Table 12-5 Evolution of Morphologic Changes in Myocardial Infarction

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
12-24 hr	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	
1-3 days	Mottling with yellow-tan infarct center	Coagulation necrosis, with loss of nuclei and striations; brisk interstitial infiltrate of neutrophils	
3-7 days	Hyperemic border; central yellow-tan softening	Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	
7-10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; granulation tissue at margins	
10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	

infarction of the right ventricle is unusual (only 1% to 3% of cases), as is infarction of the atria.

The frequencies of involvement of each of the three main arterial trunks and the corresponding sites of myocardial lesions resulting in infarction (in the typical right dominant heart) are as follows (Fig. 12-12*A*):

- Left anterior descending coronary artery (40% to 50%): infarcts involving the anterior wall of left ventricle near the apex; the anterior portion of ventricular septum; and the apex circumferentially
- Right coronary artery (30% to 40%): infarcts involving the inferior/posterior wall of left ventricle; posterior portion of ventricular septum; and the inferior/posterior right ventricular free wall in some cases
- Left circumflex coronary artery (15% to 20%): infarcts involving the lateral wall of left ventricle except at the apex

Other locations of critical coronary arterial lesions causing infarcts are sometimes encountered, such as the left main coronary artery, the secondary (diagonal) branches of the left anterior descending coronary artery, or the marginal branches of the left circumflex coronary artery.

The gross and microscopic appearance of an infarct depends on the duration of survival of the patient following the MI. Areas of damage undergo a progressive sequence of morphologic changes involving typical ischemic coagulative necrosis (the predominant mechanism of cell death in MI, although apoptosis may also occur), followed by inflammation and repair that closely parallels responses to injury in other tissues.

Early morphologic recognition of acute MI can be difficult, particularly when death occurs within a few hours of the onset of symptoms. MIs less than 12 hours old are usually not apparent on gross examination. However, if the infarct preceded death by 2 to 3 hours, it is possible to highlight the area of necrosis by immersion of tissue slices in a solution of **triphenyltetrazolium chloride**. This gross histochemical stain imparts a brick-red color to intact, noninfarcted myocardium where lactate dehydrogenase activity is preserved. Because dehydrogenases leak out through the damaged membranes of dead cells, an infarct appears as an unstained pale zone (Fig. 12-13). By 12 to 24 hours after infarction, an MI can usually be identified grossly as a reddish-blue area of discoloration caused by stagnated, trapped blood. Thereafter, the infarct becomes

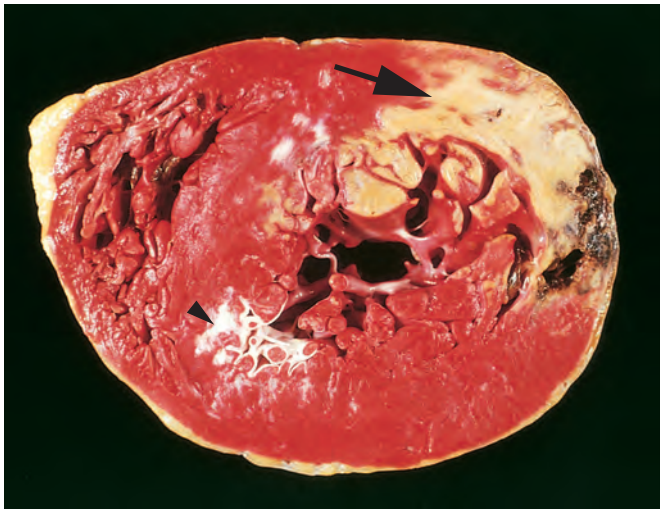

Figure 12-13 Acute myocardial infarct, predominantly of the posterolateral left ventricle, demonstrated histochemically by a lack of staining by triphenyltetrazolium chloride in areas of necrosis *(arrow).* The staining defect is due to the lactate dehydrogenase leakage that follows cell death. Note the myocardial hemorrhage at one edge of the infarct that was associated with cardiac rupture, and the anterior scar *(arrowhead),* indicative of old infarct. Specimen is oriented with the posterior wall at the top.