Management of cardiogenic shock in patients with acute coronary syndromes

ABSTRACT

Cardiogenic shock remains a major problem affecting a large proportion of patients with acute coronary syndromes, with a persistent high mortality rate. Although mechanical reperfusion with percutaneous coronary intervention has improved outcomes following acute coronary syndromes, there is limited evidence supporting the other current treatments used to manage patients with cardiogenic shock (intra-aortic balloon pumps, percutaneous left ventricular assist devices and extracorporeal membrane oxygenation). This article looks at these options, assessing current evidence and recent advances. It also discusses areas that still require research to ensure there is improvement in these high-risk patients, such as coordinated regionalised approaches to cardiogenic shock management with multidisciplinary care provided in designated tertiary shock centres.

Cardiogenic shock complicates the management of 7–10% of patients with acute coronary syndromes, and carries a high mortality rate (40–50%), being the commonest cause of death in patients with acute coronary syndromes (Goldberg et al, 2009). Mechanical reperfusion with primary percutaneous coronary intervention and advances in pharmacological treatment have improved outcomes following acute coronary syndromes (Keeley et al, 2003) but the highest risk patients who present with acute coronary syndrome continue to lag behind. In addition to percutaneous coronary intervention or coronary artery bypass grafting, catecholamines, fluids, intra-aortic balloon pumping and percutaneous left ventricular assist devices are widely used for management of cardiogenic shock. However, there is only limited evidence for any of these treatments except early revascularization, with recent data suggesting that some of these widely used therapies, e.g. intra-aortic balloon pumping, and strategies, e.g. complete coronary revascularization, may not provide the benefit first thought. This article outlines the diagnostic criteria, underlying causes and treatment of cardiogenic shock complicating acute coronary syndromes, providing a focused update on therapeutic advances, both mechanical and pharmacological, and recent clinical trial data.

Dr Krishnaraj S Rathod, Interventional Cardiology Specialist Registrar, Barts Interventional Group, Barts Heart Centre, Barts Health NHS Trust, London and Doctoral Research Fellow, NIHR Biomedical Research Centre at Barts, Barts Health NHS Trust and Queen Mary University of London, London

Dr Alexander Sirker, Consultant Cardiologist, Barts Interventional Group, Barts Heart Centre, Barts Health NHS Trust, London and NIHR Biomedical Research Centre at Barts, Barts Health NHS Trust and Queen Mary University of London, London

Professor Andreas Baumbach, Consultant Cardiologist, Barts Interventional Group, Barts Heart Centre, Barts Health NHS Trust, London and Professor of Cardiology and Professor for Device Innovation, NIHR Biomedical Research Centre at Barts, Barts Health NHS Trust and Queen Mary University of London, London

Professor Anthony Mathur, Consultant Cardiologist, Barts Interventional Group, Barts Heart Centre, Barts Health NHS Trust, London and Professor of Cardiology, NIHR Biomedical Research Centre at Barts, Barts Health NHS Trust and Queen Mary University of London, London

Dr Daniel A Jones, Consultant Cardiologist, Barts Interventional Group, Barts Heart Centre, Barts Health NHS Trust, London EC1A 7BE and Senior Clinical Lecturer, NIHR Biomedical Research Centre at Barts, Barts Health NHS Trust and Queen Mary University of London, London

Correspondence to: Dr DA Jones (daniel.jones@qmul.ac.uk)

Definition of cardiogenic shock

Cardiogenic shock is characterized by inadequate tissue perfusion as a result of a reduced cardiac output which leads to hypotension, pulmonary congestion and impaired vital organ or tissue perfusion. Cardiogenic shock is generally defined clinically, but some trials have used additional haemodynamic parameters to define cardiogenic shock, such as assessment of left ventricular filling pressures or cardiac index (Hochman et al, 1999). Although not mandated, these objective haemodynamic parameters can help confirm the diagnosis and enable comparison across cohorts and clinical trials. Definitions in clinical practice guidelines and operationalised definitions used in key clinical trials are presented in *Table 1*.

Incidence and outcome

The incidence of cardiogenic shock in patients with acute coronary syndromes differs depending on the definition of cardiogenic shock and the treatment era, ranging from 4% to 15%. It is generally accepted by large contemporary series from Denmark, the UK and the USA that rates of cardiogenic shock are increasing with little change in mortality. A report from the Nationwide Inpatient Sample Database in the USA between 2003 and 2010 demonstrated a rise in the prevalence of cardiogenic shock from 6% to 10% in the overall population. Furthermore, in patients >75 years of age (with ST elevation myocardial infarction), the prevalence increased from 7% to 12% (Kolte et al, 2014). In Denmark a study conducted over 1 year in two centres, covering two-thirds of the Danish population (Obling et al, 2018), observed cardiogenic shock in 10% of all ST elevation myocardial infarction

