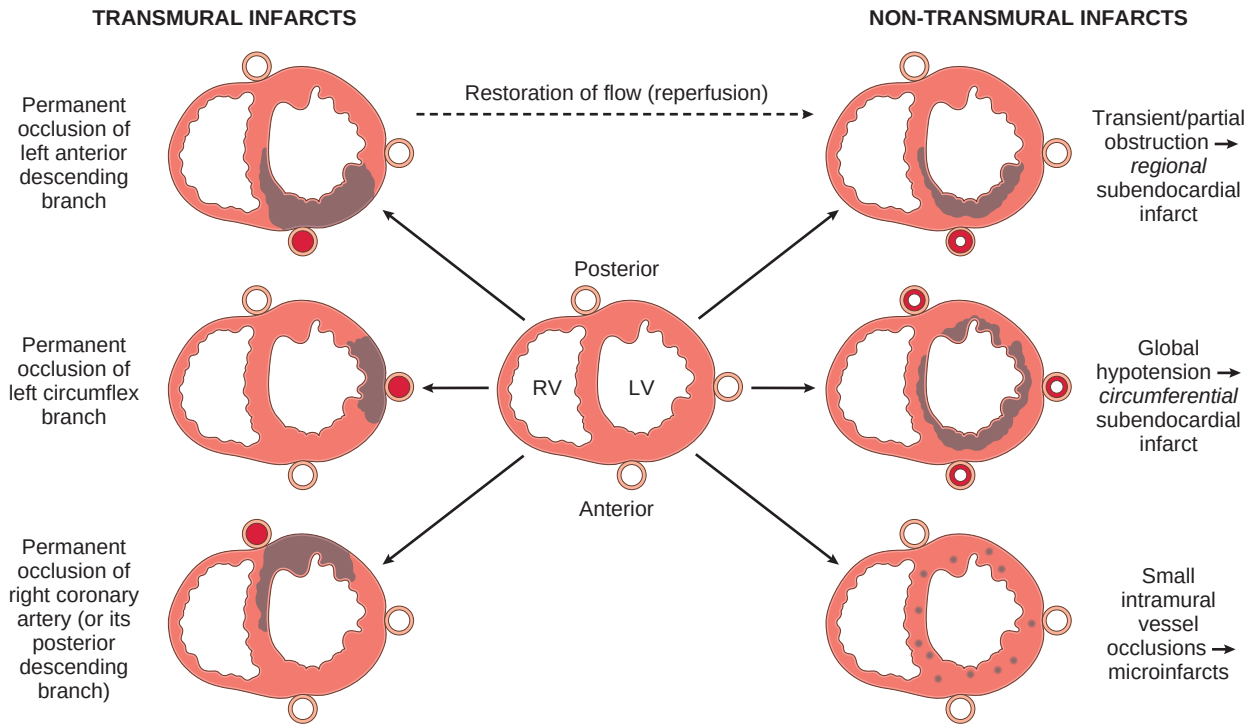

Figure 12-12 Distribution of myocardial ischemic necrosis correlates with the location and nature of decreased perfusion. *Left,* The positions of transmural acute infarcts resulting from occlusions of the major coronary arteries; *top to bottom,* left anterior descending, left circumflex, and right coronary arteries. *Right,* The types of infarcts that result from a partial or transient occlusion, global hypotension, or intramural small vessel occlusions.

Patterns of Infarction. The distribution of myocardial necrosis correlates with the location and cause of the decreased perfusion (Fig. 12-12).

- *Transmural infarction.* Myocardial infarcts caused by occlusion of an epicardial vessel (in the absence of any therapeutic intervention) are typically transmural—the necrosis involves virtually the full thickness of the ventricular wall in the distribution of the affected coronary. This pattern of infarction is usually associated with a combination of chronic coronary atherosclerosis, acute plaque change, and superimposed thrombosis (discussed earlier).
- *Subendocardial (nontransmural) infarction.* As the subendocardial zone is normally the least perfused region of myocardium, this area is most vulnerable to any reduction in coronary flow. A subendocardial infarct—typically involving roughly the inner third of the ventricular wall—can occur as a result of a plaque disruption followed by a coronary thrombus that becomes lysed (therapeutically or spontaneously) before myocardial necrosis extends across the full thickness of the wall. Subendocardial infarcts can also result from prolonged, severe reduction in systemic blood pressure, as in shock superimposed on chronic, otherwise noncritical, coronary stenoses. In the subendocardial infarcts that occur as a result of global hypotension, myocardial damage is usually circumferential, rather than being limited to the distribution of a single major coronary artery.
- *Multifocal microinfarction.* This pattern is seen when there is pathology involving only smaller intramural vessels. This may occur in the setting of microembolization, vasculitis, or vascular spasm, for example, due to endogenous catechols (epinephrine) or drugs (cocaine or ephedrine). Elevated levels of catechols also increase heart rate and myocardial contractility, exacerbating ischemia caused by the vasospasm. The outcome of such vasospasm can be sudden cardiac death (usually caused by a fatal arrhythmia) or an ischemic dilated cardiomyopathy, so-called *takotsubo cardiomyopathy* (also called "broken heart syndrome" because of the association with emotional duress).

Owing to the characteristic electrocardiographic changes resulting from myocardial ischemia or necrosis in various distributions, a transmural infarct is sometimes referred to as an "ST elevation myocardial infarct" (STEMI) and a subendocardial infarct as a "non–ST elevation infarct" (NSTEMI). Depending on the extent and location of the vascular involvement, microinfarctions show nonspecific changes or can even be electrocardiographically silent.

MORPHOLOGY

The temporal evolution of the morphologic changes in acute MI and subsequent healing are summarized in Table 12-5.

Nearly all transmural infarcts involve at least a portion of the left ventricle (comprising the free wall and ventricular septum) and encompass nearly the entire perfusion zone of the occluded coronary artery save for a narrow rim (approximately 0.1 mm) of preserved subendocardial myocardium that is preserved by diffusion of oxygen and nutrients from the ventricular lumen.

Of MIs caused by a right coronary obstruction, 15% to 30% extend from the posterior free wall of the septal portion of the left ventricle into the adjacent right ventricular wall. Isolated