Percutaneous Left Ventricular Assist Device in Acute Myocardial Infarction and Cardiogenic Shock

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Despite advances in coronary angioplasty for acute myocardial infarction (MI), the mortality rate for patients presenting with cardiogenic shock remains high. This case review describes the management of a patient with a non–ST segment elevation MI complicated by cardiogenic shock. The clinical and therapeutic utility of a percutaneous left atrial–to–femoral arterial ventricular assist device is discussed.
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Key words: Cardiogenic shock • Myocardial infarction • Heart failure • Left ventricular assist device

A 75-year-old white man presented to the emergency department (ED) with a 1-day history of 5/10 chest pressure with radiation down the left arm, nausea, and shortness of breath. There was no relief with two sublingual nitroglycerin tablets.

His past medical history was significant for an anterior wall myocardial infarction (MI) 19 years ago, hypertension, and hypercholesterolemia. Medications included carvedilol 12.5 mg by mouth twice daily, pravastatin 20 mg by mouth once daily, and aspirin 325 mg by mouth once daily. On presentation to the ED, the blood pressure (BP) was 87/69 mm Hg, heart rate (HR) was 87 beats/min, respiration rate was 18 per minute, oxygen saturation was

Table 1
Baseline Laboratory Data

Test (Units)	Value (Normal Range)	Test (Units)	Value (Normal Range)
Sodium (mEq/L)	139 (135-145)	WBC (1000/uL)	10 (4.4-10.1)
Potassium (mEq/L)	4.9 (3.5-5.2)	Hemoglobin (g/dL)	13.5 (13.4-17)
Chloride (mEq/L)	105 (95-107)	Hematocrit	39.3 (40-50)
Bicarbonate (mEq/L)	24 (21-31)	MCV (fL)	93.6 (80-100)
BUN (mg/dL)	32 (8-22)	Platelets (1000/μL)	177 (140-413)
Creatinine (mg/dL)	1.6 (0.7-1.5)	PT (s)	11.2 (10-12)
CK	799 (55-170)	INR	1.1
CK-MB (ng/mL)	49.9 (< 10)	aPTT (s)	27.4 (24-34)
CK-MB Index	6.2 (< 4)	Troponin (ng/ml)	0.76 (< 0.10)

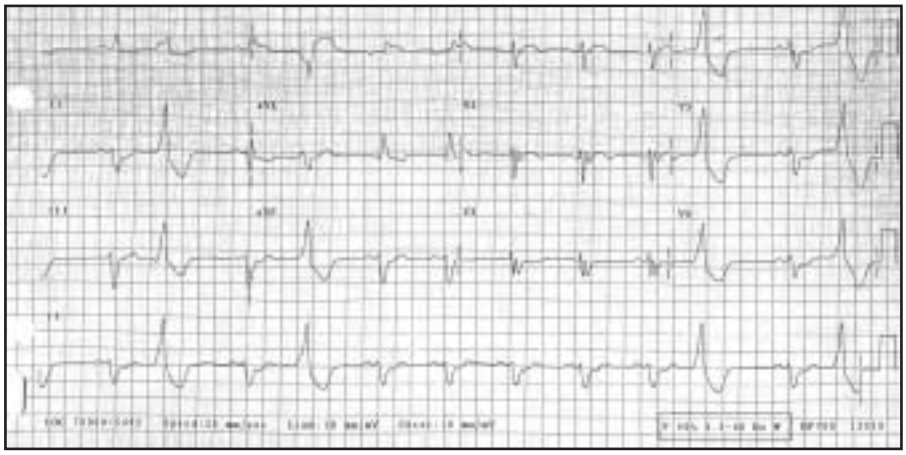
aPTT, activated partial thromboplastin time; BUN, blood urea nitrogen; CK, creatine kinase; CK-MB, creatine kinase myocardial band; INR, international normalized ratio; MCV, mean corpuscular volume; PT, prothrombin time, WBC, white blood count.

97% on room air, and temperature was 36.6°C. The lungs were clear to auscultation and percussion. Cardiac examination revealed a diffuse point of maximal impulse, normal S_1 and S_2, and a grade 2/6 systolic ejection murmur at the apex. The jugular venous pressure (JVP) was not elevated, and the carotid upstrokes and peripheral pulses were intact. Initial laboratory data are summarized in Table 1. Portable chest radiograph was unremarkable. The electrocardiogram (ECG) on presentation is shown in Figure 1. The initial creatine kinase–myocardial band was markedly elevated at 49.9 ng/mL, and the serum creatinine was elevated at 1.6 mg/dL.

