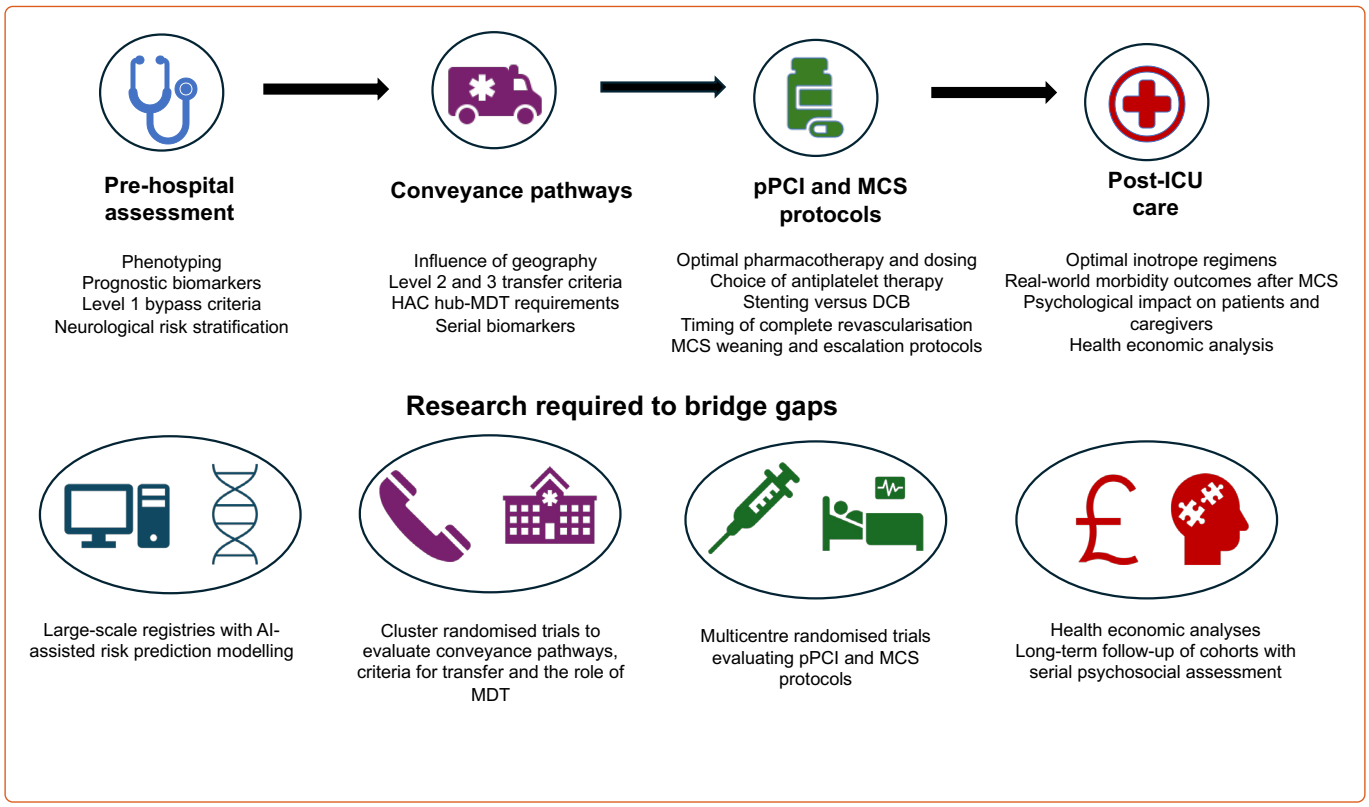
Centre/Registry	Prehospital Expedited Conveyance to Level 1	Interhospital Transfer and Management	Transfer Criteria	Hub Shock MDT	Outcomes of Patients in Each Level	Level of Evidence (ESC)*
NCSI[20,94]: Prospective multicentre implementation of a standardised early MCS (Impella-first) shock protocol across participating 80 US centres	No EMS bypass protocol reported.	**PCI** • No mandated PCI-before-transfer protocol described • Protocol emphasised MCS placement before PCI • Cath lab pre-activation at receiving centre reported **MCS** • Protocolised Impella-first strategy with MCS before PCI • 16% received MCS before transfer **Transfer** • 27% transferred from another institution • Standardised protocol implemented across participating centres • Specific interhospital triage decision framework not detailed	**AMI-CS defined as:** AMI plus ≥2 of: • SBP ≤90 mmHg or vasopressor/inotrope requirement • Evidence of hypoperfusion • Cardiac index <2.2 l/min/m^2 or CPO ≤0.6 W	**Team** •Interventional cardiology, critical care, perfusion team **Operation time**: 24/7 activation system **Format of discussion** • Single-call shock activation with in-hospital mobilisation • No formal spoke–hub tele-triage algorithm described	Outcomes reported for protocol-treated cohort only; no stratified level mortality data Supplementary material lists exclusion categories; total screened denominator not reported (criteria not mutually exclusive) **Outcomes:** Survival to discharge 72% Inter-hospital transfer independently associated with lower in-hospital mortality (OR 0.4; 95% CI [0.18–0.92])	**Level C** (prospective multicentre observational registry with protocol implementation; no randomised comparator)
University of Utah[23,91]: Prospective observational registry of patients developing CS at a spoke and transferred to a quaternary hub	Protocol begins at presentation to spoke hospital No EMS bypass protocol reported	**PCI** • No mandated PCI before transfer protocol •PCI capability at spoke and hub **MCS** • 39% received temporary MCS, of which 80% at spoke site before transfer **Transfer** • No predefined regional transfer algorithm described • 712 transferred from spokes; 412 with CS form study cohort	**CS defined as**: • SBP <90 mmHg or MAP <65 mmHg • Evidence of hypoperfusion • Cardiac index <2.2 l/min/m^2 or inotropic support SCAI stage assessed at hub	**Team** • Utah Cardiac Recovery Shock Team (established 2015) • Advanced HF, interventional cardiology, cardiac surgery and MCS teams **Operation hours**: 24/7 **Format of discussion** • Single-page shock team activation • HF cardiologist screens cases; MDT discussion at hub level • No documented protocolised spoke–hub algorithm	No reporting of spoke CS patients not transferred No formal triage log **Outcomes**: • 69.7% survived to discharge (30.3% in-hospital mortality) • 67% 30-day survival; 60.7% 1-year survival • 59.7% native heart survival • 10% required advanced HF therapies Independent predictors of improved survival: • Shorter spoke stay • Earlier MCS deployment	**Level C** (prospective observational registry of transferred patients without control group)

