



CARDIOGENIC SHOCK GUIDELINES

These guidelines are intended for use by medical staff working in Critical Care areas (**Coronary Care Unit, General Critical Care Units, and Cardiothoracic Critical Care Unit**) within NHS Lothian. They are intended for guidance only and do not replace the need for early, senior, specialist input.

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1. Recognition of Cardiogenic Shock	<p>Cardiogenic shock (CGS) should be considered in patients with evidence of cardiovascular compromise AND a suspected or confirmed Cardiac aetiology.</p> <table><tr><th>Cardiovascular Compromise <i>As evidenced by one of the following:</i></th><th>AND</th><th>Cardiac Aetiology <i>As evidenced by one of the following:</i></th></tr><tr><td>Hypotension (SBP \leq90mmHg) or requiring vasopressors or inotropes to maintain SBP $>$90mmHg</td><td></td><td>Known pathology associated with low cardiac output state, e.g.<ul style="list-style-type: none">Acute Myocardial IschaemiaCardiomyopathyPost cardiectomy or cardiopulmonary bypassSevere valve pathologyCardiotoxic drug overdose</td></tr><tr><td>OR</td><td></td><td>OR</td></tr><tr><td>Clinical evidence of end organ hypoperfusion, e.g.<ul style="list-style-type: none">Cold/clammy skin and peripheries,Altered mental status,Oliguria</td><td></td><td>Echocardiogram demonstrating low cardiac output state (Ejection Fraction $<$30% or LVOT VTI $<$10cm) (with any underlying aetiology)</td></tr><tr><td>OR</td><td></td><td>OR</td></tr><tr><td>Laboratory evidence of end organ hypoperfusion,<ul style="list-style-type: none">Lactate $>$2.0mmol/L,Elevated creatinine,Deranged liver function</td><td></td><td>Cardiac output monitoring demonstrating low cardiac output state (Cardiac Index \leq 2.2L/min/m²) (with any underlying aetiology)</td></tr></table> <p>Patients with cardiogenic shock can be normotensive or even hypertensive</p> <p>Consider all markers of organ perfusion (lactate, renal function, cerebation etc).</p> <p>Consideration should be given to a cardiogenic element to any shock state if:</p> <ul style="list-style-type: none">Minimal response to fluid resuscitation or standard therapyOn high-dose vasopressors with minimal responsePersistently high lactate	Cardiovascular Compromise <i>As evidenced by one of the following:</i>	AND	Cardiac Aetiology <i>As evidenced by one of the following:</i>	Hypotension (SBP \leq 90mmHg) or requiring vasopressors or inotropes to maintain SBP $>$ 90mmHg		Known pathology associated with low cardiac output state, e.g. <ul style="list-style-type: none">Acute Myocardial IschaemiaCardiomyopathyPost cardiectomy or cardiopulmonary bypassSevere valve pathologyCardiotoxic drug overdose	OR		OR	Clinical evidence of end organ hypoperfusion , e.g. <ul style="list-style-type: none">Cold/clammy skin and peripheries,Altered mental status,Oliguria		Echocardiogram demonstrating low cardiac output state (Ejection Fraction $<$ 30% or LVOT VTI $<$ 10cm) (with any underlying aetiology)	OR		OR	Laboratory evidence of end organ hypoperfusion , <ul style="list-style-type: none">Lactate $>$2.0mmol/L,Elevated creatinine,Deranged liver function		Cardiac output monitoring demonstrating low cardiac output state (Cardiac Index \leq 2.2L/min/m ²) (with any underlying aetiology)
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2. Location of care	<p>In NHS Lothian, patients identified as having CGS with evidence of hypoperfusion should, if escalation is appropriate, be managed in a critical care setting. They should be discussed with NHS Lothian cardiology team to consider whether transfer to the Royal Infirmary of Edinburgh (RIE) is required. Consideration should also be given to early discussion with Golden Jubilee National Hospital in select cases (see appendix 3).</p> <p>Within RIE patients should be managed in a critical care area: 111, 114 or 118. Within SJH and WGH, should be managed acutely in HDU/ITU and discussed with cardiology team.</p> <p>See appendix 2 for details on the capabilities of RIE critical care areas.</p>																		
3. Initial investigation	<ul style="list-style-type: none">12 lead ECGFBC, U&Es, LFTs, Coagulation, TFT, troponin, glucose, lactate, Ca/Mg/PO4, D-dimer																		

