Guidelines for the Management of Acute Heart Failure and Cardiogenic shock

The clinical presentation of acute heart failure ranges from the sudden appearance of dyspnea to frank cardiogenic shock. The management of acute heart failure differs for the various patient groups within the clinical spectrum of this condition The physician should exclude noncardiac disorders whose clinical presentation can be similar in many respects to that of acute heart failure (eg, noncardiogenic pulmonary edema). Also, noncardiac support measures (eg, ventilator therapy) are important in the overall management of these critically ill patients.

Acute heart failure can be grouped clinically into *acute cardiogenic pulmonary edema, cardiogenic shock,* and *acute decompensation of chronic left heart failure.*

With few exceptions, patients presenting with acute heart failure require hospital admission, particularly those with an initial episode of failure.

Therapeutic interventions to produce hemodynamic improvement and stability must be undertaken expeditiously in these patients. In addition, it is imperative to obtain quickly those diagnostic tests necessary to detect causes of heart failure which are best treated by special therapeutic approaches. Myocardial injury/infarction, high degree atrioventricular (AV) block, ventricular tachycardia, pericardial tamponade and pulmonary embolism are examples of the latter causes of failure. Once etiologies of this type have been excluded, further diagnostic testing to determine the etiology of the heart disease generally can be deferred to a more appropriate time.

Acute Cardiogenic Pulmonary Edema

A brief medical history and directed physical examination are generally sufficient to initiate therapy. An intravenous catheter should be placed, blood obtained for essential laboratory studies and the patient placed on oxygen therapy.

a)The sublingual administration of nitroglycerin (0.4 to 0.6 mg, repeated every 5 to 10 minutes four times as needed) is of value. Nitroglycerin is effective in patients with acute cardiogenic pulmonary edema due to both ischemic and nonischemic causes. If systemic blood pressure is acceptable (generally a systolic blood pressure $\geq$95 to 100 mm Hg), nitroglycerin can be administered intravenously (starting dose 0.3 to 0.5 µg/kg body weight per min) as well.[2]

Sodium nitroprusside (starting dose 0.1 µg/kg per minute) may be selected for patients not immediately responsive to nitrate therapy and for those whose pulmonary edema is, in large part, attributable to severe mitral or aortic valvular regurgitation or marked, systemic hypertension.[2] The dose is advanced as needed to improve the patient's overall clinical and hemodynamic status, using a systemic systolic pressure of 85 to 90 mm Hg as the usual lower limit for dose incrementation in patients previously normotensive and as long as adequate perfusion of vital organs is maintained.

b)Furosemide (20 to 80 mg intravenously) should be given shortly after the diagnosis of acute pulmonary edema is established.

c)Morphine sulfate (3 to 5 mg intravenously) is effective in ameliorating many of the symptoms of acute pulmonary edema and can be administered safely to most patients with this condition. However, morphine sulfate should be administered with caution to patients with chronic pulmonary insufficiency and those with respiratory or metabolic acidosis in whom suppression of ventilatory drive can cause a drastic lowering of systemic pH

d) Intubation and mechanical ventilation are of value in patients with severe hypoxia that does not respond rapidly to therapy and in those with respiratory acidosis.

e)Patients with severe refractory pulmonary edema may benefit from intra-aortic balloon counterpulsation. This procedure is of particular value if the patient is to undergo urgent cardiac catheterization and definitive intervention. Intraaortic balloon counterpulsation should not be used in patients with significant aortic valvular insufficiency or aortic dissection. A rare patient presenting with severe refractory pulmonary edema and a correctable lesion may need to proceed directly to the operating room after prompt diagnosis (usually by clinical examination and echocardiography) of the precipitating lesion; examples include rupture of a papillary muscle with acute, marked mitral regurgitation and acute aortic dissection complicated by proximal coronary artery occlusion or marked aortic valvular insufficiency, or both.

In most patients acute cardiogenic pulmonary edema can be stabilized with appropriate intervention and frequent, intermittent bedside evaluation without the routine introduction of indwelling pulmonary or systemic arterial catheters. Placement of a pulmonary artery balloon catheter should be considered in this setting if (1) the patient's clinical course is deteriorating; (2) recovery from the acute presentation is not proceeding as expected; (3) high dose nitroglycerin or nitroprusside is required for clinical stabilization; (4) dobutamine or dopamine are needed to augment systemic blood pressure and peripheral perfusion; or (5) uncertainty exists regarding the diagnosis of acute cardiogenic pulmonary edema.