In the ED, the patient received an intravenous (IV) bolus of normal saline (250 mL), which improved the BP. The patient was diagnosed with a non–ST segment elevation MI (NSTEMI) and treated with aspirin 325 mg, clopidogrel 300 mg, and enoxaparin 60 mg subcutaneously every 12 hours. The chest discomfort initially resolved; however, there was recurrent chest pain 4 hours later that was unrelieved with morphine. Eptifibatide (180 μg/kg IV bolus followed by an infusion of 2 μg/kg/min) was started. A repeat ECG was obtained (Figure 2). As the chest pain became severe, it was associated with increasing difficulty in breathing.

On arrival to the cardiac catheterization laboratory, the patient was in marked respiratory distress. The BP dropped to 80/50 mm Hg. Physical examination now revealed elevated JVP, rales, an audible S_3, weakened peripheral pulses, and cold extremities. Pressors including IV dopamine and norepinephrine were started. The patient was emergently intubated and given mechanical ventilation. An intra-aortic balloon pump (IABP) was placed. Right heart catheterization results are given in Table 2. Coronary angiography revealed a patent left main artery, severe diffuse disease in the left anterior descending (LAD) artery, totally occluded proximal left circumflex (LCX) artery, and severe 80% mid stenosis in the right coronary artery (Figure 3). The patient underwent successful percutaneous coronary intervention (PCI) of the LCX with implantation of a 3.5 × 24–mm Driver™ stent (Medtronic AVE, Santa Rosa, CA). At the end of the procedure, there was grade 3 epicardial flow in the target vessel (Figure 4); however, the patient remained in cardiogenic shock, with a diastolic augmented pressure of 60 mm Hg despite maximum pressor and IABP support. Because of hypotension (BP = 55/40 mm Hg) and refractory shock, the TandemHeart® (CardiacAssist Inc., Pittsburgh, PA) percutaneous left ventricular assist device (PVAD) was placed. Using

Figure 1. *Electrocardiogram on presentation to the emergency department reveals a normal sinus rhythm, left anterior fascicular block with a nonspecific intraventricular conduction delay, unifocal premature ventricular contractions, ST depression in V_4-V_6, and T wave inversions in I and aVL.*



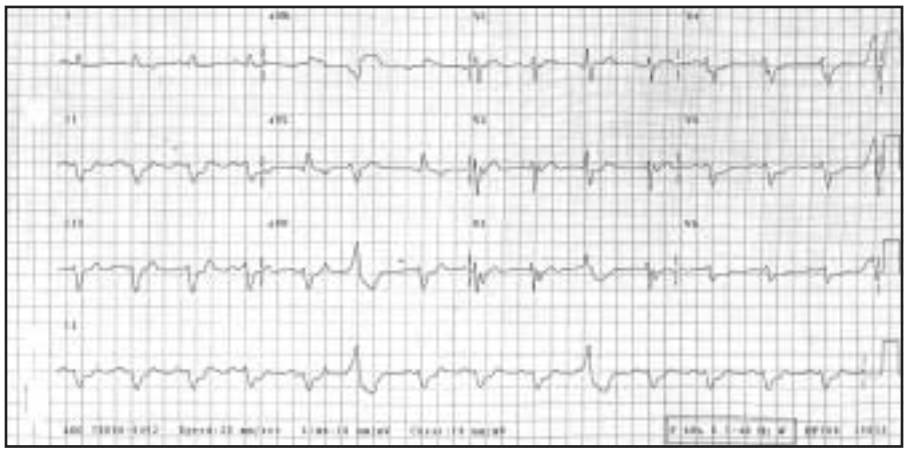


Figure 2. *Electrocardiogram obtained approximately 4 hours after presenting to the emergency department with reoccurrence of chest pain, revealing a normal sinus rhythm, left anterior fascicular block, intraventricular conduction delay, unifocal premature ventricular contractions, nonspecific ST and T wave changes in the lateral precordial leads, and T wave inversion in I and aVL.*

