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Cardiogenic Shock

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Major advances in the management of congestive heart failure¹ and acute myocardial infarction² have ushered in a new era of optimism about the prognosis of patients with these disorders. Cardiac failure with cardiogenic shock continues to be a frustrating clinical problem, however, because of persistent mortality rates of 30 to 90 percent and substantial morbidity among hospitalized patients^{3,4}. The management of this condition requires a rapid, well-organized approach. Avoiding futile intervention that is unlikely to prolong life or give patients a reasonable chance of functional recovery is also increasingly important in this era of limited resources and public awareness of the consequences of high-risk, low-yield therapy.

Definition

The syndrome of cardiogenic shock 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 demands⁵. Thus, the purest clinical definition of cardiogenic shock includes poor cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. When hemodynamic monitoring is available, the diagnosis is indicated by the combination of low systolic blood pressure (<90 mm Hg or a value 30 mm Hg below basal levels for at least 30 minutes), an elevated arteriovenous oxygen difference (>5.5 ml per

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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 (>15 mm Hg). One caveat is that the ability of the individual patient to tolerate poor tissue perfusion depends somewhat on the suddenness of the event; some patients with chronic valvular or myocardial disease meet the above criteria but remain ambulatory and functional.

Most patients are initially evaluated at the bedside, where a reasonably accurate clinical diagnosis of cardiogenic shock may be made according to the following criteria: hypotension as defined above; evidence of poor tissue perfusion, including oliguria, cyanosis, cool extremities, or altered mentation; and persistence of shock after the correction of nonmyocardial factors contributing to poor tissue perfusion and myocardial dysfunction, most commonly hypovolemia, arrhythmias, hypoxia, and acidosis.

Pathophysiology

Autopsy studies show that cardiogenic shock is generally associated with loss of more than 40 percent of left ventricular myocardium,^{6,7,8} although the less common syndrome of shock due to predominant right ventricular infarction has now been recognized⁹. A right ventricular infarction causes reduced diastolic compliance and systolic dysfunction of the right ventricle, resulting in a volume-sensitive state, in contrast to the pressure-sensitive state produced by infarction of the left ventricle. In addition, the cumulative nature of myocardial damage must be recognized; often a small additional amount of infarction in a patient in whom compensation for previous myocardial damage is marginal can result in cardiogenic shock ([Table 1](#)). Furthermore, loss of a functional component of the heart (a valve or valvular support, free wall, or ventricular septum) because of acute ischemia or necrosis can also result in cardiogenic shock in isolation or can contribute to shock caused by the loss of left ventricular function^{10,11,12,13,14,15}. Finally, a variety of miscellaneous cardiovascular causes can result in acute deterioration leading to cardiogenic shock; these include acute myocarditis, sustained arrhythmia, acute primary valvular catastrophe in the absence of coronary artery disease, and decompensation in patients with previous end-stage cardiomyopathy.

View this table: [Table 1. Pathophysiology of Cardiogenic Shock.](#)
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The progressive deterioration that occurs in the absence of intervention in cases of cardiogenic shock can be seen as a vicious circle¹⁶. Initial compensatory mechanisms include activation of the sympathetic nervous system, effects on renal and neurohormonal regulation, and local vasoregulation ([Figure 1](#)). The activation of the sympathetic nervous system is triggered by baroreceptors and chemoreceptors, leading to an increase in heart rate and arterial and venous vasoconstriction, an increase in myocardial contractility, and shifting of fluid into the vascular compartment. The renin-angiotensin system is activated by inadequate renal perfusion pressure and sympathetic stimulation of the renal nerves. An excess of angiotensin II leads to peripheral vasoconstriction and

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View this table: **Table 2.** Therapeutic Approach to Cardiogenic Shock.
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Clinical Epidemiology

The incidence of cardiogenic shock is difficult to measure. Differences in emergency medical systems probably create differential rates of death before arrival at the hospital, thus reducing the number of patients given the diagnosis. Improvements in diagnostic capabilities, by such means as better use of pressure monitoring or imaging, could have the opposite effect: an apparent increase in incidence resulting from more accurate diagnosis. Among patients with acute myocardial infarction who survive to hospital admission, the incidence of cardiogenic shock ranges from 6 to 20 percent^{19,20}. The Worcester Heart Attack Study³ found that the incidence of cardiogenic shock actually increased between 1975 and 1988 after adjustment for other base-line covariates, although the raw or unadjusted rate did not change.

