INTRODUCTION

The incidence of cardiogenic shock in community studies has not decreased significantly over time. Despite decreasing mortality rates associated with increasing utilisation of revascularisation, shock remains the leading cause of death for patients hospitalised with acute myocardial infarction (MI). Although shock often develops early after MI onset, it is typically not diagnosed on hospital presentation. Failure to recognise early haemodynamic compromise and the increased early use of hypotension inducing treatments may explain this observation.

Recently, a randomised trial has demonstrated that early revascularisation reduces six and 12 month mortality. The current American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommend the adoption of an early revascularisation strategy for patients < 75 years of age with cardiogenic shock.

The extent of myocardial salvage from reperfusion treatment decreases exponentially with time to re-establishing coronary flow. Unfortunately, there has been little progress in reducing time to hospital presentation over the past decade, and this perhaps accounts for the stagnant incidence of cardiogenic shock in community studies (7.1%).

The incidence of shock in the PURSUIT trial was 2.9% (1995–97) similar to the 2.5% incidence reported in the non-ST elevation arm of the GUSTO II-B trial (1994–95). A number of strategies that centre on reducing the time to effective treatment may help decrease the incidence of shock. These include public education to decrease the time to hospital presentation, triage and early transfer of high risk patients to selected centres, and early primary percutaneous coronary intervention (PCI) or rescue PCI for failed thrombolysis in high risk patients.

DEFINITION

The clinical definition of cardiogenic shock is decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. Hemodynamic criteria are sustained hypotension (systolic blood pressure, 90 mm Hg for at least 30 minutes) and a reduced cardiac index (2.2 L/min per m²) in the presence of elevated pulmonary capillary occlusion pressure (15 mm Hg). Circulatory shock is diagnosed at the bedside by observing hypotension and clinical signs indicating poor tissue perfusion, including oliguria; clouded sensorium; and cool, mottled extremities. Cardiogenic shock is diagnosed after documentation of myocardial dysfunction and exclusion or correction of such factors as hypovolemia, hypoxia, and acidosis.

EPIDEMIOLOGY & CAUSES

The most common cause of cardiogenic shock is extensive acute myocardial infarction, although a smaller infarction in a patient with previously compromised left ventricular function may also precipitate shock. Shock that has a delayed onset may result from infarction extension, reocclusion of a previously patent infarcted artery, or decompensation of myocardial function in the noninfarction zone because of metabolic abnormalities.

It is important to recognize that large areas of nonfunctional but viable myocardium can also cause or contribute to the development of cardiogenic shock in patients after myocardial infarction. Cardiogenic shock can also be caused by mechanical complications—such as acute mitral regurgitation, rupture of the interventricular septum, or rupture of the free wall—or by large right ventricular infarctions. Other causes of cardiogenic shock include myocarditis, end-stage cardiomyopathy, myocardial contusion, septic shock with severe myocardial depression, myocardial dysfunction after prolonged cardiopulmonary bypass, valvular heart disease, and hypertrophic obstructive cardiomyopathy (Table 1). In a recent report of the SHOCK (SHould we emergently revascularize Occluded Coronaries for shock) trial registry of 1160 patients with cardiogenic shock, 74.5% of patients had predominant left ventricular failure, 8.3% had acute mitral regurgitation, 4.6% had ventricular septal rupture, 3.4% had isolated right ventricular shock, 1.7% had tamponade or cardiac rupture, and 8% had shock that was a result of other causes. Patients may have cardiogenic shock at initial presentation, but shock often evolves over several hours. In the SHOCK trial registry, 75% of patients developed cardiogenic shock within 24 hours after presentation; the median delay was 7 hours from onset of infarction.
PATHOLOGY AND PATHOPHYSIOLOGY

Autopsy studies show that cardiogenic shock is generally associated with loss of more than 40% of left ventricular myocardium. Although the less common syndrome of shock due to predominant right ventricular infarction has now been recognized. Autopsy also show that more than two-third of patients with cardiogenic shock in myocardial infarction having stenosis more than 75% of luminal diameter of all three major coronary arteries, usually involving the left anterior descending coronary artery. Majority of the patients of cardiogenic shock are found to have thrombotic occlusion of the artery supplying the major part of recent infarction.