© 2019 MA Healthcare Ltd

Table 1. Definitions of cardiogenic shock used in clinical trials and guidelines

	Definition	
Trial	**Clinical criteria**	**Haemodynamic criteria**
SHOCK trial (in myocardial infarction complicated by left ventricular dysfunction (predominantly)) (Hochman et al, 1999)	Systolic blood pressure <90 mmHg for ≥30 minutes or support to maintain systolic blood pressure ≥90 mmHg and end-organ hypoperfusion (urine output <30 ml/h or cool extremities)	Cardiac index of ≤2.2 litre/min/m^2 and pulmonary capillary wedge pressure ≥15 mmHg
IABP-SHOCK II (in acute myocardial infarction) (Thiele et al, 2013)	Systolic blood pressure <90 mmHg for ≥30 minutes or catecholamines to maintain systolic blood pressure >90 mmHg and clinical pulmonary congestion and impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 ml/h or lactate >2.0 mmol/litre)	
European Society of Cardiology heart failure guidelines (Steg et al, 2012)	Systolic blood pressure <90 mmHg for ≥30 minutes or support to maintain systolic blood pressure ≥90 mmHg and end-organ hypoperfusion (urine output <30 ml/h or cool extremities)	Cardiac index of ≤2.2 litre/min/m^2 and pulmonary capillary wedge pressure ≥15 mmHg

IABP-SHOCK II = Intraaortic Balloon Pump in Cardiogenic Shock II; SHOCK = SHould we emergently revascularise Occluded Coronaries for cardiogenic shocK.

patients, which was present on admission in over half the cases, with only a small portion (28%) developing shock post-procedure. This was still associated with mortality rates of 50%. Similar findings have been reported from a 10-year survey of all primary percutaneous coronary interventions performed in London (Rathod et al, 2018). The overall rate of cardiogenic shock complicating ST elevation myocardial infarction was 8.9%, but with increasing rates from 7.0% in 2005 to 13% in 2015. It is not clear why this rate increased, but one possibility could be that there is enhanced reporting of cardiogenic shock over the years, particularly following the initiation of operator-reported outcomes.

Causes

ST elevation myocardial infarction with subsequent left ventricular dysfunction is the commonest cause of cardiogenic shock complicating acute coronary syndromes. The median time after ST elevation myocardial infarction for the occurrence of shock is 5–6 hours (Hochman et al, 1999), whereas in patients presenting with non-ST elevation myocardial infarction, cardiogenic shock tends to develop after a longer time. The difference in underlying pathophysiology is likely to reflect this time difference, but autopsy studies have generally shown that a loss of more than 40% of functional myocardium is required to cause cardiogenic shock (Wackers et al, 1976). However, mechanical complications, such as ventricular septal defect, free wall rupture and papillary muscle rupture, historically thought to be late complications after acute coronary syndromes, most commonly present within 24 hours of hospitalization (Kutty et al, 2013) and also contribute to cardiogenic shock after acute coronary syndromes. An index of suspicion and rapid echocardiography are required for such diagnoses with studies demonstrating that cases of cardiogenic shock involve ventricular septal defects in 3.9%, ischaemic mitral regurgitation in 6.9% and importantly 1.4% for cardiac tamponade (Hochman et al, 2000).

Non-acute coronary syndrome-related cardiogenic shock is worth bearing in mind especially in patients with normal coronary arteries on angiography and may be caused by decompensated valvular heart disease, acute myocarditis and arrhythmias with heterogeneous treatment options revolving around the identification and treatment of the underlying cause. *Figure 1* demonstrates the vicious cycle that can occur in cardiogenic shock following acute coronary syndromes unless adequate and timely revascularization takes place.

Treatment

Revascularization

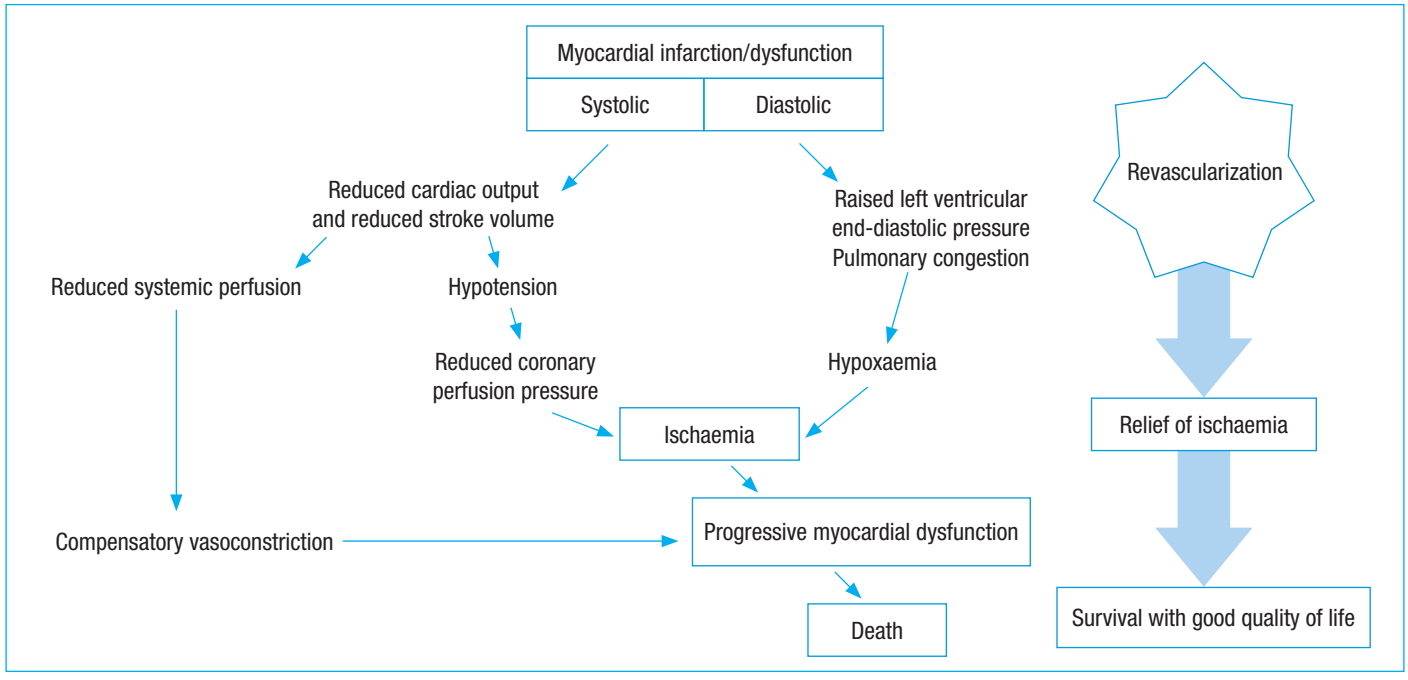
Early revascularization is the most important treatment strategy in cardiogenic shock complicating acute myocardial infarction and is the only strategy supported by trial data to reduce mortality in this patient group. These data were provided by the SHould we emergently revascularise Occluded Coronaries for cardiogenic shocK (SHOCK) trial (Hochman et al, 1999). Although the trial failed to meet the primary endpoint (superiority of early revascularization over medical therapy on 30-day mortality: 46.7% *vs* 56.0%, *P*=0.11) there was a significant mortality reduction at longer follow up of 6 months, 1 and 6 years. Despite the majority of patients being treated by percutaneous coronary intervention (64%) in this trial, 36% underwent early coronary artery bypass grafting, a fact often overlooked by many. The number needed to save one life by early revascularization compared to initial medical stabilization is <8.

