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ACUTE MYOCARDIAL INFARCTION

MECHANISM AND TREATMENT OF MYOCARDIAL INFARCTION SHOCK

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Cardiogenic shock complicates one in six patients hospitalized with acute transmural myocardial infarction[1,2]. Whereas effective management of arrhythmias in myocardial infarction has halved the death rate from 30 to 15% in many coronary care units, efforts to overcome cardiogenic shock have been unsatisfactory and the outcome is fatal in more than four out of five patients who develop pump failure of this marked degree [1,2]. Shock now constitutes the major cause of fatality in patients reaching the hospital with infarction and accounts for 100,000 deaths yearly in the United States.

A. **Pathophysiology**: Cardiac Function

It is emphasized that shock following myocardial infarction is fundamentally characterized by profound depression of cardiac performance which leads to lowered cardiac output, diminished arterial blood pressure and inadequate organs perfusion. The basic physiologic defect in this condition is severe decrease of ventricular contractility, principally due to loss of myocardial contractile units. The degree of impairments of cardiac function and contractile state is governed by the extent of the infarction itself and shock occurs when more than 40% of the left ventricular muscle mass is destroyed[1-3].

Ventricular function is diminished in nearly all patients with acute transmural myocardial infarction, including those without complications[4-6]. Thus, there is a spectrum of depression of ventricular performance in transmural infarction: most extreme in cardiogenic shock, intermediate in congestive heart failure without hypotension, and least in uncomplicated infarctions (Fig. 1). In addition to loss of contractile units, cardiac dysfunction results from diminished inotropic state in ischemic areas and from regional ventricular dyssynergy. Further, in some patients, there may be mitral insufficiency due to papillary muscle incompetence and rupture of the interventricular septum. Viewed in terms of the Frank-Starling principle which relates cardiac performance to ventricular filling pressure (Fig. 1), the ventricular function curve is most depressed and flattened in cardiogenic shock. Thus, despite operation of the heart at the apex of this abnormal curve in cardiogenic shock, cardiac output is reduced to such a marked degree that organ perfusion is too low to maintain life.

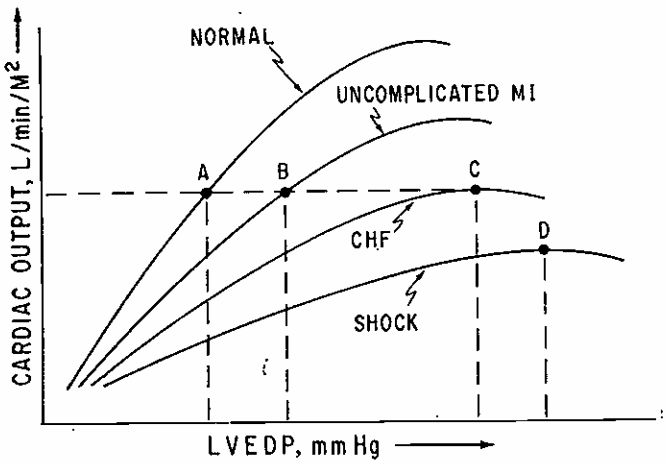

Fig. 1. Ventricular function curves in normal subject, acute uncomplicated transumral myocardial infarction (MI), acute MI with congestive heart failure (CHF) and acute MI with cardiogenic shock. Points A, B and C all represent the same cardiac output, but each is at a different level of left ventricular end diastolic pressure (LVEDP) shown by the vertical broken lines. In shock, despite operation of the left ventricle at the apex of its depressed function curve (point D) with marked elevation of LVEDP, an adequate cardiac output to sustain life cannot be delivered.

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Severe pump failure occurs more commonly with extensive acute anterior myocardial infarction, compared to inferior wall infarction[7,8]. Experimental evidence suggests that the size of the infarction is related to myocardial oxygen requirements at the time of coronary occlusion[9]. Although ventricular compliance may be transiently increased in the first day following infarction[10], compliance is usually diminished throughout the initial five days of the acute episode [10-12]. This increased stiffness of the chamber actually tends to improve the lowered stroke volume from the dysfunctioning ventricle by raising left ventricular end-diastolic pressure and preload and by diminishing ventricular distensibility during ejection[12]. The persistency of an abnormal pattern of segmental contraction following infarction correlates temporally with the presence of pathologic Q waves and the nature and extent of chronic dyssynergy is related to ST-T wave changes[13].

