Faculty of Medicine
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ROLE OF PERCUTANEOUS MECHANICAL SUPPORT DEVICES IN MANAGEMENT OF POST M.I CARDIOGENIC SHOCK

An Essay

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AIM of WORK

Our aim in this study is to summarize the current knowledge regarding post- Myocardial Infarction Cardiogenic shock and discuss the role of percutaneous mechanical support devices in its management, stressing important takeaway points for emergency physicians.

Introduction

Historically, cardiogenic shock (C.S) was nearly always fatal after myocardial infarction (MI). Mortality rates were consistently reported at approximately 80% until the 1990s, when the mortality improved but only slightly, to 50% to 60%. Since then, advances in medical therapies, aggressive reperfusion, and the increasing availability of percutaneous coronary intervention (PCI) and cardiac bypass for patients in C.S. resulted in further decrease in mortality rates**.** **(Reynolds,et al.,2008)**

C.S. occurs in 5% to 10% of patients hospitalized with M.I (ST-segment elevated MI) and is a common cause of death in this group an unknown additional number of prehospital patients die from C.S. making the exact incidence uncertain. **(Hochman,et al.,2008)**

Risk factors for development of post-MI CS include older age, anterior location of M.I, hypertension, diabetes, multivessel occlusions, left bundle branch block, and prior history of cardiac disease or heart failure **(Reynolds, et al., 2008)**

The purest clinical definition of C.shock includes poor cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. The diagnosis is indicated by the combination of low systolic blood pressure (BP <90 mmHg or a value 30 mmHg below basal levels for at least 30 minutes), an elevated arteriovenous

oxygen difference (>5.5 ml per deciliter) , and a depressed cardiac index (<2.2 liters per minute per square meter of body-surface area) in the presence of an elevated pulmonary capillary wedge pressure (>15mm Hg) **(Collins et al., 2006)**

C.S. progresses through a vicious cascade of events that link both systolic and diastolic dysfunction. Systolic dysfunction leads to reductions in cardiac output and stroke volume which then lead to reduced coronary perfusion and ischemia. While diastolic dysfunction leads to increased LV end-diastolic pressure and pulmonary congestion, which then leads to hypoxia and ischemia. Ischemia is the common link and leads to further systolic and diastolic dysfunction, which promotes the cascade of events. **(circulation journal, 2008)**

In the past, almost no one survived cardiogenic shock. Now, about most of the people who go into cardiogenic shock survive. This is because of prompt recognition of symptoms and improved treatments, such as medicines and devices. These treatments can restore blood flow to the heart and help the heart pump better. In some cases, devices that take over the pumping function of the heart are used. **(Francis et al., 2006)**

Cardigenic shock is a strenuous clinical challenge. Noninvasive management through inotropic support allows frequent clinical improvement, yet one is repeatedly confronted with refractory cases necessitating more invasive support. **(Stephane et al., 2011)**

The development of ventricular assist devices has broadened the means with which one can treat acute heart failure. Percutaneous ventricular assist devices (pVAD) have risen from recent technological advances. They are smaller, easier, and faster to implant, all important qualities in the setting of acute heart failure. **(Diego Arroyo et al., 2011)**

The idea of a mechanical assistance first appeared in the 1950s, yet the first device which is the intra-aortic balloon pump (IABP) only appeared in the late 1960s. It remains, today, the most common, cheapest, and easily available cardiac mechanical device. **(Christenson et al., 2003)**

Although C.S. may stem from many causes (eg, acute valvular disorders, cardiac tamponade), this essay focuses solely on post-MI C.S. and discusses therapeutic recommendations and stresses important takeaway points for emergency physicians.