Other causes of shock in Acute MI include mechanical defect like rupture of ventricular septum, papillary muscle, or free wall with tamponade, right ventricular infarct (RVMI) or marked reduction of preload due to condition like hypovolemia.

Results from the GUSTO trial are similar: Among patients with shock, 11% were in shock on arrival and 89% developed shock after admission. Among patients with myocardial infarction, shock is more likely to develop in those who are elderly, are diabetic, and have anterior infarction. Patients with cardiogenic shock are also more likely to have histories of previous infarction, peripheral vascular disease, and cerebrovascular disease. Decreased ejection fractions and larger infarctions (as evidenced by higher cardiac enzyme levels) are also predictors of the development of cardiogenic shock. Cardiogenic shock is most often associated with anterior myocardial infarction. In the SHOCK trial registry, 55% of infarctions were anterior, 46% were inferior, 21% were posterior, and 50% were in multiple locations. These findings were consistent with those in other series. Angiographic evidence most often demonstrates multivessel coronary disease (left main occlusion in 29% of patients, three-vessel disease in 58% of patients, two-vessel disease in 20% of patients, and one-vessel disease in 22% of patients). This is important because compensatory hyperkinesis normally develops in myocardial segments that are not involved in an acute myocardial infarction; this response helps maintain cardiac output. Failure to develop such a response because of previous infarction or high grade coronary stenoses is an important risk factor for cardiogenic shock and death.

INCEDECE

Recent estimates of the incidence of cardiogenic shock have ranged from 5% to 10% of patients with myocardial infarction. The precise incidence is difficult to measure because patients who die before reaching the hospital are not given the diagnosis. In contrast, early and aggressive monitoring can increase the
apparent incidence of cardiogenic shock. The Worcester Heart Attack Study, a communitywide analysis, found an incidence of cardiogenic shock of 7.5%; this incidence remained stable from 1975 to 1988. In the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries (GUSTO-I) trial, the incidence of cardiogenic shock was 7.2%, a rate similar to that found in other multicenter thrombolytic trials.

The onset of cardiogenic shock in a patient following ST elevation MI heralds a dismal in-hospital prognosis. The 7.2% of patients developing shock in the GUSTO-I trial accounted for 58% of the overall deaths at 30 days. Similarly, the 30 day death rates developing shock in the GUSTO-I trial accounted for 58% of MI heralds a dismal in-hospital prognosis. The 7.2% of patients found in other multicenter thrombolytic trials.

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A primary goal in preventing shock should be an effort to reduce the large proportion of patients presenting with acute ST elevation MI who do not receive timely reperfusion treatment. Successful early reperfusion of the infarct related coronary artery while maintaining integrity of the downstream microvasculature limits ongoing necrosis, salvages myocardium, and may prevent the development of shock in many vulnerable patients. In-hospital development of shock often follows failed thrombolysis or successful thrombolysis followed by evidence of recurrent MI (ST re-elevation), infarct extension (ST elevation in new leads), and recurrent ischaemia (new ST depression). These complications may be significantly reduced by a primary PCI strategy.

**DIAGNOSIS**

Most patients are initially evaluated at the bedside where a reasonably accurate clinical diagnosis of cardiogenic shock may be made according to the following character:

1. Evidence of organ hypo perfusion like cold clammy skin, especially on hand and feet, may be associated with peripheral cyanosis in nail beds, oliguria, and impaired mentation.

2. Marked persistent (more than 30 min) hypotension with systolic arterial pressure less than 80 to 90 mm of Hg.

3. Left ventricular end diastolic pressure (LVEDP) OR Pulmonary capillary wedge pressure (PCWP) more than or equal to 18 mm of Hg.

4. Marked reduction of cardiac index (< 1.8 L/min/m²).

5. **Evidence of Primary cardiac abnormalities.**

An electrocardiogram should be obtained immediately, since evidence of serious abnormalities should direct the investigation toward the myocardium; conversely a normal electrocardiogram virtually excludes the possibility of cardiogenic shock caused by myocardial infarction. A routine chest film can provide valuable clues about the presence of infarction, pulmonary edema or aortic dissection. Echocardiography is an excellent aid in shorting through the differential diagnosis of cardiogenic shock, it also provide information on regional and global systolic wall function valvular integrity, and the presence or absence of pericardial effusion. Insertion of swan-ganz catheter is useful for initiating and monitoring therapy, since the proper use of vasoactive agents in these circumstances requires the simultaneous assessment of cardiac filling pressure and peripheral perfusion.