	<ul style="list-style-type: none"> • Arterial Blood Gas • Chest X-ray • Echocardiogram <ul style="list-style-type: none"> ○ May be a focused echocardiogram in the first instance but any abnormal findings, or uncertainty regarding diagnosis, should prompt urgent comprehensive transthoracic echo by a Cardiologist, Cardiac Physiologist, Cardiac Anaesthetist or BSE accredited critical care physician OR trans-oesophageal echocardiography.
4. Monitoring	<ul style="list-style-type: none"> • All patients should have <ul style="list-style-type: none"> ○ ECG, SpO₂, respiratory rate, neurological status (AVPU/GCS), urine output ○ Consideration should be given to placement of an arterial line. This allows accurate, continuous measurement of blood pressure and frequent sampling of arterial blood. ○ Consideration should be given to placement of a central venous catheter. This allows safe and secure administration of inotropes or vasopressors, measurement of central venous pressure and of central venous oxygen saturation. • Regular measurement of plasma lactate should be undertaken until clear improvement. • 12-hourly U&Es and LFTs until normal • Cardiac Output Monitoring (e.g. Pulmonary Artery Catheter) should be considered in patients not responding to initial therapy or in whom there is diagnostic uncertainty.
5. General Management	<ul style="list-style-type: none"> • Gas exchange <ul style="list-style-type: none"> ○ Maintain SpO₂ >93% ○ Aim PaCO₂ 4-5.5kPa ○ Consider mechanical ventilation if: <ul style="list-style-type: none"> ▪ High work of breathing ▪ Tiring ▪ FiO₂ >0.6 required to maintain adequate oxygenation • Electrolytes <ul style="list-style-type: none"> ○ K⁺ 4.5-5.0 ○ Mg⁺⁺ 1 -1.30 ○ Ionised Calcium (on blood gas analyser) 1.2-1.4
6. Initial Referrals	<ul style="list-style-type: none"> • Cardiology – all patients with cardiogenic shock should be referred to cardiology for consideration of advanced diagnostics and interventions (including angiography) • Cardiothoracic Surgery – refer if: <p>Suspected or echocardiography-confirmed mechanical ischaemic complication causing cardiogenic shock, e.g. Acute severe mitral regurgitation; Ventricular Septal Defect; Free wall rupture</p> <ul style="list-style-type: none"> ○ Decompensated valvular pathologies ○ Cardiogenic shock following cardiothoracic surgery