Revascularization in patients with multi-vessel coronary artery disease

Approximately 50–80% of patients with cardiogenic shock present with multi-vessel coronary artery disease (Hochman et al, 1999) – these patients have a higher mortality than patients with single vessel disease (Hochman et al, 1999). Randomized trials have demonstrated the benefit of complete *vs* culprit-only revascularization

© 2019 MA Healthcare Ltd

Figure 2. Diagram demonstrating how an acute myocardial infarction can lead to left ventricular dysfunction and hypoperfusion, which can then lead to increased preload and afterload. Finally, this results in a vicious cycle of hypoperfusion and acidosis via compensatory neurohormonal mechanisms.



in patients without cardiogenic shock, but there is no consensus on the optimal management of significant 'non-culprit' coronary artery lesions in patients with cardiogenic shock, uncertainty reflected in current guidelines (O'Gara et al, 2013) and a meta-analysis of observational studies suggesting no benefit (de Waha et al, 2017).

A contemporary randomized controlled trial was needed. As a result CULPRIT-SHOCK, a multicentre, international trial, was undertaken, which randomized 706 patients with multi-vessel disease and acute myocardial infarction complicated by cardiogenic shock. This demonstrated that at 30 days, the composite primary end-point of death or renal replacement therapy had occurred in 158 of the 344 patients (45.9%) in the culprit-lesion-only percutaneous coronary intervention group and in 189 of the 341 patients (55.4%) in the multi-vessel percutaneous coronary intervention group (risk ratio 0.83; 95% confidence interval 0.71–0.96; *P*=0.01) (Thiele et al, 2017). The time to haemodynamic stabilization, the risk of catecholamine therapy and the duration of such therapy, the levels of troponin T and creatine kinase, and the rates of bleeding and stroke were similar between the groups, supporting the concept of culprit-only revascularization in cardiogenic shock. This well-conducted clinical trial sheds doubt on current guidelines disputing the benefit of multi-vessel intervention in patients with cardiogenic shock.

Fluids, inotropes and vasopressors

Cardiogenic shock treatment includes initial stabilization with volume expansion to obtain optimal filling pressures, vasopressors and inotropes, plus additional therapy for multi-organ system dysfunction, e.g. renal. Fluid administration should be based on pathophysiological considerations and has not been studied in adequate randomized controlled trials. Despite the frequent use of pharmacological support in cardiogenic shock, there is little clinical outcome data to guide the selection. The SOAP II trial (Sepsis Occurrence in Acutely Ill Patients) examined first-line vasopressor selection in patients with generalized shock and included a prespecified cardiogenic shock subgroup (De Backer et al, 2010). Dopamine was associated with a higher rate of arrhythmias overall and was associated with a higher mortality in patients with cardiogenic shock.

Current European Society of Cardiology guidelines (Steg et al, 2012) state that inotropic or vasopressor agents should be considered in patients with cardiogenic shock, with IIa C recommendations for dopamine and dobutamine and class IIb B recommendation for noradrenaline, which is slightly confusing given the results of the SOAP II trial discussed above. The most recent national guidelines focusing specifically on cardiogenic shock from Austria and Germany are more precise in stating that dopamine should no longer be used in the treatment of cardiogenic shock (Werdan et al, 2012). The authors therefore recommend that noradrenaline should first be titrated until the systolic arterial pressure rises to at least 80 mmHg, with intravenous dobutamine subsequently commenced, because of its β2-adrenergic effects, to try and improve cardiac contractility.