As compared with patients who have acute myocardial infarction without cardiogenic shock, patients who have shock are older, more frequently have anterior myocardial infarction, more often have had a previous infarction, and more commonly have a history of angina or congestive heart failure^{20,21,22,23}. Several studies have found a higher prevalence of diabetes among patients with cardiogenic shock, but other risk factors for the development of atherosclerosis are not more common among patients in whom shock develops. Pathological and angiographic studies have consistently found a greater prevalence of occlusion of the left anterior descending artery, multivessel coronary artery disease, and persistent occlusion of the infarct-related artery among patients with cardiogenic shock. Patients in whom cardiogenic shock develops after hospital admission tend to be older than others, are more apt to have a history of infarction and diabetes mellitus, and have depressed systolic function with higher creatine kinase or lactate dehydrogenase values^{21,22,23,24}. The results of recent angiographic and echocardiographic studies emphasize the importance of the zone of myocardium remote from the acute infarction in the development of cardiogenic shock^{25,26,27}. The normal response of one wall of the heart to infarction is to develop "compensatory" hyperkinesis of the uninvolved myocardium. When the uninvolved myocardium is fibrotic or when its blood flow is compromised by high-grade stenosis, this compensatory mechanism is thwarted. The absence of hyperkinesis in the ventricular wall opposite the region of the acute infarct is an important risk factor for the development of cardiogenic shock and for death^{24,26,28,29}.

Clinical Assessment

When there is evidence of inadequate tissue perfusion, noncardiac causes of shock should be sought while resuscitative measures are initiated ([Table 2](#)). Hypovolemia, sepsis, pulmonary embolus, aortic dissection, rupture of an abdominal aortic aneurysm, and pericardial tamponade are among the most common causes of shock in the absence of myocardial decompensation. Unless there is evidence of pulmonary edema, intravascular volume should be measured by the immediate administration of saline to the patient with other signs of shock. 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. Careful documentation of the findings of the physical examination, including the blood pressure, heart rate, results of cardiac and pulmonary auscultation, mental status, and physical signs of peripheral-tissue perfusion, will aid rapid therapeutic decision making. A routine chest film can provide valuable clues about the presence of infection, pulmonary edema, or aortic dissection. Echocardiography is an excellent noninvasive aid in sorting through the differential diagnosis of cardiogenic shock; it also provides information on regional and global systolic wall function, valvular integrity, and the presence or absence of pericardial effusion. Insertion of a 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 pressures and peripheral perfusion.

General Management

Resuscitative and supportive efforts should be initiated immediately, at the same time as the diagnostic evaluation. Critical elements include adequate oxygenation and ventilation, correction of electrolyte and acid-base abnormalities, relief of pain, and restoration of sinus rhythm. If the patient is to be treated with thrombolytic therapy, avoidance of the nasal route of airway intubation will reduce the risk of major bleeding. In patients with inadequate tissue perfusion and adequate intravascular volume, infusion of inotropic or vasopressor drugs should be begun immediately. Dobutamine is preferable to other sympathomimetic amines unless substantial hypotension is present; it augments diastolic coronary blood flow and collateral blood flow to the ischemic area while increasing myocardial contractility, raising cardiac output, and lowering left ventricular filling pressures^{30,31}. When moderate hypotension and hypoperfusion are present, dopamine is preferable, since vasoconstriction in the peripheral vessels is often needed to maintain vital organ-tissue perfusion. Norepinephrine is used when profound hypotension is present to maximize blood pressure while other resuscitative efforts are instituted. The phosphodiesterase inhibitors amrinone and milrinone can increase contractility without adrenergic stimulation, leading to improved cardiac output and pulmonary pressures³² with less effect on myocardial work³³. Because of their longer half-life, however, especially in patients with renal impairment, these agents should be reserved for those in whom therapy with catecholamines has failed to improve cardiac performance or those in whom arrhythmia or ischemia limits the catecholamine dose. Long-term studies show a disturbing increase in mortality among patients with heart failure who are treated with phosphodiesterase inhibitors³⁴.