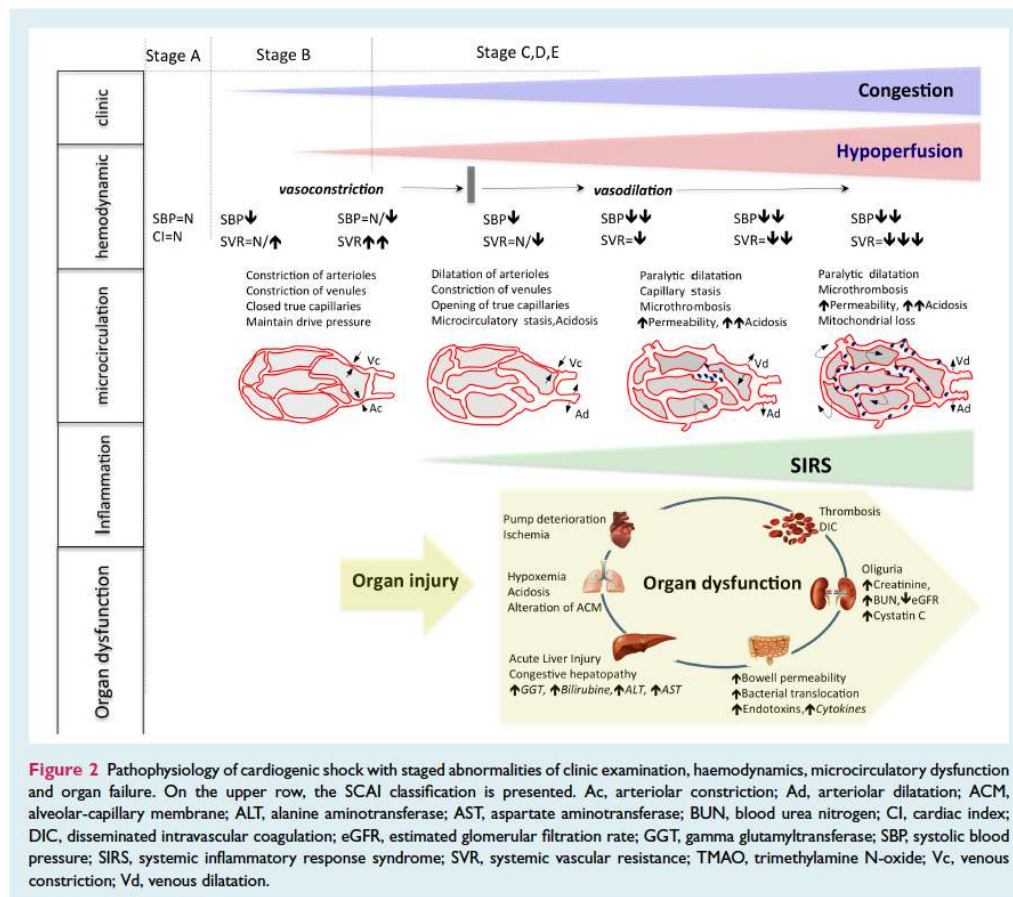
	<ul style="list-style-type: none"> ○ Patient requires revascularisation and has known multivessel or complex coronary artery disease which may benefit from CABG. ● Critical Care – If in need of multiple vasoactive agents, multiple organ support or advanced ventilation. Or failing to improve on initial therapy.
7. Pharmacological support	<p>Conduct a thorough assessment of cardiovascular physiological status to determine the most appropriate pharmacological support.</p> <p>Use a combination of clinical examination, echocardiography +/- cardiac output monitoring to determine the degree of cardiac impairment and the contributions of inappropriate vasodilation/hypovolaemia.</p> <p>Repeatedly assess and record haemodynamics and markers of end organ perfusion to titrate pharmacological support appropriately.</p> <p>Targets:-</p> <p>Physiological targets may require adjustment for individuals. As a starting point, target:</p> <ol style="list-style-type: none"> 1. Blood pressure <ol style="list-style-type: none"> a. SBP >90 or b. MAP 60-65mmHg 2. Normalisation of end organ perfusion <ol style="list-style-type: none"> a. Lactate <2 b. Normal cerebral function c. Urine output >0.5ml/kg/hr d. Improvement in liver function e. Venous oxygen saturation <p>Hypovolaemia –</p> <ol style="list-style-type: none"> 1. A cautious fluid bolus ($\leq 250\text{ml}$) may be considered if clinical or echocardiographic suggestion of hypovolaemia. 2. Repeat fluid boluses should only be undertaken with repeated clinical and echocardiographic assessment +/- cardiac output monitoring. <p>Decreased cardiac output</p> <p>End organ perfusion is a more important end point than cardiac output per se. Pharmacological support of cardiac output should be titrated primarily against end organ function.</p> <p>All pharmacological support comes with potential adverse effects. No inotropic drug has been clearly shown to have an outcome benefit. The support utilised should be the minimum required to restore physiological normality and adequate end organ perfusion</p> <ol style="list-style-type: none"> 1. Inotropes (see appendix for dosing table) <ol style="list-style-type: none"> a. Dobutamine – A titratable synthetic catecholamine and reasonable first line option. May cause a decrease in systemic vascular resistance therefore hypotension; concurrent use of vasopressors may be necessary.