A poor prognosis in myocardial infarction is portended by persistently low cardiac indices and marked elevation of left ventricular end-diastolic pressure, failure to augment stroke volume in response to plasma volume expansion, marked reduction of stroke work index, sustained systemic arterial desaturation, and low urine output [1,2,14,15]. Since right ventricular function is often normal in myocardial infarction, systemic venous pressure may be normal. However, right ventricular performance may be directly impaired in patients with right coronary occlusion and diaphragmatic infarction[16]. Pulmonary blood volume is usually increased, while total blood volume is normal or even reduced following treatment with sympathomimetic drugs. Systemic arterial hypoxemia is due to impaired alveolar-capillary diffusion and venous admixture caused by pulmonary edema. Metabolic acidosis in cardiogenic shock is consequent to marked reduction of cardiac output.

In at least 50% of patients with cardiogenic shock, this condition occurs early within a few hours of the infarction[17]. These patients are usually relatively young, in their third to fifth decade of life, with acute anterior myocardial infarction due to thrombotic occlusion of the left anterior descending coronary artery. Further, they often have accelerated coronary disease and atherosclerosis risk factors. Cardiomegaly may be absent and usually is no more than moderate. Left ventriculography has demonstrated extensive dyskinesis of the anterior free wall and apex, usually greater than 50% of the ventricular silhouette, with increased extent and velocity of shortening of the posterior base of the chamber[18]. Selective coronary arteriography has shown complete obstruction of the proximal left anterior descending coronary artery[18] and pathologic examination has revealed that there is often distal patency of this vessel[17], Although the patency may not be evident on angiography[18].

In approximately one-sixth additional patients in cardiogenic shock, severe mechanical abnormalities are also present such as mitral incompetence, ventricular septal defect or cardiac tamponade[17]. In our experience it is unusual for a patient with diaphragmatic infarction to develop cardiogenic shock based solely on loss of left ventricular muscle; when shock occurs with this location of infarction it is usually due to the added insult to cardiac function of a mechanical disturbance for which a careful search should be carried out.

In the remaining one-third of patients with myocardial infarction shock, this syndrome develops more slowly after a few to several days of severe intractable congestive heart failure following acute infarction[17]. These patients usually have moderate to marked cardiomegaly and chronic symptomatology of ventricular pump dysfunction with previous episodes of myocardial ischemia and infarction. In addition, they usually are older, above 60 years of age, and have diffuse multivessel coronary disease. Pump dysfunction in these individuals can often be considered terminal heart failure due to generalized left ventricular disease. On occasion, however, this subgroup with congestive heart failure and gradual development of shock occurs in younger individuals with localized coronary stenosis in whom the extent of anterior infarction is somewhat less than that which produces acute onset of cardiogenic shock.

B. **Pathophysiology**: Peripheral Circulation

Although failure of cardiac pumping action with consequent decline of cardiac output is the major determinant in the initiation and persistance of myocardial infarction shock, abnormal integrity of circulatory response appears myocardial infarction shock, abnormal integrity of circulatory response appears to play a secondary role in this condition. Thus, in many patients with shock following myocardial infarction, there is incomplete rise of systemic vascular resistance when cardiac output falls which is postulated to result from competitive reflex vasodilation, initiated by stretch and chemoreceptors in the left ventricular wall, opposing carotid and aortic baroreceptor vasoconstrictor activity[19]. Thereby the increase of peripheral vascular resistance is insufficient to maintain blood pressure at a normal level in the presence of low cardiac output.

Supporting this hypothesis of incomplete vascular constriction in myocardial infarction shock are studies carried out in experimental animals [19,20] and in patients[21,22]. In this regard, we have shown arteriolar dilation in the forearm, utilizing plethysmographic techniques, consequent to selective coronary angiography in patients[21]. Further, we have observed impairment of reflex vasoconstriction in the forearm and in total systemic resistance with head-up tilting and the Valsalva maneuver in patients with acute myocardial infarction (Fig. 2)[22].