Pathophysiology of Post- myocardial infarction Cardiogenic Shock

Cardiogenic shock (C.S), as a complication of acute myocardial infarction (AMI) continues to be an unfortunately very serious problem that carries a high mortality rate. **(Griffith, 1954).**

The syndrome of cardiogenic shock (C.S) has been defined as the inability of the heart-as a result of impairment of its pumping function-to deliver sufficient blood flow to the tissues to meet resting metabolic demands. ***(Dole, 1983)***

Clinical Definition:

As early as 1912, Herrick described the clinical features of cardiogenic shock in patients with severe coronary artery disease: a weak, rapid pulse; feeble cardiac tones; pulmonary rales; dyspnea; and cyanosis. These signs are not always present, however. In the full-blown state of shock, some of the characteristic signs of shock are unequivocally evident. In the earlier phases of shock or in less severe circumstances, these signs may be more subtle. For example, a reduction in urine output or slight confusion may represent a state preceding shock. The signs of shock may also be affected by chronic or current medical therapy. For example, a patient taking oral β-blockers on a chronic basis may not be tachycardic during shock, although the heart rate may be much more rapid than in

the basal state. Therefore, these signs should be evaluated in the context of the specific clinical setting. The systemic signs of hypoperfusion that may be detected in cardiogenic shock include an altered mental state; cold, clammy skin; and oliguria. **(*Hollenberg, 1998)***

Determinants of Cardiogenic shock

1- Cardiac Output manifestations:

Cardiogenic shock has been defined as a state of tissue hypoxia caused by reduced systemic cardiac output in the presence of adequate intravascular volume. **(Hasdai, 2000)**

This broad definition accounts for the great variability in the diagnosis of shock among different clinicians and investigators. In several series, cardiac index measurements of 2.2 L/min/m2 or less were considered supportive of the diagnosis, others have considered measurements of only 1.8 L/min/m2 or less to be indicative of cardiogenic shock **(Braunwald, 1998).**

In addition to the boundaries of specific index measurements used, there may be variability in measuring methods. Currently, cardiac output is measured primarily using pulmonary artery catheter. This technique demands some

expertise and carries certain, albeit minimal risks. It is therefore, not universally used in patients with cardiogenic shock **(Topol, 1984)**.

2- Blood Pressure Changes:

Although systemic hypotension is essential to the diagnosis of the syndrome, the severity of hypotension defining shock varies. Commonly, the cut point for the systolic blood pressure is less than 90 mm Hg. Clinical studies consider the diagnosis of cardiogenic shock in patients with blood pressure measurements greater than 90 mmHg if they require medications and support devices to maintain normal hemodynamic parameters, as the patient may initially have signs associated with the clinical state of cardiogenic shock in the presence of systolic blood pressure measurements greater than 90 mm Hg. Therefore, hypotension alone should not be the basis for the diagnosis in the absence of signs of peripheral hypoperfusion. **(Menon, 2000)**

Pathophysiology of cardiogenic shock

Before the advent and widespread use of thrombolytic therapy, up to 20% of patients with AMI developed cardiogenic shock. Thrombolytic therapy has helped reduce this number by (3- 7%). **(Holmes, 1995)**

Despite the reduction in the overall incidence of cardiogenic shock, the prognosis was poor, with in-hospital mortality rates approaching 70% to 80%; in the few survivors, there was a 5-year mortality rate of 60% **(Alpert, 1993)**, ***Fig. (1)***.