Levosimendan is a calcium sensitiser and ATP-dependent potassium-channel opener (Papp et al, 2012) developed for the treatment of acute decompensated heart failure, with solid evidence of efficacy and safety (Nieminen et al, 2016). Although levosimendan is one of the best studied inotropic agents in patients with acute heart failure, in view of its vasodilatory effects, with subsequent blood pressure lowering, the clinical evidence to date in patients

© 2019 MA Healthcare Ltd

with cardiogenic shock is limited. However, some clinical observations indicate that levosimendan can improve haemodynamics in the context of cardiogenic shock after acute coronary syndromes, when combined with catecholamines, to maintain adequate perfusion pressures. An expert review paper recommends its use in combination with noradrenaline in patients with cardiogenic shock related to acute coronary syndromes (Nieminen et al, 2016).

Haemodynamic monitoring

Haemodynamic data provided by pulmonary artery catheters can confirm the presence and severity of cardiogenic shock, involvement of the right ventricle, and particularly cardiac power and stroke work index provide powerful short-term prognostic value (Fincke et al, 2004). However, there has been a decline in pulmonary artery catheter use as shown by the change in criteria for including patients in cardiogenic shock trials, which relates to controversy regarding the benefit of their use, as shown in a meta-analysis (Shah et al, 2005). Although non-invasive devices may be used, their reliability in this setting has not been well studied and individualised pulmonary artery catheter use is still recommended (IIb B European Society of Cardiology recommendation) to monitor haemodynamic variables or treatment in patients with severe heart failure not responding to appropriate treatment (Steg et al, 2012).

Percutaneous mechanical support

Intra-aortic balloon pump

Intra-aortic balloon pumping is still commonly used to mechanically assist patients with cardiogenic shock where pharmacological therapy is not adequate (Sjauw et al, 2009). Made of a polyurethane membrane mounted on a vascular 7F–8F catheter, the intra-aortic balloon pump is positioned in the descending thoracic aorta just distal to the left subclavian artery. The device is timed to inflate and deflate in concert with the cardiac cycle, thereby increasing the diastolic blood pressure and reducing the systolic blood pressure. Intra-aortic balloon pumping can augment blood flow in the coronary arteries, unload the left ventricle and therefore enhance myocardial oxygen supply by improving the peak diastolic pressure and lowering the end-systolic pressure by means of diastolic inflation and rapid systolic deflation (Kern et al, 1993).

Although a number of factors are physiologically beneficial with the use of intra-aortic balloon pumping, evidence from several meta-analyses, registries and randomized controlled trials (IABP-SHOCK II) raise significant doubts regarding its effectiveness as an adjunct to the management of cardiogenic shock, even suggesting no benefit of its use in clinical practice (Thiele et al, 2013; Ahmad et al, 2015). Cochrane meta-analyses have found intra-aortic balloon pumping use associated with significantly higher rates of major bleeding and other arterial complications, which highlights its potential detrimental effects (Unverzagt et al, 2015).

In addition, another mechanism for the ineffectiveness of intra-aortic balloon pumps in patients with cardiogenic shock may be related to the lack of haemodynamic efficacy with counterpulsation pumps when native left ventricular function is severely impaired. During cardiogenic shock, irreversible damage can occur as a result of diminished organ perfusion and the systemic inflammatory response, so providing mechanical haemodynamic support may not be enough to reverse the damage that has already occurred (Ouweneel et al, 2017). The American College of Cardiology/American Heart Association and European Society of Cardiology guidelines have downgraded the level of recommendation for the use of intra-aortic balloon pump therapy (Steg et al, 2012; O'Gara et al, 2013), and neither recommends the routine use of intra-aortic balloon pumping in patients with cardiogenic shock (Windecker et al, 2014). Correspondingly clinical rates of intra-aortic balloon pumping are low (15–30%) (Cohen et al, 2003) and falling (shown by a study from a large registry of 76 474 patients; Sandhu et al, 2015), highlighting the need for other methods of mechanical support to manage patients with cardiogenic shock or more targeted approaches to focus on those most likely to benefit.

Percutaneous left ventricular assist devices

Percutaneous ventricular assist devices, e.g. Impella or HeartMate PHP, and other newer therapies such as extracorporeal membrane oxygenation have been developed but data to date are limited. The Impella 2.5 (Abiomed, Danvers, MA) has not been found to be effective on outcomes in both a European registry (Impella-EURO-SHOCK) (Lauten et al, 2013) and a small randomized controlled trial (25 patients) where Impella was compared with intra-aortic balloon pumping, despite superior haemodynamic measurements in Impella patients (Seyfarth et al, 2008). Recently, this device has been Food and Drug Administration approved for use in patients with cardiogenic shock and one randomized controlled trial (IMPRESS in Severe Shock trial) has shown no significant benefit in the reduction of 30-day mortality in patients with cardiogenic shock compared to intra-aortic balloon pumping (Ouweneel et al, 2017). Ongoing randomized controlled trials are evaluating Impella CP in the setting of cardiogenic shock, e.g. the Danish Cardiogenic Shock Trial NCT01633502, which will help answer the question if completed.

The HeartMate PHP is a different newer catheter-based percutaneous blood pump with collapsible axial flow impeller and cannula at the distal end which sits across the aortic valve rather than in the left ventricle. The SHIELD I trial, a prospective, non-randomized, multicentre, open-label trial, evaluated its use in 50 patients undergoing high-risk percutaneous coronary intervention, with encouraging results – haemodynamic stability was achieved in all patients with a low incidence of adverse events (Dudek et al, 2018).