Table 8 (Cont)

Centre/Registry	Prehospital Expedited Conveyance to Level 1	Interhospital Transfer and Management	Transfer Criteria	Hub Shock MDT	Outcomes of Patients in Each Level	Level of Evidence (ESC)*
Inova/Virginia Shock Registry (INOVA-SHOCK)[95]: Prospective evaluation of standardised regional CS (AMI-CS and HF-CS) network across a Level 1 (hub) and Level 2/3 (spoke) model	Network begins at hospital presentation No formal EMS bypass algorithm specified	**PCI** • STEMI without shock ➜ PCI at presenting PCI-capable centre • AMI-CS at Level 2/3 ➜ transfer to Level 1 hub via structured referral pathway • No mandated PCI-before-transfer strategy for shock patients described **Transfer** • Level 2 and Level 3 spoke centres transfer AMI-CS to Level 1 hub • Single-call activation initiates hub acceptance and transfer • Structured hub-and-spoke referral model illustrated in supplementary algorithm	**CS defined as:** • SBP ≤90 mmHg or vasopressor requirement • Evidence of hypoperfusion • Cardiac index <1.8 l/min/m^2 with elevated filling pressures Majority SCAI C–E at presentation	**Team** • Multidisciplinary shock team including HF, interventional cardiology, critical care and cardiothoracic surgery **Format of discussion** • Single-call MDT activation at hub • By emergency room, other hospital units or other hospitals • Uniform treatment algorithm across centres. • Biweekly hub–spoke quality review with feedback • Spoke centres had clinical and administrative champions	No screening log or outcomes for patients not transferred After implementation of the regional shock network: **Outcomes:** • In-hospital survival improved over study years (47% ➜ 58% ➜ 77%) • CICU mortality reduced compared with pre-network era • Increased use of advanced MCS	**Level C** (prospective observational network evaluation with temporal comparisons)