Early in the initial evaluation of patients with acute pulmonary edema, the physician must determine whether acute myocardial injury/infarction is present. At this stage, this determination is based on clinical assessment and the electrocardiogram (ECG). Evidence of acute myocardial injury/infarction should raise consideration of urgent myocardial reperfusion therapy. Cardiac catheterization and coronary arteriography followed by the most appropriate interventional procedure (if readily available), or thrombolytic therapy should be considered.

Transthoracic Doppler-two-dimensional echocardiography is indicated in all patients who present with acute cardiogenic pulmonary edema, unless there are obvious precipitating factors and the patient's cardiac status had been adequately evaluated previously. Depending on the urgency for confirming or establishing a diagnosis, the procedure is performed as soon as possible after initial stabilization. Transesophageal echocardiography may be required to diagnose or more clearly define certain lesions (eg, ruptured chordae tendinae, aortic dissection).

TABLE I Initial Diagnostic Evaluation of Acute Pulmonary Edema

1. Focused history/physical examination
2. Twelve-lead ECG
3. Continuous ECG monitoring
4. Blood-serum studies: complete blood count (CBC); electrolytes, blood urea nitrogen (BUN), creatinine and cardiac enzyme levels
5. BP monitoring
6. Digital pulse oximetry/arterial blood gases
7. Chest radiograph
8. Transthoracic Doppler-two-dimensional echocardiography (TEE where indicated)
9. Cardiac catheterization/coronary arteriography for suspected coronary artery disease (1) if acute intervention for myocardial injury/infarction is anticipated; (2) to determine the cause(s) for refractory acute pulmonary edema
10. Careful intake /output chart

TABLE II Therapeutic Management of Acute Pulmonary Edema

1. Oxygen therapy
2. Nitroglycerin, sublingually or intravenously
3. Intravenous administration of a diuretic (eg, furosemide)
4. Morphine sulfate
5. Administration of cardiovascular support drugs to attain and stabilize clinical-hemodynamic status (eg, intravenous infusion of nitroprusside, dobutamine, dopamine)
6. Thrombolytic therapy or urgent revascularization (angioplasty or coronary artery bypass surgery) for acute myocardial injury/infarction
7. Intubation and mechanical ventilation for severe hypoxia that does not respond rapidly to therapy and for respiratory acidosis
8. Definitive correction of the underlying cause (eg, mitral valve replacement or repair of acute, severe mitral regurgitation) when indicated and clinically feasible

CARDIOGENIC SHOCK

Introduction

If cardiogenic shock is not caused by a reparable lesion, or if the lesion is not repaired in an efficient and effective manner, the mortality rate is ≥85%.Therefore, cardiogenic shock should be approached with diagnostic and therapeutic vigor in an attempt to identify a treatable lesion and to intervene in a definitive manner. Patients presenting with hypoperfusion but adequate blood pressure may be considered to be in near shock and should be approached in the same manner to prevent the progression to frank shock and death.

Definition and diagnosis

Cardiogenic shock is a disorder caused by decreased systemic cardiac output in the presence of adequate intravascular volume, resulting in tissue hypoxia. Shock may be the result of severe left-ventricular dysfunction,

Definition of cardiogenic shock

Cardiogenic shock is defined as a systolic blood pressure of <90 mm Hg for 1 h that is:

Not responsive to fluid administration alone,

Secondary to cardiac dysfunction, or

Associated with signs of hypoperfusion (ALTERED MENTAL STATUS,COLD CLAMMY SKIN, OLIGURIA)or cardiac index <2·2 L/min per m^2 and pulmonary-capillary wedge pressure >18 mm Hg

Aetiology and pathophysiology

The diagnosis of cardiogenic shock is made after having excluded other potential causes of hypotension and systemic hypoperfusion. In addition, conditions such as pneumothorax or pericardial tamponade (unrelated to myocardial rupture) may be associated with clinical findings that resemble cardiogenic shock.

The underlying mechanism for cardiogenic shock complicating acute coronary syndromes should be determined. About 40% of patients with cardiogenic shock as a complication of acute myocardial infarction will have had a prior infarction,

Right-ventricular infarction is a common cause of shock among patients with ST-segment-elevation myocardial infarction; The initial reports of cardiogenic shock were in patients with large infarctions associated with the loss of more than 40% of the left ventricle..

Ventricular-septal defects, myocardial rupture, and acute mitral regurgitation can all cause a state of cardiogenic shock in the presence of adequate left-ventricular function. These causes of cardiogenic shock are often termed mechanical, which is indicative of their dissociation from the degree of reduced cardiac contractility.