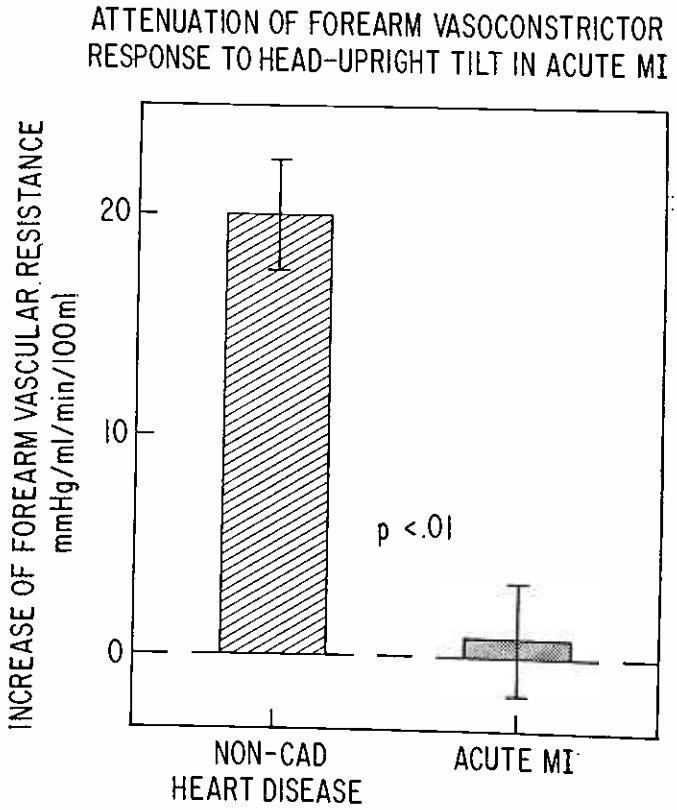

Fig. 2. Reflex forearm vascular resistance responses to head-upright tilting in acute myocardial infarction (MI) compared to hospitalized cardiac patients without coronary artery disease (non-CAD).

C. **Management**: Medical Therapy

At the beginning of consideration of treatment, the physiologic importance of maintaining normal blood pressure in this particular type of cardiogenic shock should be recognized[23]. Thus, in myocardial infarction shock with low coronary arterial perfusion pressure, autoregulation of myocardial blood flow is impaired in the diseased coronary bed and the coronary vessels become dependent on adequate levels of systemic arterial diastolic pressure to maintain coronary blood flow. With very low levels of arterial blood pressure, the critical closing pressure of the coronary vasculature may be reached. Therefore, prolonged hypotension produces death of ischemic but potentially viable myocardium surrounding the infarcted area and results in greater depression of left ventricular function. Thus, there is further decline of cardiac output and more profound lowering of arterial pressure, which leads to additional reduction of coronary blood flow, continued deterioration of cardiac function and ultimately a fatal outcome. Consequently, in myocardial infarction shock, primary therapeutic consideration should be given to maintenance of coronary perfusion pressure to supply adequate flow through the stenotic atherosclerotic vessels to improve contractile state of the ischemic myocardium.

In the treatment of myocardial infarction shock, adverse factors other than loss of myocardial contractile units should be considered and corrected. Thus, arrhythmias producing either excessively rapid or slow heart rate may reduce cardiac output in themselves. Hypoxia due to disturbances of respiratory function and pulmonary venous admixture can contribute to impairment of cardiac function. Excessive pain may be an additive influence in the production of shock. Detrimental iatrogenic factors should be considered such as impairment of cardiorespiratory function provoked by large doses of morphine. When all such contributory factors to cardiogenic shock are eliminated and the severely depressed hemodynamic state persists, therapy with volume expansion and vasoactive agents is indicated.

It has become evident, however, that the most judicious employment of pharmacologic therapy in acute myocardial infarction shock allows successful outcome in no more than 10% of these patients. In our experience, survival is most likely to be achieved in the small proportion of patients who respond quickly with an increase in systemic blood pressure and a fall in the elevated left ventricular end-diastolic pressure. It is emphasized that the very severely depressed ventricular function curve characteristic of myocardial infarction shock is inadequate to sustain life for more than a few hours in the presence of hypotension, or for more than a few days in the presence of severe intractable congestive failure without hypotension.

In the medical management of cardiogenic shock, volume expansion is used first when the left ventricular end-diastolic pressure is below 15 mm.Hg. to elevate ventricular preload, cardiac output and arterial blood pressure without inducing pulmonary congestion[24]. However, the usefulness of this approach is limited since the flatness of the disturbed ventricular function curve does not provide much rise in output with increasing end-diastolic pressure. Further, the end-diastolic pressure is often too high, above 15 mm.Hg, to allow trial of volume expansion.

In the case of severe hypotension (systolic arterial pressure below 75 mm. Hg), blood pressure is increased by administration of the vasoconstrictor-positive inotropic agent norepinephrine[25]. When hypotension is not marked, a positive inotropic drug which also causes relatively mild generalized vasodilation, such as dopamine, is given[25] and, if hypotension is not corrected, pharmocologic therapy is switched to norepinephrine.

In the presence of congestive heart failure without shock, marked elevations of left ventricular end-diastolic pressure above 18 mm.Hg. are reduced to below 15 mm.Hg. by the administration of furosemide[26]. Digitalis is usually ineffective in cardiogenic shock but produces salutary changes in one-half of patients in congestive failure without hypotension[27].

At the present time, new approaches to pharmacologic therapy have not improved survival in myocardial infarction shock, although certain of them have not been thoroughly

tested. These agents include glucagon[28], steroids[29], alpha-adrenergic blockade[30], mannitol[31], glucose and insulin[32], hyaluronidase[33] and allopurinol[34]. Since the fundamental pathologic defect in cardiogenic shock is the extent of loss of ventricular muscle, it is difficult to anticipate that new drugs per se will offer major advances in the future in the management of cardiogenic shock. Still a different mechanism of treatment to be evaluated in myocardial infarction is antithrombosis with urokinase and streptokinase.