An alternative mechanical support device is the TandemHeart percutaneous ventricular assist device. The TandemHeart is a left atrial-to-femoral artery bypass

© 2019 MA Healthcare Ltd

system that can pump and deliver flow rates up to 4.0 litres/min. In Burkhoff et al's (2006) study 42 patients with cardiogenic shock were randomized to treatment with intra-aortic balloon pumping or TandemHeart percutaneous ventricular assist device. TandemHeart significantly improved haemodynamic parameters, even in patients failing on intra-aortic balloon pumping. A similar sized study by Thiele et al (2005) found much the same, with improvement in cardiac output and other haemodynamic indices with the percutaneous ventricular assist device compared to an intra-aortic balloon pump.

Finally, the iVAC2L is a transfemoral pulsatile percutaneous ventricular assist device. The percutaneous femoral approach means that iVAC2L can support, with a pulsatile flow, patients with impaired left ventricular function who require a mechanical circulatory support as bridge to decision or bridge to bridge (left ventricular assist device). Use of the iVAC2L is effective in high risk percutaneous coronary intervention procedures (Samol et al, 2017).

Current European Society of Cardiology guidelines recommend considering the short-term use of percutaneous mechanical circulatory support devices in refractory cardiogenic shock in order to stabilize patients and preserve organ perfusion (oxygenation) as a bridge to recovery of myocardial function, cardiac transplantation or even left ventricular assist device therapy (Ibanez et al, 2018). This recommendation is based on expert consensus as the scientific evidence is lacking, although there is most data for the Impella device. As well as the type of mechanical support, the timing of its initiation could also have an effect on clinical outcome of cardiogenic shock. Mechanical support is often started relatively late after admission to the intensive cardiology care unit when it becomes clear that revascularization and the escalating use of inotropes and vasopressors have failed to improve the haemodynamic condition of the patient. One hypothesis is that shortening the 'door to unloading' and 'door to support' time may allow more effective reperfusion therapy in patients with ST elevation myocardial infarction presenting in cardiogenic shock (Kapur and Davila, 2017).

Peripheral veno-arterial extracorporeal membrane oxygenation

In recent years, peripheral veno-arterial extracorporeal membrane oxygenation has been increasingly used as a method of temporary mechanical cardiac support for patients with cardiogenic shock complicating ST elevation myocardial infarction. Extracorporeal membrane oxygenation is a form of cardiopulmonary life support, where blood is drained from the vascular system, circulated outside the body by a mechanical pump and then reinfused (veno-arterial or venous-venous). While outside the body, haemoglobin becomes fully saturated with oxygen and carbon dioxide is removed. Oxygenation is determined by flow rate, and carbon dioxide elimination can be controlled by adjusting the rate of countercurrent gas flow through the oxygenator (Schmidt et al, 2013).

Veno-arterial extracorporeal membrane oxygenation provides both respiratory and haemodynamic support and is therefore used in patients with cardiogenic shock. During veno-arterial extracorporeal membrane oxygenation, blood will bypass both the heart and the lungs. Blood is extracted from the right atrium or vena cava (for drainage), and returned to the arterial system through peripheral cannulations via femoral, axillary or carotid arteries (for infusion) bypassing both the heart and the lungs. Extracorporeal membrane oxygenation can be rapidly set up if the expertise and infrastructure is available locally and could provide a cost-effective treatment for cardiogenic shock. However, there are no randomized controlled trials comparing extracorporeal membrane oxygenation to intra-aortic balloon pumping or other support therapies and a meta-analysis of 20 studies of over 1800 patients with cardiogenic shock or cardiac arrest showed high complication rates with extracorporeal membrane oxygenation, with high rates of lower extremity ischaemia and major or significant bleeding (Cheng et al, 2014), suggesting more study is needed. However, European Society of Cardiology guidelines currently recommend considering the short-term use of veno-arterial extracorporeal membrane oxygenation in refractory cardiogenic shock (Ibanez et al, 2018) based on expert consensus.

Regional shock networks

Regional systems of care that allow immediate transfer of patients with cardiogenic shock to advanced 'shock' centres (van Diepen et al, 2017) need to be developed globally. By lowering the door to support time in these regional networks, patients with cardiogenic shock could be treated before they start developing irreversible haemodynamic as well as metabolic shock. The French-based RESCUE study, which transferred patients with cardiogenic shock to a central hub centre using a mobile extracorporeal membrane oxygenation team, successfully demonstrated the feasibility of a regional hub and spoke cardiogenic shock network (Beurtheret et al, 2013). This study had a network of 22 tertiary and 53 non-tertiary centres that transferred cardiogenic shock patients to three designated centres using a mobile extracorporeal membrane oxygenation team. Of 75 patients that were stabilized, there were no adverse events during transfer, 32 patients were alive at discharge and 30 patients were still alive at 1-year follow up. This demonstrated the feasibility of mobile teams facilitating early support and management of patients with cardiogenic shock in specialized centres. *Figure 2* shows the location of veno-arterial extracorporeal membrane oxygenation centres in the UK.

Expert opinion

Cardiogenic shock remains the commonest cause of in-hospital death in patients presenting with acute coronary syndromes, and only a few treatment strategies, i.e. early revascularization, are based on randomized trial evidence.