Cardiogenic shock occurs in 2·9% of patients with unstable angina and in 2·1% with non-ST-elevation myocardial infarction. Shock is a more common complication of ST-segment-elevation myocardial infarction than of other types of acute coronary syndromes .The occurrence of cardiogenic shock ranged from 4·2% to 7·2%.The median time to the occurrence of shock among patients with persistent ST-segment elevation who develop in-hospital shock is 10 or 12 h, with most experiencing shock within the first 48 h after enrolment

PRINCIPLES OF MANAGEMENT

The general principles of management are rapid recognition of the condition; rapid exclusion or treatment, or both, of readily reversible causes; and prompt stabilization of the clinical and hemodynamic status. As in most emergency situations, many of these activities are performed simultaneously without a routine or set sequence for all patients.

An ECG should be obtained, continuous ECG monitoring instituted, an intravenous catheter inserted and an indwelling arterial cannula placed for continuous blood pressure monitoring. An indwelling pulmonary artery catheter may be inserted.

If arrhythmias are present, their contribution to the hemodynamic state and need for rapid cardioversion or pacing must be determined.

In patients presenting with cardiogenic shock, a relative or absolute reduction in left ventricular filling pressure as the cause of hypotension must be excluded. Because of prior diuretic therapy or acute interspace volume shifts, it is estimated that 10% to 15% of patients with an acute myocardial infarction may be significantly volume depleted. Right ventricular infarction, pericardial tamponade and certain instances of pulmonary embolization are other common examples of acute heart failure that fall into this category. Unless there are signs of left heart volume overload (eg, S_3 gallop, moist pulmonary rales, vascular or pulmonary congestion on chest radiograph), normal saline solution should be administered intravenously at a reasonably fast rate ($\geq$500-mL bolus, followed by 500 mL/h). Jugular venous pressure is not a consistently reliable indicator of left heart filling pressure, and, thus, elevation of jugular pressure

does not obviate the need for fluid administration in a number of clinical situations (eg, pericardial tamponade, right ventricular myocardial infarction).

In patients with an acute inferior infarction with shock, a right ventricular infarction should be suspected, resulting in right ventricular failure and inadequate filling of the left heart system. Right-sided precordial

ECG leads in addition to the standard leads should be used in these patients. An injury pattern is often but not uniformly observed on the right precordial lead tracings (V_3R, V_4R) in patients with a right ventricular infarction. The diagnosis of right ventricular infarction frequently can be made on the basis of clinical findings such as an increase in jugular venous pressure during inspiration. An echocardiogram or the insertion of a pulmonary artery balloon catheter, or both, are helpful in patients in whom the diagnosis is unclear. Right-sided cardiac pressure recordings generally show mean right atrial and right ventricular filling pressures equaling or exceeding pulmonary artery occlusive (wedge) pressure with normal or low pulmonary artery pressures. Echocardiography can be used to establish or confirm the presence of right heart involvement, assess tricuspid valve competence, evaluate the extent of left heart damage and left ventricular function and exclude pericardial tamponade (which can have a similar clinical presentation)..

Fluid volume administration is a major component of therapy for patients with right ventricular infarction to maintain the elevated right-sided filling pressure necessary to maintain cardiac output. Fluid administration initially may be guided by clinical variables (eg, systemic blood pressure, peripheral perfusion, urine output, ventricular gallop sounds), but hemodynamic monitoring by a pulmonary artery catheter is generally required to optimize volume administration. Failure of fluid volume administration to achieve clinical and hemodynamic improvement and stabilization in these patients requires additional therapeutic approaches (eg, dobutamine, intra-aortic balloon counterpulsation or interventional procedures). The use of diuretic drugs or vasodilator agents in patients with a right ventricular infarction can result in severe hypotension.

Occasionally, vasodilator agents and diuretic drugs produce hypotension in patients with an acute myocardial infarction and pulmonary edema because the translocation of fluid into the lung reduces intravascular volume.

Severe hypotension (systolic blood pressure $\leq$70 mm Hg) or clinical shock, or both, occurring in the presence of volume overload or persisting after bolus saline administration should be approached with moderate (4 to 5 µg/kg body weight per minute), then, if necessary, increasing doses of dopamine.Dobutamine may be combined.