**MANAGEMENT**

When a patient with cardiogenic shock initially evaluated supportive and resuscitative effort must be swift and definitive to maximize the possibilities of survival. The initial management protocol to combat shock is given in the table which is detailed later.
IONOTROPIC CATECHOLAMINE WITHOUT
PREDOMINANT VASOCONSTRICTIVE
PROPERTIES:

Dobutamine: A β2 agonist with predominant effect of β2 receptors thus it increases cardiac contractility as dopamine without causing substantial vasoconstriction and with less tachycardia and arrythmogenesis. It also augments diastolic coronary blood flow and collateral blood flow to ischemic area.

In practice, it has little effects on peripheral vascular resistance or lowers it and increases cardiac output. It does not raise blood pressure when severe hypotension is present. It usually administered at a starting dose 2.5 – 5 μg/kg/min and increased to 20 μg/kg/min depending on hemodynamics and heart rate.

When patients has substantial hypotension (Systolic blood pressure <90mm of Hg) and evidence of tissue hypoperfusion dopamine uses as intravenous ionotropic agents, but when blood pressure is adequate but cardiac output must be augmented, dobutamine is preferred. When a patients is receiving a high dose (>15 μg/Kg/min) of dopamine, norepinephrine is generally substituted in a dose of 2-20 μg/min.

3. Phosphodiesterase Inhibitors:

Phosphodiesterase inhibitors, amrinone, milrinone can increases contractility without adrenergic stimulation, leading to impaired cardiac output and pulmonary pressure with less effect on myocardial work. This agent should be reserved for those in whom therapy with catecholamine has failed to improve cardiac performance or those in whom arrhythmia or ischemia limits the catecholamine use. Long term studies show a disturbing increases in mortality among patients with heart failure who are treated with phosphodiesterase inhibitors.

B. Vasodilators:

It can be beneficial for patients who are in shock, but extreme caution should be used because of the risk of precipitating further hypotension and thereby reducing coronary blood flow. Either intravenous nitroglycerine or sodium nitroprusside can be used; nitroglycerine is less potent as an arteriolar vasodilator but may have the advantage of not producing coronary a. cesteala (preferential coronary blood flow to non ischemic vascular bed). Vasodilators are particularly important when mitral valvular regurgitation is a major part of the path physiological processes. Vasodilators should generally be withheld until the blood pressure is stabilized and hemodynamic monitoring is begun so as to ensure that beneficial effects are produced by the drug.

On occasion, the severity of cardiac pump dysfunction will require the use of two divergent therapeutic modalities in order to facilitate left ventricular emptying. The most commonly utilized of this combined therapies is nitroprusside and dopamine. The principal advantage offered by nitroprusside in this combination is a reduction in left ventricular preload. The cardiac output not appreciably increased by the addition of nitroprusside to dopamine therapy. The advantage offered by dopamine in this combination is an augmentation of cardiac output and the maintenance of systemic arterial pressure. A less frequently combination dobutamine and nitroprusside, has been shown to result in higher cardiac output and lower pulmonary capillary wedge pressures than with either drug alone.

C. Intra Aortic Balloon Pumping (IABP):

The capacity of the intraaortic balloon pump to increases diastolic coronary arterial perfusion and simultaneously to decreases after load with out increasing myocardial oxygen consumption makes it an attractive method of stabilizing the patients with cardiogenic shock.

By inflating in the aorta during diastole and by deflating during systole the IABP reduces after load during ventricular systole and increases coronary perfusion during diastole. The decrees in after load and increased coronary perfusion account for it TMs efficacy in cardiogenic shock and ischemia.