Vasodilators 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 nitroglycerin or sodium nitroprusside can be used; nitroglycerin is less potent as an arteriolar vasodilator,³⁵ but it may have the advantage of not producing coronary "steal" (preferential coronary blood flow to nonischemic vascular beds)³⁶. Vasodilators are particularly important when mitral valvular regurgitation is a major part of the pathophysiologic process. 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.

Patients with the syndrome of right ventricular infarction leading to cardiogenic shock are particularly sensitive to volume depletion and prone to hemodynamic deterioration resulting from bradycardia and the loss of atrioventricular synchrony precipitated by advanced heart block. The focus of therapy in such patients should be the immediate restoration of adequate left ventricular filling pressure, the maintenance of sinus rhythm or synchronized pacing, and the use of dobutamine to stimulate right ventricular systolic function^{37,38,39}.

Intraaortic Balloon Pumping

The capacity of the intraaortic balloon pump to increase diastolic coronary arterial perfusion and simultaneously to decrease afterload without increasing myocardial oxygen consumption makes it an attractive method of stabilizing the patient with cardiogenic shock. Although the use of the balloon pump can produce temporary clinical and hemodynamic improvement,^{40,41,42,43,44,45,46} survival was prolonged in one observational study only among patients who subsequently underwent revascularization⁴⁷. Despite the absence of adequate clinical trials, intraaortic balloon pumping has become a standard component of therapy for cardiogenic shock in the era of myocardial reperfusion with thrombolytic agents and percutaneous transluminal coronary angioplasty (PTCA). Although vascular and bleeding complications have been reported in 5 to 20 percent of treated patients,⁴⁸ the use of smaller vascular sheaths has reduced this risk, and vascular complications are almost always transient.

Thrombolytic Therapy

Although thrombolytic therapy generally reduces the risk of death by 20 to 50 percent in patients with acute ST-segment elevation,^{49,50} no large, randomized clinical trial has demonstrated that such treatment improves the survival of patients with cardiogenic shock. In the Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI) study,⁴⁹ the 30-day mortality rates were 69.9 percent for 146 patients treated with streptokinase and 70.1 percent for 134 patients treated with placebo. The confidence limits for treatment effects in these patients substantially overlapped the 20 percent reduction in mortality observed in the total study population. Patients with a systolic blood pressure of less than 100 mm Hg at randomization who were treated with streptokinase in the Second International Study of Infarct Survival (ISIS-2)⁵⁰ had a lower mortality rate than those who received placebo, but these patients may not all have met the criteria for shock. In two recent, large comparative trials, streptokinase was associated with lower mortality (55 to 65 percent) than tissue plasminogen activator (63 to 78 percent) in the group of patients who had cardiogenic shock at entry^{51,52}.

Angiographic studies in patients with cardiogenic shock who are treated with thrombolytic therapy⁴ document reperfusion in only 40 to 50 percent of infarct-related arteries. These data, combined with the disappointing result in patients with cardiogenic shock in the trials described above,^{49,50,51,52} arouse concern that treatment with thrombolytic therapy alone may not be sufficient to improve survival in such patients. Since thrombolytic therapy generally reduces mortality in more severely ill patients,⁵³ however, it may be inappropriate to use small subgroup analyses to assess its benefits in patients with cardiogenic shock, especially since the confidence limits for the treatment effect overlap the expected effect of this therapy in all treated patients.