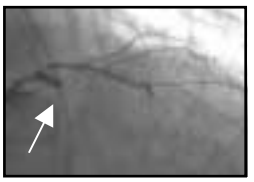


Figure 3. *Total proximal occlusion of the left circumflex artery (arrow).*

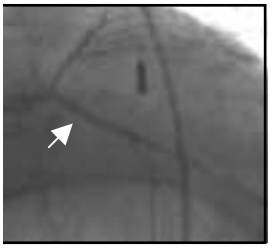


Figure 4. *Postinterventional recanalization of the left circumflex artery (arrow).*

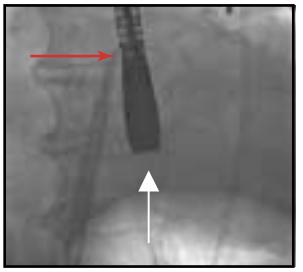


Figure 5. *Transseptal cannulation of the left atrium (red arrow) with wire placement within the left atrium. A transesophageal probe (white arrow) was used to facilitate transseptal puncture; however, it is often not required.*

conventional transseptal puncture techniques, a 25F venous cannula was advanced from the right femoral vein into the left atrium (Figure 5). A 15F cannula was inserted into the left femoral artery to complete the circuit whereby oxygenated blood was shunted from the left atrium into the aorta, thereby "unloading" the damaged ventricle. The flow rate was started at 3 L/min. While in the catheterization laboratory, the patient developed ventricular tachycardia, which was treated with cardioversion and an IV bolus of amiodarone (300 mg). Sinus bradycardia (HR = 26) followed and required a temporary transvenous pacemaker.

On the first postinterventional day, the patient developed oliguric renal failure and was started on continuous venovenous hemofiltration. The TandemHeart device provided approximately two thirds of the cardiac output. The BP ranged from 78-102/43-65 mm Hg, with augmented pressures of 73-103 mm Hg. On the second day the cardiac index (CI) increased to 2.3 L/min/m^2 and the PVAD flow was reduced to 1.5 L/min. The flow rate was reduced even further, and on the third day, the device was explanted with vascular surgical repair of the femoral vessels. On the fourth day, the IABP, the transvenous pacemaker, and hemofiltration were discontinued. The patient was extubated on the sixth day, transferred to the step-down unit on the 11th day, and discharged home on the 46th day.

Table 2
Right Heart Catheterization Results

Right atrial pressure (mm Hg): 16/11, mean 13
Pulmonary artery pressure (mm Hg): 39/32, mean 34
Pulmonary capillary wedge pressure: 32 mm Hg
Superior vena cava oxygen saturation: 62%
Pulmonary arterial oxygen saturation: 60%
Femoral arterial oxygen saturation: 87%
Cardiac output/cardiac index:
Preintervention: 3.43 L/min/1.43 L/min/m^2
Postintervention with PVAD: 4.5 L/min/1.9 L/min/m^2

PVAD, percutaneous ventricular assist device.

Discussion

Coronary artery disease remains the leading cause of death in the United States, accounting for approximately one third of all deaths in people over the age of 35.[1,2] Despite recent advances in the care of acute coronary

disease, cardiogenic shock as a complication of acute MI continues to be associated with a dismal prognosis.[3-5] Furthermore, cardiogenic shock complicates 7%-10% of cases of acute MI and is associated with a 70%-80% mortality.[6] The majority of patients present with an ST elevation MI (STEMI); however, cardiogenic shock may also occur in patients presenting with NSTEMI or unstable angina.[7,8] Holmes and colleagues[7] found significant differences in baseline characteristics between shock patients with and without ST elevation. Specifically, patients with NSTEMI tended to be older, to be diabetic, to have 3-vessel disease, and to present later (76.2 hours vs 9.6 hours, $P < .001$). Cardiogenic shock was less likely to occur in patients with NSTEMI (2.5% vs 4.2%, $P < .001$) but was associated with a higher mortality than that for shock patients with STEMI (72.5% vs 63%, $P = .05$).