It is particularly useful as a stabilizing bridge to facilitate diagnostic angiography, and revascularization and repair of mechanical complications of acute MI.
Safely the pump can be inserted at the bedside; operator experience is critical to successful placement and management. The most important contraindication of IABP insertion is

1. Aortic regurgitation
2. Severe peripheral vascular disease.

Although vascular and bleeding complications have been reported in 5-10% of treated patients, the use of smaller vascular sheaths has reduced this risk. Vascular complications almost are transient. Hemorrhage at the site of insertion, particularly after removal, is relatively common. Meticulous maintenance of the insertion site, physical examination of the lower extremities, and attention to the timing and characteristic of balloon inflation are necessary.

**RECOMMENDATION FOR INTRA AORTIC BALLOON COUNTERPULSATION**

**Class I**

Cardiogenic shock not quickly reversed with pharmacologic therapy as a stabilizing measure for angiography and revascularization. Acute mitral regurgitation or ventricular septal defect complicating myocardial infarction as a stabilizing therapy for angiography and repair/revascularization.

Recurrent intractable ventricular arrhythmias with hemodynamic instability.

Refractory post myocardial infarction angina as a bridge to angiography and revascularization.

Randomized to tPA or rt PA in GUSTO-III. Although these results are not definite because of broad confidence limits on the observations, angiographic evidence supports a low reperfusion rate in patients with shock compared with other patients with ST segment elevation MI. This is confirmed in hypotensive experimental models. For this reason, thrombolytic therapy is reserve for whom an angiographic suit and emergency revascularization are not immediately accessible.

**E. Combination of Intra – aortic Balloon Counter pulsation and Fibrinolytic Therapy**:

Use of IABP facilitates thrombus dissolution by tPA in experimental model with and without hypotension. Non randomized data from NRMI and a community experience suggest lower mortality rates with this combined therapy. For these reasons, when primary PCI is not feasible and fibrinolytic therapy is administered the concurrent use of the IABP for patients in cardiogenic shock may be beneficial.

**F. Percutaneous Transluminal Coronary Angioplasty (PTCA)**:

Early revascularization is recommended for patients less than 75 years of age with ST elevation or LBBB who develop shock within 36 hrs of MI are suitable for revascularization that can be performed within 18 hrs of MI and are suitable if performed within 36 hrs of shock. (2008 AHA Guideline).

Several retrospective trials have examine the effect of angiography on mortality rate in patients with cardiogenic shock and researchers have consistently found that patients with successful reperfusion have much better outcomes than those without successful reperfusion.

The GUSTO-I trial, a sub group analysis of the 2972 patients with cardiogenic shock showed that 30 day mortality rate was significantly lower in patients who had Angioplasty (43% compared with 61% for patients with shock on arrival; 32% compared with 61% for patients who developed shock). In this trial patients treated with an aggressive strategy (coronary angiography performed on or at 24 h of shock onset with revascularization PTCA or bypass surgery. Had a significantly lower mortality rate (38%, compared with 62%). This benefit was present even after adjustment for base line characteristic and persisted for up to one year.

Recent reports have suggested that coronary stenting may improve outcome after either failed or suboptimal PTCA or as a primary approach.

A recent report of stenting in patients with shock reported success rate of 94% with placement of stent in 47% of patients; the in hospital mortality rate 26%. Another study of stenting of failed angioplasty in patients with cardiogenic shock reported a mortality rate of 27%.

The role of ant platelet, platelet glycoprotein GP IIb/IIIa antagonists have shown to improve short term clinical outcome after angioplasty especially in patients at high risk for complications. However published experience. With GP IIb/IIIa receptor inhibition in patients with cardiogenic shock is far limited to case reports, but extrapolation from other settings suggest that they may play an important adjunctive role in patients with shock who undergo angioplasty.

**G. Coronary Artery Bypass Surgery (CABG)**:

There are so many studies that show, favourable outcome for patients with cardiogenic shock who had CABG. In patients with cardiogenic shock left main coronary artery disease or triple vessels diseases are common and potential contribution of ischemia in non infarct zone to myocardial infarction in patients with shock would support complete revascularization.