D. **Management**: Mechanical Assistance

Several mechanical devices have been developed for the temporary support of the damaged left ventricle after infarction. These devices are designed on the physiologic principle of reducing myocardial oxygen demands and improving coronary blood flow, while providing support of the systemic circulation. With the short-term application of circulatory assistance, time is allowed for the possible recovery of cardiac muscle with the hope that improved ventricular function can be maintained when artificial support is discontinued.

The most practical and beneficial technique is that of counterpulsation in which phasic alterations of aortic pressure are applied synchronously with the cardiac cycle. In this approach, an intraaortic balloon catheter is inserted into the descending thoracic aorta through the femoral artery[35]. Diastolic augmentation of coronary blood flow is achieved by raising ascending aortic diastolic pressure by rapid inflation of the balloon during ventricular relaxation. During subsequent deflation of the balloon during systole, the resistance to left ventricular ejection is reduced and thereby myocardial oxygen requirements are diminished. The counterpulsation method has been extended to non-invasive devices which utilize intermittent external body compression synchronized with the cardiac cycle[36]. Such an apparatus is the lower extremity suit which hydraulically provides phasic positive and negative ambient pressures in diastole and systole respectively[37].

Although hemodynamic improvement attends counterpulsation, this salutary effect is usually temporary and deterioration of pump function often follows discontinuation of assistance. Thus, only a relatively small fraction of patients with myocardial infarction shock unresponsive to pharmacologic management have recovered as a result of application of counterpulsation. Perhaps a more beneficial application of counterpulsation in the future may be in patients with a high risk of developing cardiogenic shock, such as those with acute anterior myocardial infarction even without hypotension or congestive failure, in an effort to save as much ventricular myocardium as possible.

E. **Management**: Cardiac Surgery

In certain patients with refractory cardiogenic shock and severe intractable heart failure, emergency left ventricular angiography and coronary arteriography can be performed with reasonable safety to determine whether acute revascularization by saphenous vein bypass, infarctectomy, mitral valve replacement or repair of a ruptured interventricular septum is likely to be efficacious[38-41]. The extremely high mortality rate of myocardial infarction shock, despite careful pharmacologic management, argues for an aggressive approach in this grave situation particularly in middle-aged and younger patients without a history of heart failure before the acute infarction. Our current policy is to carry out ventriculography and coronary arteriography as soon as it is established that the patient is unresponsive to medical treatment, within an hour or two with cardiogenic shock and between one to three days in the case of marked, refractory congestive failure without hypotension. From the information thereby obtained, surgical intervention with saphenous bypass is recommended as the procedure of choice when satisfactory patency is demonstrated of the distal portion of the proximally obstructed left anterior descending coronary artery. For revascularization to restore myocardial function, success appears to be substantially enhanced when the bypass is carried out within 2-6 hours following infarction and shock.

When the obstructed left anterior descending vessel is not suitable for bypass, acute segmental ventricular resection may be successful as the primary procedure if the segment with abnormal motion, most often the anterior apical portion of the ventricle, is well defined and there is normal or increased extent and velocity of shortening of the remaining myocardium, usually the posterior-diaphragmatic area[41]. Appropriate condidates for this operative intervention may have areas of dyssynergy of greater than 60% of the chamber and extensive resections up to 50% in patients with ventricular dilation have been accomplished with survival.

F. **Conclusions**

The basic physiologic defect in acute myocardial infarction shock is marked depression of ventricular performance which is principally related to the extent of loss of contractile units. Thus, hypotension is primarily the result of reduced cardiac output. In addition, an inadequate rise in systemic vascular resistance contributes secondarily to the diminished blood pressure. This incomplete constriction in the systemic arteriolar beds is postulated to be the result of competitive reflex vasodilation, initiated by stretch and chemoreceptors within the left ventricular wall, which opposes carotid and aortic arch baroreceptor reflex vasoconstrictor activity.

Specific drug therapy is applied in cardiogenic shock after establishment of adequate plasma volume has not reversed the hemodynamic derangement. However, an ideal agent for this purpose is lacking and is unlikely to become available. Judicious medical therapy is successful in about 10% of patients with myocardial infarction shock and the use of mechanical assist devices allows survival in maybe an additional 10%. Emergency coronary and ventricular angiography with coronary bypass revascularization or infarctectomy might prove to be life-saving in perhaps 20% more patients. However, the mortality in this shock syndrome remains distressingly high and fatal in most patients. The major inroads in the management of myocardial infarction shock must await the development of a permanently implantable artificial heart.

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