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Cardiogenic shock, as a complication of an acute MI, is a state of inadequate tissue perfusion due to extensive left ventricular injury (typically ≥ 40%), resulting in insufficient blood flow to the tissues to meet resting metabolic demands.[4,9,10] It is characterized by marked and persistent (> 30 minutes) hypotension with a systolic BP < 90 mm Hg or > 30 mm Hg below the usual BP, elevated arteriovenous oxygen difference (> 5.5 mL/dL), depressed CI (< 2.2 L/min/m^2), elevated pulmonary capillary wedge pressure (PCWP; > 15 mm Hg), and evidence of peripheral hypoperfusion (oliguria, cyanosis, altered mental status) despite correction of fluid status and arrhythmias.[4,11,12] The most common mechanism is left ventricular pump failure; however, shock may also be caused by ventricular septal or papillary muscle rupture, myocarditis, or right ventricular infarction (Table 3).[13,14]

Major predictors of cardiogenic shock include older age, tachycardia, hypotension, and high Killip class, which account for more than 95% of the predictive information.[15] Cardiac power (cardiac output times mean arterial pressure) has been found to be the strongest hemodynamic predictor of mortality.[16]

Table 3
Causes of Cardiogenic Shock

- Acute myocardial infarction
  - Left ventricular pump failure
  - Mechanical complications
    - Acute mitral regurgitation
    - Ventricular septal defect
    - Left ventricular free wall rupture
    - Pericardial tamponade
  - Right ventricular infarction
- Other conditions
  - End-stage cardiomyopathy
  - Myocarditis
  - Septic shock with severe myocardial depression
  - Left ventricular outflow tract obstruction
    - Aortic stenosis
    - Hypertrophic obstructive cardiomyopathy
  - Obstruction to left ventricular filling
    - Mitral stenosis
    - Left atrial myxoma
  - Acute mitral regurgitation (chordal rupture)
  - Acute aortic insufficiency
  - Myocardial contusion
  - Prolonged cardiopulmonary bypass

Treatment

Current American College of Cardiology/American Heart Association guidelines recommend early revascularization, either PCI or coronary artery bypass grafting, for patients < 75 years old (class I) or > 75 years old (class IIa) who develop shock within 36 hours of an acute MI.[17] Prompt treatment of hypotension and hypoperfusion is essential. Resuscitative and supportive efforts (ie, adequate oxygenation and ventilation, correction of acid-base and electrolyte abnormalities, relief of pain, restoration of sinus rhythm) should be initiated immediately, at the same time as the diagnostic evaluation.[4] Both pharmacologic and nonpharmacologic modalities can be employed to reverse hypotension.

A growing trend has been to use more aggressive therapeutic interventions early in patients who have cardiogenic shock.[3] Intra-aortic balloon pumping is recommended for patients with cardiogenic shock that is not quickly reversed with pharmacologic therapy (class I).[17] The IABP can stabilize cardiogenic shock patients by increasing diastolic coronary perfusion while decreasing afterload, without an increase in myocardial oxygen consumption. Despite these favorable effects, small randomized trials in the prethrombolytic era failed to show a survival advantage of IABP alone.[4,14,18,19] This is probably because the IABP is not completely passive and requires some level of active left ventricular function.[20]

There are high-risk patients who continue to experience hemodynamic collapse despite maximal pharmacologic therapy, PCI, and IABP. These patients may benefit