Published series include more than 200 patients who have undergone surgical revascularization as a treatment of cardiogenic shock overall perioperative mortality has been low as compared with medical therapy alone, average in 40% but substantial selection bias is present in this non randomized study.
studies. Lone potential advantage of surgical revascularization is that myocardium remote from the acute infarction can also be revascularized, permitting better compensatory function.1

THE SHOCK trial35 compared direct invasive strategies that included urgent coronary angiography, PCI or CABG with initial medical stabilization including IABP, thrombolytic therapy and delayed revascularization as clinically determined. The primary end point 30 day mortality showed a non significant 9.3% absolute and a 17% relative reduction in favor of early revascularization. Six month and one year mortality rate were lower (P-0.027 P- 0.025 ) in favor of early revascularization consistent with 13 lives saved per 100 patients treated. There was an interaction between treatment effect and age younger than 75 years versus age more than 75 year or older. However, the SHOCK registry demonstrated that the elderly who was clinically selected to undergo early revascularization appear to derived benefit that is similar to younger age.

There are strong recommendation about urgent CAG with IABP support and PCI or CABG, depending on the coronary anatomy for this patients younger than 75 years with cardiogenic shock due to predominant left ventricular failure complicating acute ST segment elevation, or new left bundle branch block MI, elderly patients should be clinically selected according to their physiological age, co-morbidities and patients a s preference. Despite the absence of data for shock due to non ST segment elevation MI or predominant right ventricular infarction, some author recommended similar aggressive strategy.

H. Other New Approaches :

New percutaneous techniques may have important applications in patients with cardiogenic shock. In particular percutaneous cardiopulmonary bypass38 and other methods designed to augment cardiac output39 may be able to sustained vital organ function while the reperusions of the infarct related artery and others areas of severe stenosis is attempted. Aggressive effort to sustained life can now include use of left ventricular assisted device as a bridge to cardiac transplantation.40

SPECIAL SITUATION

Right ventricular myocardial infarction (RVMI) with shock :

RVMI occur up to 30% of cases with inferior and posterior MI, and it is clinically significant up to 10% of patients.41 Mortality rate of inferior wall MI associated with RVMI is high, 25.30% ; as opposed to an overall mortality rate of 6% in inferior wallMI.41 RV involvement should always be considered and should be specifically short out in inferior wall MI with clinical evidence of low cardiac output put because of therapeutic approaches are quite different in the presence of RV involvement from those who have predominantly left ventricular failure.

Diagnosis

Clinically the trad of elevated jugular venous pressure, systemic hypotension and clear lung fields in presence of an inferior and posterior infarction suggest the possibility of RVMI, jugular venous distention that is enhanced by inspiration (Kussmaula TM s sign) in the setting of inferior wall MI is highly suggestive of RV involvement but may not be manifest with volume depletion.

ECG shows more than one mm of ST segment elevation in lead V3R and V4R which is the single most powerful predictor of RVMI in inferior wall MI. Echocardiography also helpful in diagnosis and can show depressed cardiac contractility. Right atrial pressure more than 10 mm of Hg that is greater than 80% of pulmonary capillary wedge pressure is a sensitive and specific sign of RVMI.42 Other causes of heart failure in inferior wall MI also should be excluded (cg. Arrhythmia, ongoing ischemia, previous infarction in another location, mechanical complications like papillary muscle rupture and VSD.)

MANAGEMENT OF SHOCK IN RVMI

Maintain Right Ventricular Preload :
- Volume loading (IV normal saline)
- Avoid use of nitrate and diuretics

Maintain A-V synchrony.
- AV sequential pacing for symptomatic high degree heart block

Prompt cardio version for hemodynamically significant SVT.

Inotropic Support :
- Dobutamine (if cardiac output fails to increase after volume loading )

Reduce Right Ventricular After Load with Left Ventricular Dysfunction

Intra Aortic Balloon Pump (IABP)

Arterial vasodilators (nitroprusside, hydralazine)

ACE inhibitors.

Reperfusion

Thrombolytic agent

Primary PTCA

CABG (in patients with multi vessel disease).

(IV; intra venous, AV : Atrioventricular , SVTY Supraventricular Tachycardia, ACE : Angiotensine converting enzyme. PTCA : Percutaneous Transluminal Coronary Angioplasty, CABG : Coronary Artery Bypass Graft).

A recent study using direct angioplasty showed that restoration of normal flow resulted in dramatic recovery of right ventricular function and a mortality rate only 2% , where as unsuccessful reperfusion was associated with persistent homodynamic
compromise and a mortality rate of 58%.