	<p>b. Milrinone – A selective phosphodiesterase inhibitor with pulmonary vasodilator properties hence useful in right heart failure +/- pulmonary hypertension. Likely to cause systemic vasodilation and often requires concurrent use of vasoconstrictors. Seek expert advice if unfamiliar with use.</p> <p>c. Adrenaline – a naturally occurring catecholamine with positive inotropic and vasoconstrictor properties. Several observational studies have identified an association between adrenaline use and mortality in CGS; it's not clear whether this is a causative relationship.</p> <p>d. Levosimendan – a calcium sensitiser which can be considered if other inotropic agents have failed. May have a particular use in patients on long term beta blockade.</p> <p>Decreased systemic vascular resistance (SVR) The increase in circulating endogenous catecholamines driven by CGS commonly provokes an increase in SVR with an associated increase in afterload. However, warm peripheries, a wide pulse pressure, and low SVR on cardiac output monitoring suggests vasodilation to be a contributing factor. This vasodilation may be the consequence of a co-existing pathological process, or a consequence of the inflammatory response generated in the latter stages of cardiogenic shock. The myocardium is perfused in diastole, a low diastolic pressure may lead to hypoperfusion and worsening cardiac function.</p> <ol style="list-style-type: none"> 1. Vasopressors – <ol style="list-style-type: none"> a. Norepinephrine is the most commonly used vasopressor and is appropriate in most cases of decreased SVR. b. Vasopressin offers an alternative agent to norepinephrine. It does not increase pulmonary vascular resistance therefore may be advantageous in cases of right heart failure. It is less arrhythmogenic and may be advantageous if arrhythmias are a major issue.
8. Mechanical support	<p>Intra-aortic Balloon Pump Indicated in select patients such as CGS secondary to acute severe mitral regurgitation or ventricular septal defect. Decreasing use has been in tandem with evidence from large multicentre, prospective trials of no mortality benefit in patients with CGS of acute ischaemic origin with early revascularisation. IABP may be used post PCI or cardiopulmonary bypass at the discretion of the Cardiologist, Cardiac Surgeon or Cardiac Anaesthetist.</p> <p>Extracorporeal Membrane Oxygenation ECMO is resource intensive and carries inherent risk. It is therefore reserved for severe or refractory cases of CGS with a realistic prospect of cardiac recovery, surgical correction of a mechanical cardiac complication or transplant. Early discussion with the local critical care team or GJNH as appropriate is encouraged. ECMO may be utilised as a:</p> <ul style="list-style-type: none"> • Bridge to recovery – for pathologies expected to resolve in a short time frame

	<p>(e.g. myocardial stunning, cardiotoxic drug overdose)</p> <ul style="list-style-type: none"> • Bridge to transplant – to facilitate transfer to the GJNH, transplant assessment and pending organ. • Bridge to bridge – e.g. long term Left Ventricular Assist Device. • Bridge to decision – to gather additional information, neuro-prognosticate, determine suitability for transplant etc. <p>The optimal point to introduce ECMO is uncertain. It should ideally be initiated prior to the establishment of multiple organ failure.</p> <p>Patients should be discussed early with the advanced heart failure service at the GJNH.</p> <p>ECMO should be considered and discussed with the relevant RIE teams in the following circumstances:</p> <ol style="list-style-type: none"> 1. (“sliding on inotropes”) Worsening end organ function despite pharmacological support; INTERMACS 2) 2. (“crash and burn” Hypotension refractory to inotropes; INTERMACS 1) 3. Recurrent malignant arrhythmias (VF or VT storm) 4. FiO2 >0.8 required to maintain adequate oxygenation despite mechanical ventilation 5. Concern from the clinical team regarding trajectory. <p>For consideration of ECMO in non-cardiac-surgical patients, contact on call general intensive care team and ask to speak to a member of the ECMO team.</p> <p>For consideration of ECMO in cardiac surgical patients, contact the on call Cardiothoracic Surgeon.</p>
<p>9. Referral to Scottish National Advanced Heart Failure Team</p>	<p>Indications for referral:</p> <p>In the absence of contraindications for cardiac transplantation, indications include:</p> <ul style="list-style-type: none"> ○ Inability to wean IV inotropic treatment ○ Anticipatory need for percutaneous MCS in cardiogenic shock ○ Ventilatory support with use of positive airway pressure for intractable pulmonary oedema ○ Refractory ventricular arrhythmia ○ Downwards trajectory of clinical condition despite optimal medical management <p>See appendix 1 for details.</p> <p>Patients accepted for transfer will need appropriate stabilisation prior to transportation. This may include mechanical ventilation, intra-aortic balloon pump or ECMO.</p> <p><referral number></p> <p><referral form (details of info require)></p>

APPENDIX 1 – PATHOPHYSIOLOGY OF CARDIOGENIC SHOCK

Cardiogenic shock (CS) is a complex condition in which end-organ hypoperfusion results from a low cardiac output state. CS is frequently complicated by multiorgan dysfunction system that requires a multidisciplinary approach in a critical care setting.