© 2019 MA Healthcare Ltd

The application of best practice with patients treated according to guidelines (early reperfusion for all patients and optimal intensive care treatment) can reduce the mortality of cardiogenic shock to 40% but more research is needed. Too many questions remain unanswered:

- What is the best definition of cardiogenic shock?
- Is the current clinical definition too broad to identify those who really benefit?
- Should pulmonary artery catheters be routinely used to guide therapies?
- Which vasopressor agent should be used and when?
- Which mechanical support device should be used and when?

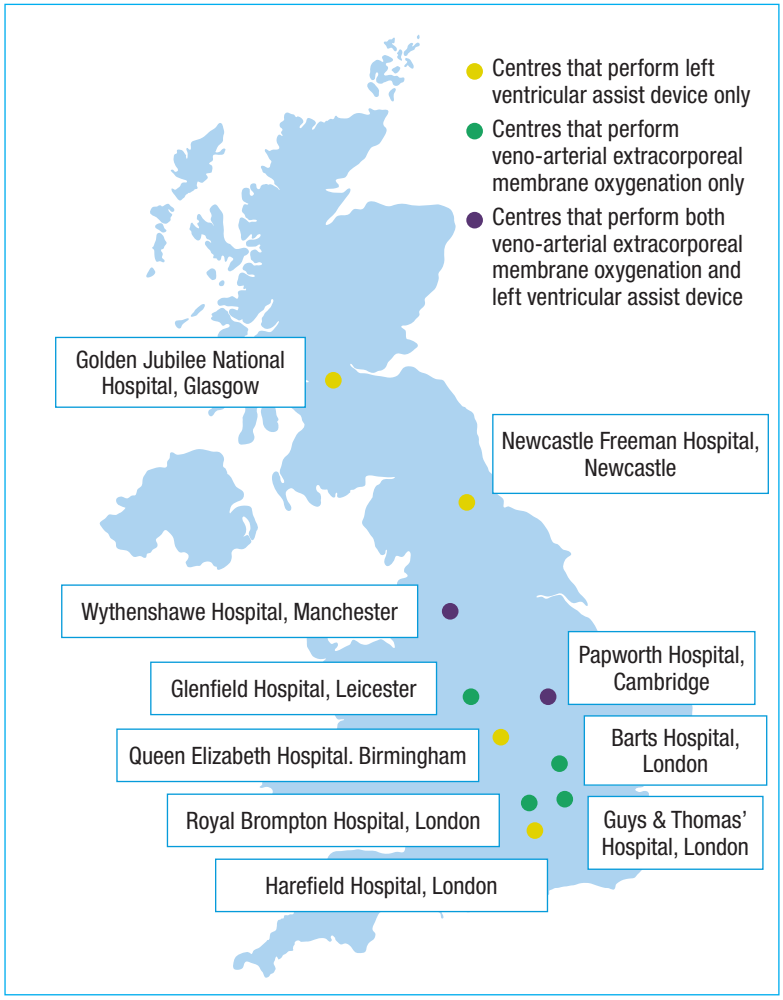
Going forward these questions need answers and future studies in cardiogenic shock, so difficult to perform, are needed. Crucially outcomes may improve with coordinated regionalised approaches to cardiogenic shock management with multidisciplinary care provided in designated tertiary shock centres, that have the expertise, clinical volume and resources necessary to centralize the delivery of the medical, surgical and mechanical therapies to optimize outcomes. **BJHM**

Figure 1 *is adapted from http://www.emdocs.net/cardiogenic-shock/*
Conflict of interest: none.

KEY POINTS

- Cardiogenic shock complicates the management of 7–10% of patients with acute coronary syndromes, and carries a high mortality rate (40–50%).
- ST elevation myocardial infarction with subsequent left ventricular dysfunction is the commonest cause of cardiogenic shock complicating acute coronary syndromes.
- Early revascularization is the most important and currently only evidence-based treatment strategy in cardiogenic shock complicating acute myocardial infarction.
- Further data for promising new therapies such as extracorporeal membrane oxygenation and Impella are needed to support their wider use.
- Regional systems of care that allow immediate transfer of patients with cardiogenic shock to advanced 'shock' centres need to be developed globally.

Figure 2. Map of veno-arterial extracorporeal membrane oxygenation and left ventricular assist device adult centres in the UK.



Ahmad Y, Sen S, Shun-Shin MJ et al. Intra-aortic balloon pump therapy for acute myocardial infarction. JAMA Intern Med. 2015 Jun 01;175(6):931–939. https://doi.org/10.1001/jamainternmed.2015.0569

Beurtheret S, Mordant P, Paoletti X et al. Emergency circulatory support in refractory cardiogenic shock patients in remote institutions: a pilot study (the cardiac-RESCUE program). Eur Heart J. 2013 Jan 02;34(2):112–120. https://doi.org/10.1093/eurheartj/ehs081

Burkhoff D, Cohen H, Brunckhorst C, O'Neill WW; TandemHeart Investigators Group. A randomized multicenter clinical study to evaluate the safety and efficacy of the TandemHeart percutaneous ventricular assist device versus conventional therapy with intraaortic balloon pumping for treatment of cardiogenic shock. Am Heart J. 2006 Sep;152(3):469.e1–469.e8. https://doi.org/10.1016/j.ahj.2006.05.031