If hypotension or clinical shock or near shock persists at dopamine doses $\geq$15 µg/kg per minute, institution of intra-aortic balloon counterpulsation should be considered for patients with a potentially reversible condition or as a bridge to transplantation. If intra-aortic balloon counterpulsation is not available, norepinephrine can be added to increase systemic blood pressure to acceptable levels (systolic pressure $\geq$80 mm Hg) and the patient transferred on an emergency basis to a more comprehensive medical facility. Patients with volume overload (or after adequate volume loading) in near shock or with a lesser degree of systemic hypotension often respond favorably to dobutamine (2 to 3 µg/kg per minute initially) or low to moderate doses of dopamine (2 to 5 µg/kg per minute initially).Newer inotropes and inodilators may be used.

During treatment, attention should remain focused on (1) the status of the patient's intravascular volume; (2) the condition of the patient's ventricular function; (3) the presence of myocardial injury/infarction; and (4) whether correctable, mechanical lesions are present.

1. Status of intravascular volume. The best means of assessing and monitoring intravascular volume in these patients is by hemodynamic measurements through the pulmonary artery catheter. However, in the presence of left ventricular dysfunction, the usual pressure criteria used to assess intravascular volume do not apply. The optimal left ventricular diastolic filling pressure, as estimated by the pulmonary artery occlusive (wedge) pressure (or pulmonary artery diastolic pressure when comparable), for most patients with shock or near shock secondary to acute myocardial infarction resides between 14 to 18 mm Hg.

2. Status of patient's ventricular function. Transthoracic Doppler-two-dimensional echocardiography is helpful in assessing the status of ventricular function and guiding additional studies and interventions. The finding of segmental hypokinesia or akinesia suggests the presence of occlusive coronary artery disease, although similar

findings may occur in some patients with acute myocarditis or idiopathic dilated cardiomyopathy. Global left ventricular enlargement and dysfunction generally indicate a more diffuse or chronic process.Mechanical defects such as tamponade, IVS Rupture,Papillary muscle rupture,mitral regurgitation, free wall rupture,RV MI may be apparent.

The thermodilution pulmonary artery catheter, though not widely recommended now, can provide diagnostic information and a general functional assessment of ventricular and overall cardiovascular performance. Depressed stroke volume in the setting of elevated pulmonary artery occlusive (wedge) pressure generally indicates a significant reduction in left ventricular function. Elevated V (systolic) waves in the wedge position

suggest the presence of mitral regurgitation, although the absence of elevated V waves does not exclude mitral regurgitation. Significant oxygen desaturation in mixed venous blood drawn from the pulmonary artery indicates depressed systemic oxygen delivery.

3. Existence of myocardial injury/infarction. Emergency cardiac catheterization and selective coronary arteriography should be considered in the patient with cardiogenic shock or near shock and ECG evidence of acute myocardial injury/infarction.[3,17,18,19] Reperfusion of the injured/infarcted region of the acutely occluded coronary artery in patients with shock not responding to fluid administration has been reported[20] to reduce the mortality rate from <85% to ≤60%. Ideally, the patient is transferred to the catheterization laboratory shortly after initial stabilization.

4. Are correctable, mechanical lesions present? Clinical evaluation and transthoracic Doppler-two-dimensional echocardiography are the primary methods to diagnose or exclude most of these lesions initially. The most commonly encountered potentially reversible defects in this clinical setting are pericardial tamponade; massive pulmonary embolism; rupture of chordae tendinae, papillary muscle or ventricular septum; critical valvular stenosis or acute regurgitation; aortic dissection with complicating lesions (eg, acute coronary occlusion, acute aortic valvular regurgitation); acute obstruction or incompetency of a prosthetic heart valve; and cardiac tumors. Additional diagnostic testing, such as transesophageal echocardiography and cardiac catheterization may be necessary to more precisely define the lesion(s) and disease process before definitive surgical intervention.

. **TREATMENT& OUTCOME TRENDS**

Trends in outcome

The outcome of cardiogenic shock complicating acute coronary syndromes seems to have improved during a 23-year period, with the greatest improvement in the past decade (1990s). The in-hospital death

rate among patients with cardiogenic shock was greater than 70% from 1975 to 1990, but declined to 59% in 1997..