Appendix 2 – Location of Care

The European Society of Cardiology recommends that “All patients with cardiogenic shock should be rapidly transferred to a tertiary care centre which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term MCS.”. In NHS Lothian therefore patients **cardiogenic** shock which requires the institution of inotropic or vasopressor should be transferred to the Royal Infirmary of Edinburgh or, in certain circumstances, directly to the Golden Jubilee National Hospital (see section on Scottish National Acute Heart Failure Service below). Within the Royal Infirmary of Edinburgh, patients with cardiogenic shock may be managed in General Critical Care (118/116), Coronary Care (114) or Cardiothoracic Critical Care (111/112). The capabilities of these areas are outlined in the table below. If there is uncertainty regarding the most appropriate area of care, please discuss with the on-call consultant cardiologist/intensivist/cardiac anaesthetist.

	<i>CARDIOTHORACIC CRITICAL CARE 111/112</i>	<i>CORONARY CARE 114</i>	<i>GENERAL CRITICAL CARE 116/118</i>
<i>SINGLE INOTROPE (DOBUTAMINE)</i>	<i>X</i>	<i>X</i>	<i>X</i>
<i>VASOPRESSORS (NORADRENALINE, VASOPRESSIN)</i>	<i>X</i>		<i>X</i>
<i>MULTIPLE INOTROPES AND VASOPRESSORS</i>	<i>X</i>		<i>X</i>
<i>PULMONARY ARTERY CATHETERISATION</i>	<i>X</i>		<i>X</i>
<i>INTRA-AORTIC BALLOON PUMP</i>	<i>X</i>	<i>X</i>	<i>X</i>
<i>EXTRACORPOREAL MEMBRANE OXYGENATION</i>	<i>X</i>		<i>X</i>
<i>NON-INVASIVE RESPIRATORY SUPPORT (CPAP/HFNC)</i>	<i>X</i>	<i>X</i>	<i>X</i>
<i>INVASIVE MECHANICAL VENTILATION</i>	<i>X</i>		<i>X</i>
<i>NITRIC OXIDE CARE POST STERNOTOMY (INCLUDING PATIENTS WITH OPEN CHEST)</i>	<i>X</i> <i>X</i>		<i>X</i>

Appendix 3- Referral to the Scottish National Advanced Heart Failure Service (SNAHFS)

How to refer

Referrals should be made by senior medical staff after discussion with the on-call Consultant Cardiologist. Based at the Golden Jubilee National Hospital in Clydebank, there is a dedicated SNAHFS pager carried by the medical transplant fellow during daytime hours and by the GJNH cardiology fellow out of hours.

Referral forms are available at <https://hospital.nhsgoldenjubilee.co.uk/a-z-services/scottish-national-advanced-heart-failure-service/snahfs-referrals>

Indications for Urgent Inpatient Referral

In the absence of contraindications for cardiac transplantation, indications include:

- Inability to wean IV inotropic treatment
- Anticipatory need for temporary MCS in cardiogenic shock
- Ventilatory support with use of positive airway pressure for intractable pulmonary oedema
- Refractory ventricular arrhythmia
- Downwards trajectory of clinical condition despite optimal medical management

Contraindications to Cardiac Transplantation

- Active infection (patients with chronic viral infection such as hepatitis B, hepatitis C and HIV may be considered if viral titres are undetectable on treatment/following treatment with no evidence of other organ damage).
- Symptomatic cerebral or peripheral vascular disease.
- Diabetes mellitus with end-organ damage, eg, nephropathy, neuropathy, proliferative retinopathy. Poorly controlled diabetes with glycosylated haemoglobin persistently >7.5% or 58 mmol/mol is a relative contraindication.
- Current or recent neoplasm: risk of recurrence should be discussed with oncology.
- Severe lung disease: FEV1 and FVC <50% predicted or evidence of parenchymal lung disease.
- Irreversible renal dysfunction with eGFR <30 mL/min/1.73 m².
- Irreversible liver dysfunction, eg, cirrhosis.
- Recent pulmonary thromboembolism (generally in the last 3 months).
- Pulmonary hypertension with PA systolic pressure >60 mm Hg, transpulmonary gradient ≥15 mm Hg and/or pulmonary vascular resistance >5 Wood units. This is an absolute contraindication to isolated heart transplantation if irreversible with either pharmacological manipulation or mechanical unloading of the LV.
- Psychosocial factors including history of non-compliance with medication, inadequate support, ongoing/recent drug or alcohol abuse, current smoker.
- Obesity (body mass index >35 kg/m² or weight >140% of ideal body weight).