Cheng R, Hachamovitch R, Kittleson M et al. Complications of extracorporeal membrane oxygenation for treatment of cardiogenic shock and cardiac arrest: a meta-analysis of 1,866 adult patients. Ann Thorac Surg. 2014 Feb;97(2):610–616. https://doi.org/10.1016/j.athoracsur.2013.09.008

Cohen M, Urban P, Christenson JT et al; Benchmark Registry Collaborators. Intra-aortic balloon counterpulsation in US and non-US centres: results of the Benchmark® Registry. Eur Heart J. 2003 Oct;24(19):1763–1770. https://doi.org/10.1016/j.ehj.2003.07.002

De Backer D, Biston P, Devriendt J et al; SOAP II Investigators. Comparison of dopamine and norepinephrine in the treatment of shock. N Engl J Med. 2010 Mar 04;362(9):779–789. https://doi.org/10.1056/NEJMoa0907118

de Waha S, Jobs A, Eitel I et al. Multivessel versus culprit lesion only percutaneous coronary intervention in cardiogenic shock complicating acute myocardial infarction: A systematic review and meta-analysis. Eur Heart J Acute Cardiovasc Care. 2018 Feb;7(1):28–37. https://doi.org/10.1177/2048872617719640

Dudek D, Ebner A, Sobczyński R et al. Efficacy and safety of the HeartMate percutaneous heart pump during high-risk percutaneous coronary intervention (from the SHIELD I Trial). Am J Cardiol. 2018 Jun;121(12):1524–1529. https://doi.org/10.1016/j.amjcard.2018.02.046

Fincke R, Hochman JS, Lowe AM et al; SHOCK Investigators. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: A report from the SHOCK trial registry. J Am Coll Cardiol. 2004 Jul;44(2):340–348. https://doi.org/10.1016/j.jacc.2004.03.060

Goldberg RJ, Spencer FA, Gore JM, Lessard D, Yarzebski J. Thirty-year trends (1975 to 2005) in the magnitude of, management of, and hospital death rates associated with cardiogenic shock in patients with acute myocardial infarction: a population-based perspective. Circulation. 2009 Mar 10;119(9):1211–1219. https://doi.org/10.1161/CIRCULATIONAHA.108.814947

Hochman JS, Sleeper LA, Webb JG et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock.

© 2019 MA Healthcare Ltd

SHOCK Investigators. Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock. N Engl J Med. 1999 Aug 26;341(9):625–634. https://doi.org/10.1056/NEJM199908263410901

Hochman JS, Buller CE, Sleeper LA et al. Cardiogenic shock complicating acute myocardial infarction—etiologies, management and outcome: a report from the SHOCK Trial Registry. J Am Coll Cardiol. 2000 Sep;36(3) Suppl A:1063–1070. https://doi.org/10.1016/S0735-1097(00)00879-2

Ibanez B, James S, Agewall S et al; ESC Scientific Document Group. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2018 Jan 07;39(2):119–177. https://doi.org/10.1093/eurheartj/ehx393

Kapur NK, Davila CD. Timing, timing, timing: the emerging concept of the 'door to support' time for cardiogenic shock. Eur Heart J. 2017 Dec 14;38(47):3532–3534. https://doi.org/10.1093/eurheartj/ehx406

Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet. 2003 Jan;361(9351):13–20. https://doi.org/10.1016/S0140-6736(03)12113-7

Kern MJ, Aguirre F, Bach R, Donohue T, Siegel R, Segal J. Augmentation of coronary blood flow by intra-aortic balloon pumping in patients after coronary angioplasty. Circulation. 1993 Feb;87(2):500–511. https://doi.org/10.1161/01.CIR.87.2.500

Kolte D, Khera S, Aronow WS et al. Trends in incidence, management, and outcomes of cardiogenic shock complicating ST-elevation myocardial infarction in the United States. J Am Heart Assoc. 2014 Jan 27;3(1):e000590. https://doi.org/10.1161/JAHA.113.000590

Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. Cardiol Clin. 2013 Nov;31(4):519–531, vii–viii. https://doi.org/10.1016/j.ccl.2013.07.004

Lauten A, Engström AE, Jung C et al. Percutaneous left-ventricular support with the Impella-2.5-assist device in acute cardiogenic shock: results of the Impella-EUROSHOCK-registry. Circ Heart Fail. 2013 Jan;6(1):23–30. https://doi.org/10.1161/CIRCHEARTFAILURE.112.967224

Nieminen MS, Buerke M, Cohen-Solál A et al. The role of levosimendan in acute heart failure complicating acute coronary syndrome: A review and expert consensus opinion. Int J Cardiol. 2016 Sep;218:150–157. https://doi.org/10.1016/j.ijcard.2016.05.009

O'Gara PT, Kushner FG, Ascheim DD et al; American College of Emergency Physicians; Society for Cardiovascular Angiography and Interventions. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. J Am Coll Cardiol. 2013 Jan;61(4):e78–e140. https://doi.org/10.1016/j.jacc.2012.11.019

Obling L, Frydland M, Hansen R et al. Risk factors of late cardiogenic shock and mortality in ST-segment elevation myocardial infarction patients. Eur Heart J Acute Cardiovasc Care. 2018 Feb;7(1):7–15. https://doi.org/10.1177/2048872617706503

Ouweneel DM, Eriksen E, Sjauw KD et al. Percutaneous mechanical circulatory support versus intra-aortic balloon pump in cardiogenic shock after acute myocardial infarction. J Am Coll Cardiol. 2017 Jan;69(3):278–287. https://doi.org/10.1016/j.jacc.2016.10.022