Supportive therapy

The pharmacological treatment of cardiogenic shock includes the intravenous administration of dopamine or other vasopressors, and dobutamine if systemic vascular resistance is high. Diuretics or fluids should be given on the basis of the estimated left-ventricular filling pressure: diuretics for patients with raised filling pressures, and fluids for patients with decreased filling pressures (characteristic of right-ventricular infarction). Oxygen should be given by mask, and in case of deterioration in respiratory status, mechanical ventilation should be contemplated. Nitrates should be avoided because of their hypotensive effect. Patients with mechanical causes for shock should be stabilised, with the decision regarding the optimum timing for surgical intervention to be made by the cardiologist and surgeon. In certain circumstances of mechanical shock, however, the patient's condition may mandate prompt transfer to the surgical suite even before attempts at stabilisation are fully implemented.

The use of right-heart catheterisation to titrate therapy is controversial. Although much information can be gleaned from right-heart catheterisation, no guidelines or algorithms have been established for the management of shock patients based on the derived measurements.

Thrombolytic therapy

The outcome of cardiogenic shock is closely linked to the patency of the culprit coronary arteries.Accordingly, reperfusion therapy with thrombolytic agents has decreased the occurrence of shock among patients with persistent ST-segment-elevation myocardial infarction.

Thrombolytic therapy for patients who have already developed shock has been very disappointing. The lack of benefit of thrombolytic agents in treating cardiogenic shock may be attributed to decreased coronary thrombolysis in states of low perfusion pressure.

Intra-aortic balloon counterpulsation

Intra-aortic balloon counterpulsation is valuable for stabilising patients with cardiogenic shock and may improve the efficacy of thrombolytic agents. It increases diastolic coronary arterial perfusion and decreases systemic afterload without increasing myocardial oxygen demand. Few data are available to support its use in improving outcomes of patients with shock; however, preliminary data have shown that it might be beneficial in conjunction with revascularisation. Surprisingly, the intra-aortic balloon pump is underused in current clinical practice.'

Revascularisation

Given the discouraging results attained with thrombolytic therapy for patients with cardiogenic shock, there is an emerging interest in the use of mechanical revascularisation in these patients. Whereas the main focus has been on coronary angioplasty, fairly good outcomes have also been reported in surgical series of patients with cardiogenic shock.

Although the reported results with mechanical revascularisation seem to be better than those with medical therapy (including thrombolytic therapy), there may be a selection bias in the use of mechanical revascularisation.

Metabolic therapy

The myocardium often sustains severe functional and structural damage during prolonged ischaemia and systemic hypoperfusion. Thus, although the patency of the culprit artery may be restored, the impaired myocardial metabolism may

prevent the normal recovery of cardiac function. There have been experimental attempts to restore myocardial metabolism during shock, including the use of insulin to restore myocardial fatty acid metabolism. Some investigators have suggested treatment with L-carnitine or adenosine analogues. Despite the hypothetical reasoning behind these approaches, there are still no firm clinical data to support their use in clinical practice.

Table III Initial Diagnostic Evaluation of Cardiogenic Shock

1. Focused history-physical examination
2. Twelve-lead ECG (plus occasional right-sided leads)
3. Continuous ECG monitoring
4. Blood-serum studies: complete blood count, platelet count, clotting studies, electrolytes, BUN, creatinine, glucose and cardiac and liver enzymes
5. Arterial blood gases and lactate concentration
6. Chest radiograph
7. Transthoracic Doppler-two-dimensional echocardiography (TEE where applicable)
8. Indwelling arterial cannula for continuous monitoring of systemic blood pressure and for arterial blood gas sampling
9. Tabulation of fluid volume intake, urine output and other fluid volume loss
10. Cardiac catheterization/coronary arteriography if acute revascularization for acute myocardial injury/infarction is anticipated

TableIV Therapeutic Management of Cardiogenic Shock

1. Oxygen therapy
2. In the absence of obvious intravascular volume overload, brisk intravenous administration of fluid volume
3. In the presence of intravascular volume overload or after adequate intravenous fluid volume therapy, intravenous administration of cardiovascular support drugs (eg, dopamine, dobutamine, norepinephrine) to attain and maintain stable clinical-hemodynamic status

4. Urgent coronary artery revascularization for acute myocardial injury/infarction, if readily available by thrombolysis/ primary PTCA with IABP support

Acute Decompensation of Chronic Congestive Heart Failure

The general principles of management for this group of patients are clinical and hemodynamic stabilization, diagnostic studies for reversible precipitating factor(s) and optimization of long-term therapy.

The clinical manifestations of this group of patients generally are secondary to volume overload, elevated ventricular filling pressures and depressed cardiac output.