- Other multisystem disease with poor long-term survival.

Transfer

- Transfer of patients with CGS to the GJNH will be undertaken by the Scottish Ambulance Service with a nursing and medical team from the RIE.
- It is preferable to stabilise the patient prior to transfer. This may require mechanical ventilation, insertion of intra-aortic balloon pump, and/or commencement of ECMO.
- For patients transferred on IABP or ECMO, an advanced support transfer trolley is located in the 118 transfer room. This has an in built IABP and mounting bracket for ECMO consoles.
- Patients transferred on ECMO require appropriately trained medical and nursing staff plus a clinical perfusionist.

Appendix 4 - Echocardiography in Cardiogenic Shock

Echocardiography is essential in the early stages of assessment in suspected cardiogenic shock. Whilst training to a British Society of Echocardiography level 1 or FICE level scan may be adequate as a screening tool, if any abnormality is detected, or there remains any uncertainty about cardiac function, an echo by an accredited echocardiographer should be organised urgently.

The findings should be summarised in the clinical notes.

The European Society of Intensive Care Medicine (ESICM) recommends that the following parameters are recorded at the time of scanning, and that findings are interpreted in the context of these:

1. Mode of ventilation, tidal volume, plateau pressure, PEEP
2. Cardiac rhythm and rate
3. Blood pressure and support (vasoactive medications and doses)

1) Left Ventricle

- Apical, parasternal or subcostal views.
- Contractility – **hyperdynamic / normal / reduced / severely reduced**
 - A hyperdynamic LV may support hypovolaemia / sepsis.
 - However, consider acute valvular regurgitation, ventricular septal defect or right heart pathology.
- Cavity size – **small / normal / dilated**
 - LV dilatation takes time. If severely dilated on presentation (LVEDD >6.5cm) consider an underlying chronic pathology, eg. dilated cardiomyopathy, chronic ischaemic or valvular heart disease.
 - Consider dynamic LV-outflow tract obstruction if a small LV cavity with hyperdynamic function, LVH and a systolic murmur

2) Right Ventricle

- Apical and subcostal views. Often difficult to assess.
- **Contractility**
 - 'TAPSE' - longitudinal movement of the tricuspid annulus in systole, normal is >15mm.
- Consider both radial and longitudinal function. In acute pressure overload, radial systolic function is impaired early. **Chamber size**
 - Normal basal diameter in the apical 4-chamber view is 25-41mm.
 - Unlike the LV, the RV dilates early in response to pressure overload.
- Thrombus may be visible in large pulmonary embolism – this should provoke urgent cardiothoracic referral if visualised.

3) Valve Function

- Quantification of the severity of valve dysfunction requires experience & care. However, the identification of pathology is feasible and may alter treatment significantly.
- **Aortic valve**
 - Best visualised in the parasternal long axis.

- Comment if heavily calcified with reduced leaflet movement, if suspected vegetations or if severely regurgitant (colour flow assessment easier in the apical views).
- **Mitral valve**
 - Regurgitation is very common and may be the result of LV dysfunction rather than the precipitant. If chronic is normally well tolerated.
 - If acute, regurgitation due to mechanical complications of an MI or endocarditis is poorly tolerated.
 - A complex structure. The mitral valve apparatus may easily be mistaken for vegetation

Interpretation of Echocardiography in Critically Ill Patients

Sanfilippo, F. *et al.* (2020) 'The PRICES statement: an ESICM expert consensus on methodology for conducting and reporting critical care echocardiography research studies', *Intensive care medicine*. doi: 10.1007/s00134-020-06262-5.

Appendix 5 – Pharmacological Support

There is no conclusive evidence to recommend one particular agent over another. The American Heart Association (AHA) consensus scientific statement on the management of cardiogenic shock also notes that insufficient evidence exists to guide the selection of pharmacologic therapies. Therefore, use of inotropic and vasopressor agents should be guided by available hemodynamic data and clinician judgment.