Percutaneous Transluminal Coronary Angioplasty

Although there have been no randomized studies of PTCA in cardiogenic shock, several uncontrolled studies have suggested that this procedure can reduce both short-term and long-term mortality^{4,54,55,56,57}. Caution must be used in interpreting the results of many of these uncontrolled studies, for several reasons. Selection bias was introduced by the exclusion of elderly patients and those with severe existing disease. In addition, in calculating survival, none of the published series reported the number of patients who died between the time the decision

was made to proceed with angiography or angioplasty and the time when the procedures were to be done, possibly leading to overestimation of the benefits of a strategy of direct angioplasty. Until a randomized trial can be completed that compares angioplasty with medical therapy, the best assumption is that direct angioplasty is superior to supportive measures alone or to thrombolytic therapy. In the 72 percent of patients in whom PTCA is successful, in-hospital survival rates average 75 percent, as compared with 22 percent in those in whom the procedure is unsuccessful. Recent evidence from a series of randomized trials that included very few patients with true cardiogenic shock demonstrated a particular benefit of angioplasty over thrombolytic therapy for patients with large infarctions or hemodynamic compromise^{58,59,60}. In summary, if an experienced angiographic facility is immediately available, we recommend direct angioplasty as a primary mode of therapy, but in hospitals without such facilities we recommend the rapid administration of a thrombolytic agent, followed by transfer for angiography. Randomized trials comparing these therapies are needed.

Coronary-Artery Bypass Surgery and New Techniques

Published series include more than 200 patients who have undergone surgical revascularization as treatment for cardiogenic shock^{40,42,47,61}. Overall perioperative mortality has been low as compared with the rates with medical therapy alone, averaging 40 percent, but substantial selection bias is present in these nonrandomized studies. One potential advantage of surgical revascularization is that myocardium remote from the acute infarction can also be revascularized, permitting greater compensatory function. An area of great interest has been the use of specific cardioplegic solutions that may improve the degree of myocardial salvage⁶². Enthusiasm for the role of surgical revascularization in cardiogenic shock remains restrained, however, because of high operative mortality rates as compared with those for elective surgery, the inherent delays, and the high degree of surgical expertise and medical resources required.

New percutaneous techniques may have important applications in patients with cardiogenic shock. In particular, percutaneous cardiopulmonary bypass⁶³ and other methods designed to augment cardiac output⁶⁴ may be able to sustain vital organ function while the reperfusion of the infarct-related artery and other areas of severe stenosis is attempted. Aggressive efforts to sustain life can now include the use of left ventricular assist devices as a bridge to cardiac transplantation⁶⁵. Their use is generally restricted to centers with high patient volumes and full cardiovascular services.

Overall Aggressiveness of Care and Regionalization

Both providers of medical care and patients have become increasingly aware of the implications of initiating aggressive and costly therapies in situations in which the chances for meaningful functional recovery are limited^{66,67}. Since patients' preferences with respect to the aggressiveness of care are highly individual and are not strongly related to age or previous functional disability,^{68,69} there is no substitute for addressing the issue directly with the patient or the patient's surrogate decision maker. The available data suggest that for most patients with severe functional disability, poor mental status, or advanced age, aggressive therapy should not be initiated unless the patient or the patient's family has clearly expressed a preference for such a strategy. The issue of the level of support and intervention should continue to be addressed on a regular basis when patients do not respond to therapy.

In the absence of aggressive, highly technical care, mortality among patients with cardiogenic shock is exceedingly high (70 to 90 percent). Hospitals without facilities for intraaortic balloon pumping or high-risk angioplasty and surgical intervention should begin initial resuscitative measures and then make a rapid decision about transfer to a hospital with the necessary facilities and personnel. This decision should be based on the physician's estimate of the probability of success, coupled with the patient's and the family's preferences and expectations. Every hospital without tertiary care facilities should have a plan for transferring patients so that unnecessary delays are avoided.

Conclusions

Cardiogenic shock continues to be associated with high mortality. The prognosis may be improved by the rapid diagnosis of the underlying cause, stabilization of the patient, and early consideration of revascularization and correction of mechanical defects. The available evidence supports the concept that early and definitive restoration of coronary blood flow is the most important intervention when ischemic heart disease is the principal cause of the syndrome. Thus, early coronary angiography and angioplasty should be performed in patients with a preference for aggressive care. The most promising approach to this syndrome, therefore, is to prevent its development by early reperfusion in patients with myocardial infarction and by appropriate long-term pharmacologic treatment and mechanical intervention in patients with structural heart disease.

Source Information

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