Mild to moderate symptoms can be treated effectively with intravenously or orally administered diuretic drugs and reinstitution or optimization, or both, of the patients' long-term therapy for chronic heart failure. Unless the presentation is complicated by a precipitating factor (eg, recent myocardial infarction) or a concurrent threatening condition (eg, marked hypokalemia, moderate to marked azotemia, symptomatic arrhythmias), many of these patients do not require urgent hospital admission beyond several hours of observation in an emergency room or outpatient facility.

Moderate to severe symptoms usually require hospital admission, generally in a cardiac or intensive care unit. The diagnostic and therapeutic approach is similar to that for patients presenting with acute heart failure. Once symptoms at rest are largely alleviated, and reasonable clinical and hemodynamic stability is achieved for $\geq$24 hours, any intravenously administered cardiovascular support drugs can be withdrawn (usually in a decremental manner) while orally administered long-term heart failure therapy is optimized.

The underlying cardiac diagnosis has been established for the majority of patients in this group, and the precipitating factors usually can be ascertained by history, physical examination, electrocardiography, serial cardiac enzyme level determinations, an echocardiogram and selected laboratory testing. Extensive diagnostic evaluation is generally not necessary if correctable causes of heart failure have been excluded previously.

TABLEV Indications of Intra-aortic Balloon Counterpulsation

1. Cardiogenic shock, pulmonary edema and other acute heart failure conditions not responding to the proper administration of fluid volume or pharmacologic therapy, or both, in patients with potentially reversible heart failure or as a bridge to heart transplantation

2. Acute heart failure accompanied by refractory ischemia, in preparation for cardiac catheterization/coronary arteriography and definitive intervention

3. Acute heart failure complicated by significant mitral regurgitation or rupture of the ventricular septum; to obtain hemodynamic stabilization for definitive diagnostic studies or intervention, or both

TABLE VI Contraindications of IABP Therapy

1. Significant aortic insufficiency

2. Aortic dissection

3. Patients unresponsive to therapy in whom the cause is known to be uncorrectable or irreversible and who are not candidates for transplantation

4. Patients in the end stage of a terminal illness

5. Bleeding diathesis or severe thrombocytopenia

TABLE VII Indications of Placement of Pulmonary Artery Balloon Catheter

1.Cardiogenic shock or near shock that does not respond promptly to the administration of fluid volume

2.Acute pulmonary edema that does not respond to appropriate intervention or is complicated by systemic hypotension or shock/near shock

3.As a diagnostic tool to resolve any uncertainty of whether pulmonary edema is cardiogenic or noncardiogenic in origin

4.Origin and significance of new cardiac murmur

6. All indications provided echo has failed to resolve the difficulty

TABLEVIII Summary of management of Acute Heart Failure

- Patients presenting with acute heart failure usually should be admitted to the hospital. Initial diagnostic testing for patients with acute heart failure should be limited to those tests necessary to exclude etiologies requiring special therapeutic procedures. Further diagnostic testing generally can be deferred until hemodynamic stability and improvement have been attained.
- Initial laboratory testing generally can be limited to CBC, urinalysis, serum electrolytes, creatinine, cardiac enzymes, blood urea nitrogen, arterial gases and pH, ECG, chest radiograph, and transthoracic Doppler-two-dimensional echocardiography.
- Patients with acute heart failure and evidence of acute myocardial injury/infarction should be considered for urgent cardiac catheterization, coronary arteriography and definitive interventional procedures. Thrombolysis should be considered if these studies cannot be done expeditiously.
- Treatment of acute cardiogenic pulmonary edema consists initially of intravenous diuretic drugs, nitrates, oxygen and morphine. Intubation and mechanical ventilation are of value in selected patients.
- Patients with cardiogenic shock who do not have evidence of volume overload should receive a rapid infusion of intravenous fluids initially and the response observed.
- Patients with cardiogenic shock unresponsive to fluid administration or who have obvious volume overload should be given intravenous dopamine initially.
- A pulmonary artery balloon flotation catheter can be inserted in all patients with cardiogenic shock unless there is rapid response to fluid administration or if echo has been able to appropriately guide therapy. Patients with acute cardiogenic pulmonary edema generally do not require a pulmonary artery catheter. However, catheter insertion is indicated in those not responding appropriately to therapy or those in whomecho has been unable to appropriately guide therapy or it is unclear whether the pulmonary edema is due to cardiac or noncardiac causes.
- Intra-aortic balloon counterpulsation may be indicated in patients with acute heart failure not responding adequately to therapy. The device is particularly helpful in attaining hemodynamic improvement and stability while awaiting additional diagnostic or therapeutic interventions.

SUGGESTED READING

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