

Pathophysiology of Ventricular Remodeling after STEMI

Acute Period

- ◆ Cellular edema produces an inflammatory response.
- ◆ Recruitment of some stem cells leads to some tissue regeneration.
- ◆ Damaged tissue is bruised and cyanotic.
- ◆ Catecholamines are released from myocardial cells, thus increasing the risk of arrhythmias. Note: Beta blockers are particularly important in suppressing cardiac arrhythmias in ischemic tissue because they suppress catecholamine release.
- ◆ Cardiac biomarkers are released.
- ◆ White blood cells invade the necrotic tissue within 2 to 3 days.
- ◆ Scavenger cells release enzymes to break down necrotic tissue.
- ◆ The necrotic wall can become very thin during this phase, and the patient is at risk for cardiac rupture. (Gardner & Altman, 2005).

Weeks Following Acute Event

- ◆ A weak collagen matrix forms by second week, but the myocardium is still vulnerable to reinjury.
- ◆ Scar formation has started by third week
- ◆ Necrotic area is completely replaced with scar tissue by week 6 (Gardner & Altman, 2005). Note: Although scar tissue is very strong, it does not contribute to the contractile function of the myocardium.
- ◆ Surviving myocytes hypertrophy in an attempt to compensate for damaged tissue.
- ◆ Excessive non-contractile collagen is present in the newly hypertrophied myocardium, leading to a ventricle that is stiff and noncompliant.

Hemodynamic Alterations after STEMI

When myocardial function is impaired as a result of MI, stroke volume decreases. To compensate for decreased stroke volume, heart rate increases. If myocardial function is impaired to the point that adequate stroke volume cannot be maintained, diastolic filling pressures increase and pulmonary edema results. Tachycardia is a poor prognostic sign in the presence of an acute MI because it is a compensatory mechanism for decreasing stroke volume due to a failing left ventricle.

Hemodynamic alterations depend on the size and location of the infarction. A large MI affecting more than 40% of the myocardium can result in circulatory collapse and cardiogenic shock. The prognosis of patients in cardiac shock remains very poor unless successful revascularization can occur in a timely fashion. Long-term hemodynamic alterations from left ventricular dysfunction result in chronic heart failure. With current reperfusion technology, many patients with MIs are left with no clinical evidence of left-ventricular dysfunction.

A pre-shock state of hypoperfusion with a normal blood pressure may develop before circulatory collapse. Signs and symptoms of hypoperfusion in this pre-shock state include:

- ◆ Cold extremities.
- ◆ Cyanosis.
- ◆ Oliguria.
- ◆ Decreased mentation.

The initial intervention is Dobutamine to improve inotropic function. An intra aortic balloon pump should be placed and if blood pressure is adequate, a pharmacological agent should be used to reduce afterload. Revascularization to reduce ischemia and improve mortality should be attempted.