Inotropic and vasopressor drugs commonly used in cardiogenic shock

DRUG	PRIMARY MECHANISM	DOSING
Dobutamine	β_1 agonist	2.5–20 mcg/kg/min
Milrinone	<i>Phosphodiesterase 3</i> inhibitor	0.125–0.5 mcg/kg/min
Adrenaline	Mixed α, β agonist	0.01–1 mcg/kg/min
Noradrenaline	Mixed α, β agonist ($\alpha > \beta$)	0.01–1 mcg/kg/min
Vasopressin	V1 receptor in vascular smooth muscle	0.02–0.04 units/min

Appendix 6 - Post Cardiectomy Cardiogenic Shock

The incidence of cardiogenic shock in post-cardiotomy patients is approximately 2-5%¹ and associated with higher patient morbidity/mortality.^{1,2} The most common causes are myocardial stunning, myocardial ischaemia or cardiac tamponade. Other causes include: pre-existing status, arrhythmia, acute valvular failure, dynamic LVOT obstruction and mechanical complications of cardiac surgery. The presentation of post cardiac surgery CGS is often, 'warm and wet', due to the combination of cardiogenic shock with a post cardiopulmonary bypass inflammatory response.¹ Clinicians need to remain vigilant to co-existing pathologies of hypovolaemic and /or distributive shock, detailed management of these other forms of shock are out with the scope of the CGS guideline.

1) Identification of cardiogenic shock

As per prior text.

2) Location of care

Post cardiectomy patients in RIE are always cared for on the cardiothoracic critical care area (wards 111/112) due to the potential for re-opening and proximity to theatres.

3) Investigation, monitoring and referral

There is a very low threshold for early TTE/TOE examination in the peri-operative period. A large proportion of cardiac patients have vascular sheaths in situ suitable for floating a pulmonary artery catheter if there is diagnostic doubt or lack of response to inotropic/vasopressor therapy. Non-invasive cardiac output measurement devices are available.

4) General management

As prior text. Hypocalcaemia is common and should be treated promptly. Electrolytes such as potassium and magnesium should be actively corrected to help abate arrhythmias. Epicardial pacing can be optimised to the individual patient, including to enhance cardiac output. Atrial fibrillation is present in 30-50% of post cardiac surgical patients, some of whom will decompensate. Treatment of the decompensated AF follows conventional Advanced Life Support guidelines.

5) Pharmacological support

a) Inotropes and vasopressors

In U.K. cardiac critical care practice the first line inotrope and vasopressor are usually adrenaline and noradrenaline respectively with a few exceptions. In the context of RV failure with pulmonary hypertension, use of adrenaline and above moderate doses of noradrenaline is discouraged. Whilst it is up to the individual clinician, the combination of milrinone and vasopressin are advantageous in these circumstances. The other patient group to benefit from the positive inotropy and lusitropy of milrinone are those patients with diastolic dysfunction, using adrenaline in this patient group often leads to the unfavourable enhancement of diastolic dysfunction.

b) Inhaled nitric oxide

Inhaled nitric oxide has a role in reducing afterload on the failing right ventricle by vasodilation of the pulmonary arterial tree. It is particularly useful in managing the vortex of a failing right ventricle.

c) Methylene Blue

Rarely used but useful in extreme unresponsive cardiogenic shock less than 24 hours post cardiopulmonary bypass (1.5-2 mg/kg over 30-60 minutes).² It inhibits the production of intracellular nitric oxide leading to an increase in systemic vascular resistance. There are case series and small studies advocating its use, it should be noted there is a lack of high quality, large studies.

6) Temporary Mechanical Circulatory Support

As prior, increasing indications early institution of temporary MCS improves patients' outcome.^{1,3} IABP remain controversial, whilst there is strong statistical evidence of no mortality benefit at 30 days, 1 year nor 6 years in patients with ischaemic cardiogenic shock undergoing PCI, IABP remain part of the armamentarium to the cardiothoracic team for de-pairing the high risk patient from cardiopulmonary bypass or post-cardiotomy cardiogenic shock on the intensive care. There are only small studies to suggest the benefit of IABP in this context.