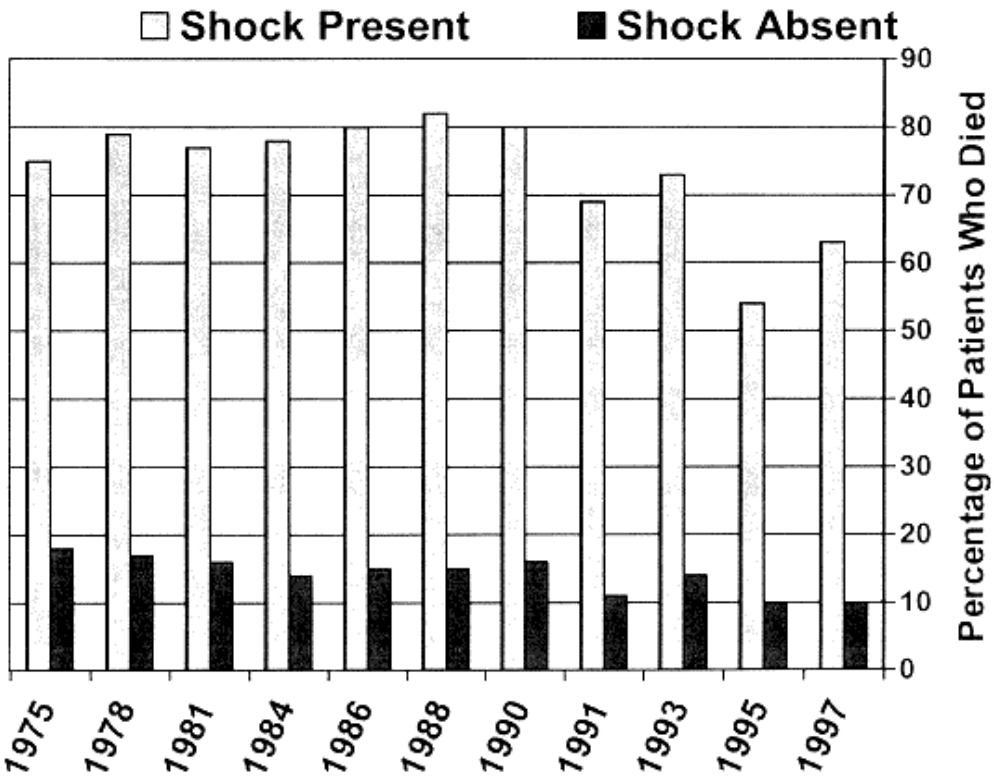

Fig (1): *Temporal trends in in-hospital death rates among patients with acute myocardial infarction according to the presence or absence of cardiogenic shock. (Goldberg R et al., N Engl. J Med 1999)*

In simplest terms, cardiogenic shock occurs when an acutely failing heart no longer can meet systemic demands and the sequelae of end-organ hypoperfusion result. cardiogenic shock comes most frequently secondary to acute myocardial infarction, but still many other cases can lead to cardiogenic shock e.g. arrhythmias, end-stage valvular heart disease, severe myocarditis, cardiac tamponade, and constrictive or hypertrophic cardiomyopathies. **(Alpert, 1993)**

Several mechanisms can lead to the development of cardiogenic shock after acute myocardial infarction. In a recent report of the SHOCK trial (Should we emergently revascularize Occluded Coronaries for shocK) registry of 1160 patients with cardiogenic shock , 74% of patients had predominant left ventricular (LV) failure (8.3%) had acute mitral valve regurgitation, 4.6% had ventricular septal rupture, 1.7% acute tamponade/free-wall rupture, 3.4% had isolated right ventricular shock, and 8% had other causes. **(Hochman, 1995)**

This latter group includes cardiac-related diagnoses such as dilated cardiomyopathy, medication excess, and cardiac catheterization complications. It is important to emphasize that the mechanical complications secondary to myocardial rupture (free wall, septum, or papillary muscle rupture) account for the minority of the cases. In the majority of the patients, the cause

of death is low cardiac output secondary to predominant LV failure, which in turn leads to organ failure, arrhythmias, and irreversible shock. **(Hochman, 1998)** ***Fig (2)***

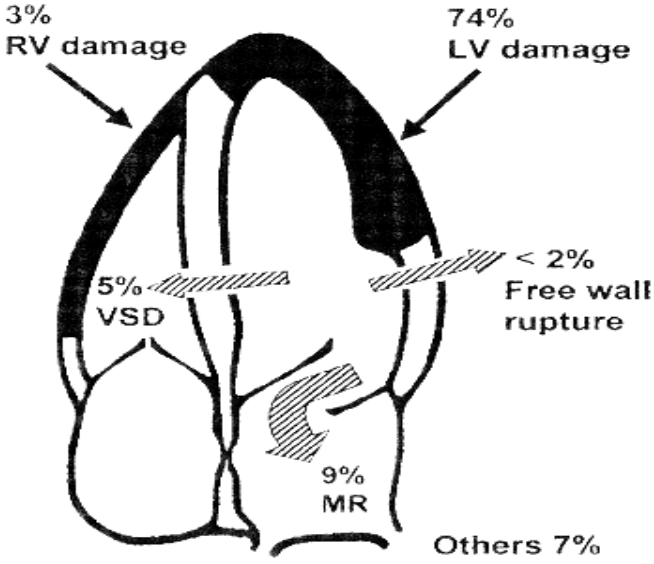

Fig.(2).