Papp Z, Édes I, Fruhwald S et al. Levosimendan: molecular mechanisms and clinical implications. Int J Cardiol. 2012 Aug;159(2):82–87. https://doi.org/10.1016/j.ijcard.2011.07.022

Rathod KS, Koganti S, Iqbal MB et al. Contemporary trends in cardiogenic shock: Incidence, intra-aortic balloon pump utilisation and outcomes from the London Heart Attack Group. Eur Heart J Acute Cardiovasc Care. 2018 Feb;7(1):16–27. https://doi.org/10.1177/2048872617741735

Samol A, Schmidt S, Zeyse M, Dürrwald S, Wiemer M. First high-risk percutaneous coronary intervention under use of the iVAC2l system in Germany. Future Cardiol. 2017 Mar;13(2):117–123. https://doi.org/10.2217/fca-2016-0064

Sandhu A, McCoy LA, Negi SI et al. Use of mechanical circulatory support in patients undergoing percutaneous coronary intervention: insights from the National Cardiovascular Data Registry. Circulation. 2015 Sep 29;132(13):1243–1251. https://doi.org/10.1161/CIRCULATIONAHA.114.014451

Schmidt M, Tachon G, Devilliers C et al. Blood oxygenation and decarboxylation determinants during venovenous ECMO for respiratory failure in adults. Intensive Care Med. 2013 May;39(5):838–846. https://doi.org/10.1007/s00134-012-2785-8

Seyfarth M, Sibbing D, Bauer I et al. A randomized clinical trial to evaluate the safety and efficacy of a percutaneous left ventricular assist device versus intra-aortic balloon pumping for treatment of cardiogenic shock caused by myocardial infarction. J Am Coll Cardiol. 2008 Nov;52(19):1584–1588. https://doi.org/10.1016/j.jacc.2008.05.065

Shah MR, Hasselblad V, Stevenson LW, Binanay C, O'Connor CM, Sopko G, Califf RM. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. JAMA. 2005 Oct 05;294(13):1664–1670. https://doi.org/10.1001/jama.294.13.1664

Sjauw KD, Engström AE, Vis MM et al. A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines? Eur Heart J. 2009 Feb;30(4):459–468. https://doi.org/10.1093/eurheartj/ehn602

Steg PG, James SK, Atar D et al; Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2012 Oct 01;33(20):2569–2619. https://doi.org/10.1093/eurheartj/ehs215

Thiele H, Sick P, Boudriot E, Diederich KW, Hambrecht R, Niebauer J, Schuler G. Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock. Eur Heart J. 2005 Jul 01;26(13):1276–1283. https://doi.org/10.1093/eurheartj/ehi161

Thiele H, Zeymer U, Neumann FJ et al; Intraaortic Balloon Pump in cardiogenic shock II (IABP-SHOCK II) trial investigators. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. Lancet. 2013 Nov;382(9905):1638–1645. https://doi.org/10.1016/S0140-6736(13)61783-3

Thiele H, Akin I, Sandri M et al; CULPRIT-SHOCK Investigators. PCI strategies in patients with acute myocardial infarction and cardiogenic shock. N Engl J Med. 2017 Dec 21;377(25):2419–2432. https://doi.org/10.1056/NEJMoa1710261

Unverzagt S, Buerke M, de Waha A et al. Intra-aortic balloon pump counterpulsation (IABP) for myocardial infarction complicated by cardiogenic shock. Cochrane Database Syst Rev. 2015 Mar 27;(3):CD007398. https://doi.org/10.1002/14651858.CD007398.pub3

van Diepen S, Katz JN, Albert NM et al; American Heart Association Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Quality of Care and Outcomes Research; and Mission: Lifeline. Contemporary Management of Cardiogenic Shock: A Scientific Statement From the American Heart Association. Circulation. 2017 Oct 17;136(16):e232–e268. https://doi.org/10.1161/CIR.0000000000000525

Wackers FJ, Lie KI, Becker AE, Durrer D, Wellens HJ. Coronary artery disease in patients dying from cardiogenic shock or congestive heart failure in the setting of acute myocardial infarction. Heart. 1976 Sep 01;38(9):906–910. https://doi.org/10.1136/hrt.38.9.906

Werdan K, Ruß M, Buerke M, Delle-Karth G, Geppert A, Schöndube FA; German Cardiac Society; German Society of Intensive Care and Emergency Medicine; German Society for Thoracic and Cardiovascular Surgery; (Austrian Society of Internal and General Intensive Care Medicine; German Interdisciplinary Association of Intensive Care and Emergency Medicine; Austrian Society of Cardiology; German Society of Anaesthesiology and Intensive Care Medicine; German Society of Preventive Medicine and Rehabilitation. Cardiogenic shock due to myocardial infarction: diagnosis, monitoring and treatment: a German-Austrian S3 Guideline. Dtsch Arztebl Int. 2012 May;109(19):343–351. https://doi.org/10.3238/arztebl.2012.0343

Windecker S, Kolh P, Alfonso F et al; Authors/Task Force members. 2014 ESC/EACTS Guidelines on myocardial revascularization. Eur Heart J. 2014 Oct 01;35(37):2541–2619. https://doi.org/10.1093/eurheartj/ehu278

© 2019 MA Healthcare Ltd