7) Special post cardiotomy circumstances

a) Cardiac tamponade

The collection of fluid, usually blood, in the pericardial space leads to compression of the cardiac chambers then low cardiac output. There are a range of presentations from acute dramatic loss of cardiac output to a more insidious onset. On considering cardiac tamponade, urgent echocardiography (TTE or TOE) should be performed. TOE is considered the superior mode of imaging in these circumstances due to anatomically where the collection usually is and the physical challenges of probe placement in post-op cardiothoracic patients.⁴ Definitive treatment is surgical drainage, either in theatre or critical care area depending on the degree of urgency. In extremis, as per Cardiac Advanced Life Support, an experienced clinician may choose to re-open the chest without imaging.⁴ The diagnosis of cardiac tamponade is a clinical one. If the patient is clinically behaving like a tamponade but lacking in echocardiography evidence, they should be treated as a cardiac tamponade until proven otherwise.

b) Myocardial ischaemia

A challenging area of practice in the post cardiotomy patient. Remember, 'time is muscle'. In the coronary bypass grafting patients, early graft failure is the most common cause.⁵ Ischaemia may be diagnosed by ECG changes (interpretation can be difficult), new regional wall motion abnormalities on echocardiography, serial troponin rise or angiography. On noting significant myocardial ischaemia, the surgical team should be informed and decisions made as to whether the patient requires a return to theatre, cardiac angiography +/- PCI or conservative management. Only a small proportion of these patients have angiography as the first intervention.

Table: Abbreviated surgical causes of perioperative myocardial injury post cardiectomy⁵

Primary Myocardial Ischaemia	Non-Ischaemic
Plaque rupture in native coronary artery or graft	Cardiac handling during surgery
Thrombus formation native coronary artery or graft	Direct injury /trauma to myocardium
Acute graft failure:	Surgical myectomy
Kinking or stretching	Reperfusion injury / inflammation post CPB
Occlusion	Inadequate cardioprotection from cardioplegia
Anastomotic stenosis	
Spasm of graft	
Arterial graft spasm	

Adapted from reference (5)

c) Dynamic Left ventricular outflow tract obstruction (LVOTO)

This is the presence of a dynamic left ventricular outflow tract obstruction due to a combination of changes in anatomy and/or physiology resulting in the intermittent reduction in cardiac output. Risk factors include: hypovolaemia, cardiac hypertrophy, aortic valve replacement (aortic stenosis), high dose inotropes.¹ Ideally patients at risk of LVOTO should be identified from intra-operative TOE leading to enhanced vigilance. Basic therapeutic interventions include: keep the patient well filled, avoid inotropes / tachycardia / vasodilators and preferential use of vasoconstrictors with minimal inotropic effect such as noradrenaline.

d) The Right Ventricle

The right ventricle is particularly sensitive to insult and should not be forgotten in the context of cardiogenic shock. Post cardiectomy right ventricular failure risk factors include: prolonged cardiopulmonary bypass time, suboptimal myocardial protection, right ventricular ischaemia and pre-existing right ventricular failure or pulmonary vascular disease. There may be diagnostic doubt regarding the cause of cardiogenic shock in the post cardiectomy patient. If in doubt get more information with an expedient echo +/- cardiac output monitoring.

References:

1. Chioncel O, Parissis J, Mebazza A, Thiele H, Desch S et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock – a position statement from the Heart Failure Association of the European Society of Cardiology. *European Journal of Heart Failure*. 2020. Available from: doi: 10.1002/ejhf1922
2. Haufler T, Krüger B, Bettex D, Rudiger A. Shock management for cardio-surgical ICU patients – the golden hours. *Cardiac Failure Review*. 2015;1(2):75-82
3. Goldberg, Collins, Vahdatpour. Cardiogenic Shock. *J Am Heart Assoc*. 2019;8. Available from: doi:10.1161/JAHA.119.011991
4. Brand J, McDonald A, Dunning J. Management of cardiac arrest following cardiac surgery. *BJA Education*. 2018;18(1):16-22
5. Theilmann M, Sharma V, Al-Attar N, Bulluck H, Bisteri G, Bunge JJH et al. ESC Joint Working Groups on Cardiovascular Surgery and the Cellular Biology of the Heart position paper: Perioperative myocardial injury and infarction in patients undergoing coronary artery bypass graft surgery. *European Heart Journal*. 2017;38:2392-2411. Available from : doi:10.1093/eurheart/eh383