**ACUTE MITRAL REGURGITATION**

Ischemic mitral regurgitation is usually associated with inferior myocardial infarction and ischemia or infarction of the posterior papillary muscle, which has a single blood supply (usually from the posterior descending branch of a dominant right coronary artery). Papillary muscle rupture usually occurs 2 to 7 days after acute myocardial infarction; it presents dramatically with pulmonary edema, hypotension, and cardiogenic shock. When a papillary muscle ruptures, the murmur of acute mitral regurgitation may be limited to early systole because of rapid equalization of pressures in the left atrium and left ventricle. More important, the murmur may be soft or inaudible, especially when cardiac output is low. Echocardiography is extremely useful in the differential diagnosis, which includes free-wall rupture, ventricular septal rupture, and infarction extension pump failure. Hemodynamic monitoring with pulmonary artery catheterization may also be helpful. Management includes afterload reduction with nitroprusside and intra-aortic balloon pumping as temporizing measures. Inotropic or vasopressor therapy may also be needed to support cardiac output and blood pressure. Definitive therapy, however, is surgical valve repair or replacement, which should be undertaken as soon as possible because clinical deterioration can be sudden. Ventricular Septal Rupture Patients who have ventricular septal rupture have severe heart failure or cardiogenic shock, with a pansystolic murmur and a parasternal thrill. The classic finding is a left-to-right intracardiac shunt (a “stepup” in oxygen saturation from right atrium to right ventricle). On pulmonary artery occlusion pressure tracing, ventricular septal rupture can be difficult to distinguish from mitral regurgitation because both can produce dramatic “V” waves. The diagnosis is most easily made with echocardiography. Rapid stabilization — using intra-aortic balloon pumping and pharmacologic measures — followed by surgical repair is the only viable option for long-term survival. The timing of surgery is controversial, but most experts now suggest that operative repair should be done early, within 48 hours of the rupture.

**FREE-WALL RUPTURE**

Ventricular free-wall rupture usually occurs during the first week after myocardial infarction; the classic patient is elderly, female, and hypertensive. The early use of thrombolytic therapy reduces the incidence of cardiac rupture, but late use may increase the risk. Free-wall rupture presents as a catastrophic event with a pulseless rhythm. Salvage is possible with prompt recognition, pericardiocentesis to relieve acute tamponade, and thoracotomy with repair.

**REVERSIBLE MYOCARDIAL DYSFUNCTION**

In addition to hibernating and stunned myocardium, potentially reversible causes of myocardial dysfunction include sepsis-associated myocardial depression, myocardial dysfunction after cardiopulmonary bypass, and inflammatory myocarditis. In sepsis and, to some extent, in myocarditis, myocardial dysfunction seems to result from the effects of inflammatory cytokines, such as tumor necrosis factor and interleukin-1. Myocardial dysfunction can be self-limited or fulminant, with severe congestive heart failure and cardiogenic shock. In the latter situation, cardiovascular support with a combination of inotropic agents (such as dopamine, dobutamine, or milrinone) and IABP may be required for hours or days to allow sufficient time for recovery. If these measures fail, mechanical circulatory support with left ventricular assist devices can be considered. These devices can be used as a bridge to cardiac transplantation in eligible patients or as a bridge to myocardial recovery; functional improvement with such support can be dramatic.

**CONCLUSIONS**

Mortality rates in patients with cardiogenic shock remain frustratingly high (50% to 80%). The pathophysiology of shock involves a downward spiral: Ischemia causes myocardial dysfunction, which in turn worsens ischemia. Areas of nonfunctional but viable myocardium can also cause or contribute to the development of cardiogenic shock. The key to a good outcome is an organized approach with rapid diagnosis and prompt initiation of therapy to maintain blood pressure and cardiac output. Expedient coronary revascularization is crucial. When available, emergency cardiac catheterization and revascularization with angioplasty or coronary surgery seem to improve survival and represent standard therapy at this time. In hospitals without direct angioplasty capability, stabilization with IABP and Thrombolysis followed by transfer to a tertiary care facility may be the best option. The SHOCK multicenter randomized trial provides important data that help clarify the appropriate role and timing of revascularization in patients with cardiogenic shock.

**REFERENCES**