Apical 4 chamber view with relative incidence of the mechanisms responsible for C.S. (M.R). mitral regurge: (VSD), ventricular septal defect
(Davies CH, Q J Med 2001)

Systemic effects of cardiogenic shock

Cardiac dysfunction in patients with cardiogenic shock is usually initiated by myocardial infarction or ischemia. The myocardial dysfunction resulting from ischemia worsens that ischemia, creating a downward spiral, ***Fig(3)***. When a critical mass of left ventricular myocardium is ischemic or necrotic and fails to pump, stroke volume and cardiac output decrease. Myocardial perfusion, which depends on the pressure gradient

between the coronary arterial system and the left ventricle and on the duration of diastole, is compromised by hypotension and tachycardia. This, in turn, exacerbates ischemia. The increased ventricular diastolic pressures caused by pump failure further reduce coronary perfusion pressure, and the additional wall stress elevates myocardial oxygen requirements, further worsening ischemia. Decreased cardiac output also compromises systemic perfusion, which can lead to lactic acidosis and further compromise of systolic performance. When myocardial function is depressed, several compensatory mechanisms are activated, including sympathetic stimulation to increase heart rate and contractility and renal fluid retention to increase preload. These compensatory mechanisms may become maladaptive and can actually worsen the situation when C. shock develops. Increased heart rate and contractility increase myocardial oxygen demand and exacerbate ischemia. Fluid retention and impaired diastolic filling caused by tachycardia and ischemia may result in pulmonary congestion and hypoxia. Vasoconstriction to maintain blood pressure increases myocardial afterload, further impairing cardiac performance and increasing myocardial oxygen demand. This increased demand, in the face of inadequate perfusion, worsens ischemia and begins a vicious cycle that will end in death if uninterrupted, **(Steven, 1999)** ***Fig. (3)***

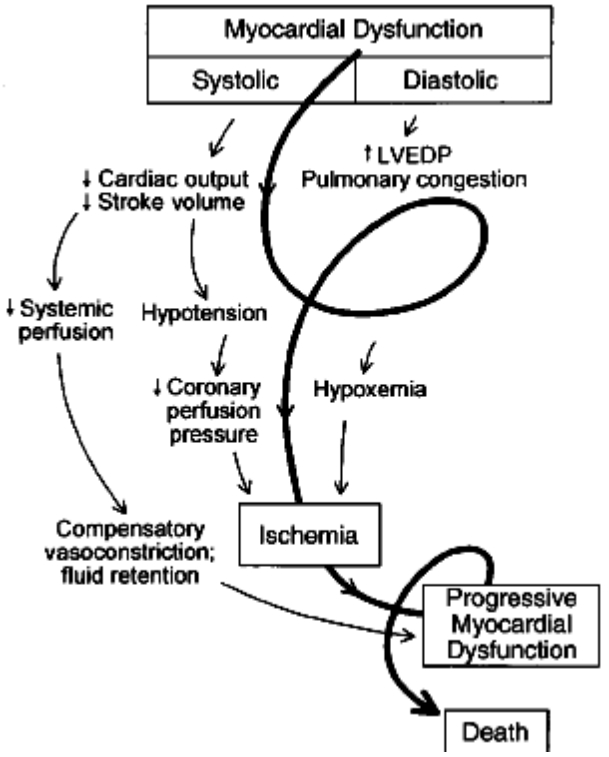

Fig (3): *The downward spiral in C. shock*
(Steven MH, et al., Cardiogenic shock Ann Intern Med 1999.)

As the shock state persists, hypoperfusion of both the myocardium and peripheral tissues will induce anaerobic metabolism in these tissues and may result in lactic acidosis. An earlier study has shown that the serum lactate level is an important prognostic factor in cardiogenic shock **(Afifi, 1974).**

The accumulation of lactic acid may cause mitochondrial swelling and degeneration, inducing glycogen depletion, which, in turn, impair myocardial function and inhibit