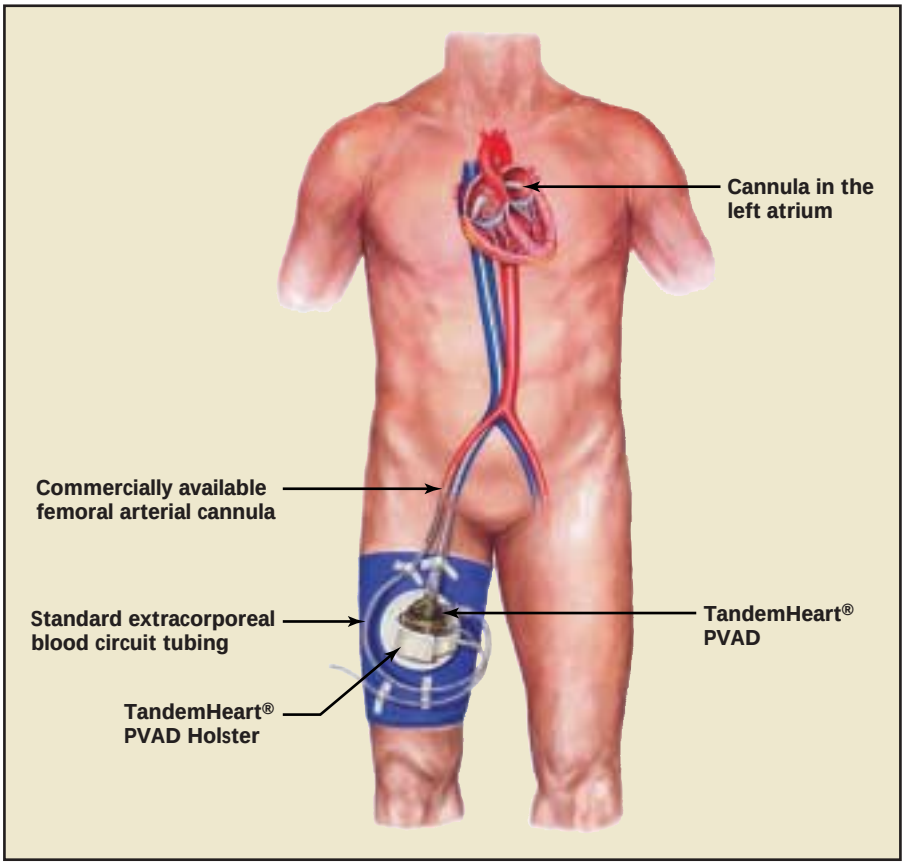

Figure 6. *The TandemHeart® percutaneous left ventricular assist device (PVAD) and associated blood circuit.*

from placement of a PVAD. Recovery of myocardial performance after successful revascularization of the infarct-related vessel may require several days, and during this time, many patients have low cardiac output.[20]

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The PVAD can be placed within 30 minutes and involves the passage of a Brockenbrough catheter into the superior vena cava and puncture of the interatrial septum at the level of the fossa ovalis with a modified Ross needle. A Brockenbrough catheter is then inserted into the left atrium (Figures 5 and 6). The interatrial puncture site is dilated to 21F, followed by insertion of a venous inflow cannula. The circuit is completed by inserting a femoral arterial perfusion catheter (14F-19F) percutaneously. The device can provide up to 4 L/min of assisted cardiac output, which may aid in reversing cardiogenic shock.[20] The main role of the device is the short-term hemodynamic stabilization until recovery of jeopardized myocardium or as a bridge to definite surgical treatment.[20] However, the device has been used prophylactically to support patients undergoing high-risk PCI (eg, in the left main or ostial LAD artery).

Myocardial recovery after revascularization occurs by unloading the left ventricle and diverting blood from the left atrium to the systemic circulation, resulting in reduced left ventricular filling pressures, cardiac workload, and oxygen demand. Thiele and associates[20] noted a significant increase in mean BP (63 ± 8 mm Hg to 80 ± 9 mm Hg, $P < .001$) as well as a reduction in the PCWP (21 ± 4 mm Hg to 14 ± 4 mm Hg, $P < .001$), central venous pressures (13 ± 4 mm Hg to 9 ± 3 mm Hg, $P < .001$), and pulmonary artery pressure (31 ± 8 mm Hg to 23 ± 6 mm Hg, $P < .001$).[20] Contraindications to placement of the TandemHeart device include severe peripheral vascular occlusive disease, moderate to severe aortic insufficiency, bleeding diathesis, and severe sepsis.

Conclusion

Cardiogenic shock continues to be associated with a high mortality rate. High-risk patients who remain in shock despite revascularization, pharmacologic support, and IABP may benefit from placement of a PVAD. The PVAD can provide several days of substantial hemodynamic support, allowing time for recovery of cardiac function, with gradual transition back to native cardiac output and peripheral perfusion. ■

References

1. Zheng ZJ, Croft JB, Giles WH, Mensah GA. Sudden cardiac death in the United States, 1989 to 1998. *Circulation*. 2001;104:2158-2163.
2. Thom TJ, Kannel WB, Silbershatz S, et al. Incidence, prevalence, and mortality of cardiovascular disease in the United States. In:

Alexander W, Schlant R, Fuster V, eds. *Hurst's The Heart*, 9th ed. New York, NY: McGraw Hill; 1998:3.
3. Goldberg RJ, Samad NA, Yarzebski J, et al. Temporal trends in cardiogenic shock complicating acute myocardial infarction. *N Engl J Med*. 1999;340:1162-1168.
4. Califf RM, Bengston JR. Cardiogenic shock. *N Engl J Med*. 1994;330:1724-1730.
5. Domanski MJ, Topol EJ. Cardiogenic shock: current understandings and future research directions. *Am J Cardiol*. 1994;74:724-726.
6. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med*. 1999;341:625-634.
7. Holmes DR, Berger PB, Hochman JS, et al. Cardiogenic shock in patients with acute ischemic syndromes with and without ST-segment elevation. *Circulation*. 1999;100:2067-2073.
8. Hasdai D, Harrington RA, Hochman JS, et al. Platelet glycoprotein iib/iiia blockade and outcome of cardiogenic shock complicating acute coronary syndromes without persistent ST-segment elevation. *J Am Col Cardiol*. 2000; 36:685-692.
9. Harnarayan C, Bennett MA, Pentecost BL, Brewer DB. Quantitative study of infarcted myocardium in cardiogenic shock. *Br Heart J*. 1970;32:728-732.
10. Alonso DR, Scheidt S, Post M, Killip T. Pathophysiology of cardiogenic shock: quantification of myocardial necrosis, clinical, pathologic and electrocardiographic correlations. *Circulation*. 1973;48:588-596.
11. Antman EM and Braunwald E. Acute myocardial infarction. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. 6th ed. Philadelphia, PA: WB Saunders Company; 2001:1178-1179.
12. Antman EM, Hochman JS, Califf RM. Acute myocardial infarction. In: Antman EM, Colucci W, Gotto A, et al, eds. *Cardiovascular Therapeutics: A Companion to Braunwald's Heart Disease*, 2nd ed. Philadelphia, PA: WB Saunders Company; 2002:256.
13. Hochman JS, Boland J, Sleeper LA, et al. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an international registry. *Circulation*. 1995;91:873-881.
14. Hollenberg SM, Kavinsky CJ, Parrillo JE. Cardiogenic shock. *Ann Intern Med*. 1999;131:47-59.
15. Hasdai D, Califf RM, Thompson TD, et al. Predictors of cardiogenic shock after thrombolytic therapy for acute myocardial infarction. *J Am Coll Cardiol*. 2000;35:136-143.
16. Fincke R, Hochman JS, Lowe AM, et al. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. *J Am Coll Cardiol*. 2004;44:340-348.
17. Antman EM, Armstrong PW, Green LA, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary. *J Am Coll Cardiol*. 2004;44:671-719.
18. O'Rourke MF, Norris RM, Campbell TJ, et al. Randomized controlled trial of intraaortic balloon counterpulsation in early myocardial infarction with acute heart failure. *Am J Cardiol*. 1981;47:815-820.
19. Flaherty JT, Becker LC, Weiss JL, et al. Results of a randomized prospective trial of intraaortic balloon counterpulsation and intravenous nitroglycerine in patients with acute myocardial infarction. *J Am Coll Cardiol*. 1985;6:434-446.
20. Thiele H, Laurer B, Hambrecht R, et al. Reversal of cardiogenic shock by percutaneous left-atrial-to-femoral-arterial bypass assistance. *Circulation*. 2001;104:2917-2922.

Main Points

- Despite recent advances in the care of acute coronary disease, cardiogenic shock as a complication of acute myocardial infarction (MI) continues to be associated with a dismal prognosis.
- Cardiogenic shock, as a complication of an acute MI, is a state of inadequate tissue perfusion due to extensive left ventricular injury (typically $\geq$ 40%), resulting in insufficient blood flow to the tissues to meet resting metabolic demands.
- Major predictors of cardiogenic shock include older age, tachycardia, hypotension, and high Killip class, which account for more than 95% of the predictive information.
- Current American College of Cardiology/American Heart Association guidelines recommend early revascularization, either percutaneous coronary intervention (PCI) or coronary artery bypass grafting, for patients $<$ 75 years old (class I) or $>$ 75 years old (class IIa) who develop shock within 36 hours of an acute MI.
- There are high-risk patients who continue to experience hemodynamic collapse despite maximal pharmacologic therapy, PCI, and an intra-aortic balloon pump. These patients may benefit from placement of a percutaneous left ventricular assist device.