**Level of evidence is classified according to the 2024 European Society of Cardiology grading system. All regional shock network studies are based on observational registry data without randomised comparison and therefore represent Level C evidence. AMI = acute MI; CICU = cardiac intensive care unit; CPO = cardiac power output; CS = cardiogenic shock; EMS = emergency medical services; ESC = European Society of Cardiology; HF = heart failure; MAP = mean arterial pressure; MCS = mechanical circulatory support; NCSI = National Cardiogenic Shock Initiative; PCI = percutaneous coronary intervention; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions.*

Figure 3: Evidence Gaps in Shock Centre Management for Acute MI-related Cardiogenic Shock



AI = artificial intelligence; DCB = drug-coated balloon; HAC = heart attack centre; ICU = intensive care unit; MCS = mechanical circulatory support; MDT = multidisciplinary team; pPCI = primary percutaneous coronary intervention.

regionalised systems of care, OHCA represents a particular challenge, because triage and transfer decisions must balance potential haemodynamic recovery against the risk of severe neurological injury. Neurological outcome is a key determinant of prognosis and frequently limits the benefit of advanced therapies.[70] Although MCS may improve haemodynamics, evidence for clinical benefit in OHCA remains inconsistent and inconclusive across observational studies and early randomised trials.[71,72] Within regional systems, extracorporeal life support/ECMO may provide haemodynamic support in select patients with refractory arrest or postarrest shock, but its benefit remains uncertain and is highly dependent on early patient selection, neurological prognosis, timing and centre expertise.[73]

Risk stratification models, including the MIRACLE$_2$ score, have been shown to have equivalent performance in those with concomitant CS and may support decision-making by identifying patient subgroups most likely to benefit from escalation of care.[74,75] Integration of such approaches into regionalised pathways has the potential to refine triage, optimise resource use and improve patient selection. An increasing proportion of patients present with advanced age, comorbidity and limited physiological reserve. In such patients, early identification of poor prognosis using clinical assessment and risk stratification tools may support decision-making regarding escalation of care. The incorporation of palliative care principles should be considered within MDT discussion and regional transfer pathways.

Survivorship, Rehabilitation and Research

Long-term survival following AMI-CS is favourable among patients discharged alive, but is frequently accompanied by substantial morbidity.[46] Survivors, particularly those with concomitant OHCA, often experience persistent physical, cognitive and psychological impairment, in addition to complications related to critical illness, MCS and cardiac surgery.[76] Data on late mortality, functional recovery and rehabilitation after AMI-CS remain limited, particularly among patients with concomitant OHCA.

Within regionalised systems of care, survivorship should be considered an integral component of specialist centre delivery rather than a downstream consequence of acute management. Although cardiac rehabilitation improves outcomes following AMI, evidence in AMI-CS remains limited and access is variable.[77] Specialist centres may be uniquely positioned to coordinate the rehabilitation and management of CS-specific complications. The integration of these processes within regional networks will be essential to optimise long-term outcomes.

Future Directions

Advancing care in AMI-CS requires improvements in diagnosis, treatment and systems of care. Key evidence gaps and proposed research priorities are summarised in *Figure 3*. Standardisation of definitions and classification, with integration of haemodynamic, clinical and biochemical markers, is needed to better characterise disease severity and phenotype.[25] Advances in machine learning and large-scale registries may enable more precise risk stratification. Uncertainty remains regarding the optimal timing and selection of PCI, MCS and post-procedural care, particularly across differing centre capabilities. Expansion of regional shock networks, including improved prehospital stratification and streamlined MDT decision making, alongside large-scale, adaptive randomised trials and collaborative research, will be essential to guide future care pathways. ❑

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