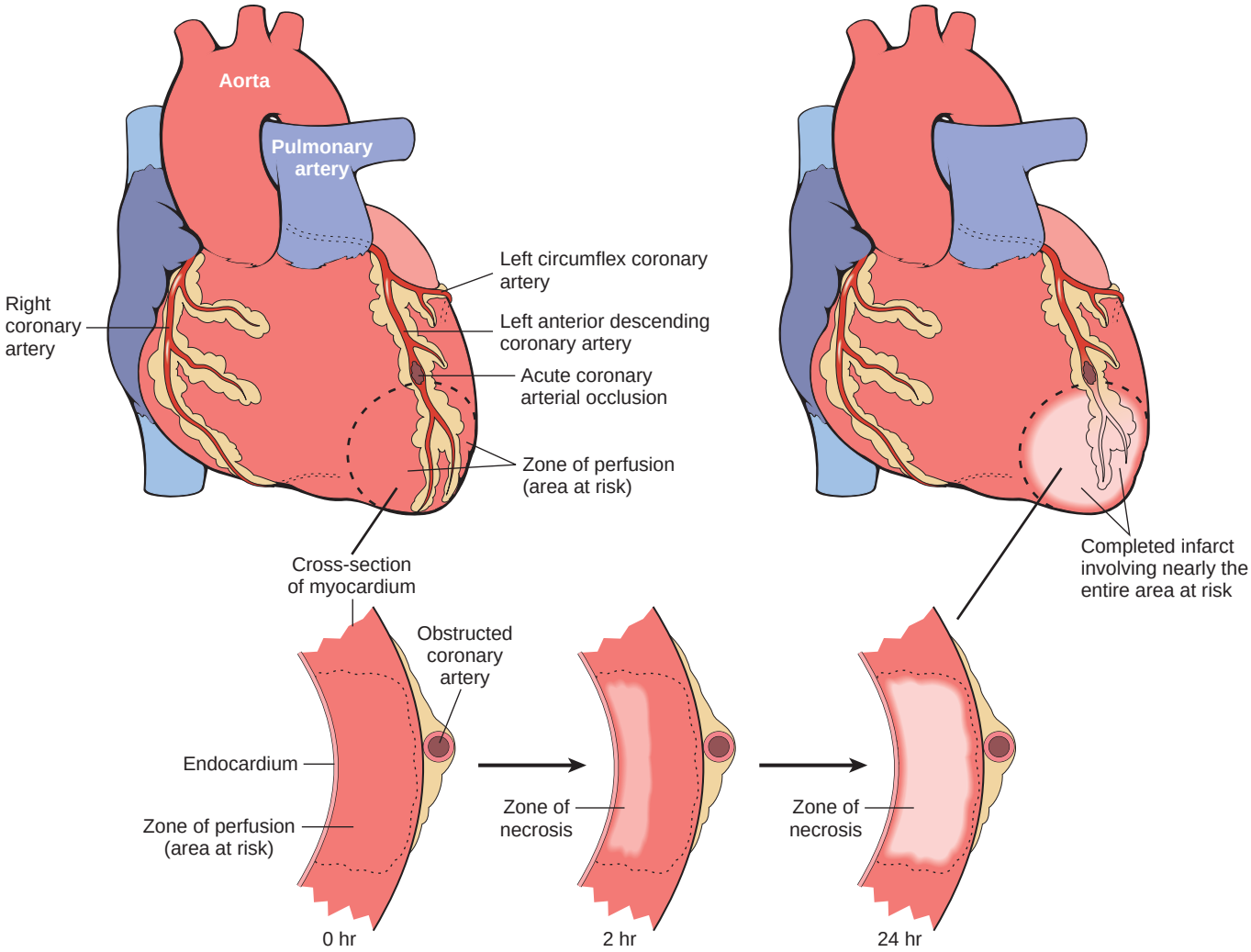

Figure 12-11 Progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone. The area that depends on the occluded vessel for perfusion is the "at risk" myocardium (*shaded*). Note that a very narrow zone of myocardium immediately beneath the endocardium is spared from necrosis because it can be oxygenated by diffusion from the ventricle.

- The extent of collateral blood vessels
- The presence, site, and severity of coronary arterial spasm
- Other factors, such as heart rate, cardiac rhythm, and blood oxygenation

Necrosis involves approximately half of the thickness of the myocardium in 2 to 3 hours of the onset of severe myocardial ischemia, and is usually transmural within 6 hours. However, in instances where chronic sublethal ischemia has induced a more well-developed coronary collateral circulation, the progression of necrosis may follow a more protracted course (12 hours or longer).

Knowledge of the areas of myocardium perfused by the major coronary arteries allows correlation of specific vascular obstructions with their corresponding areas of myocardial infarction. Typically, the LAD branch of the left coronary artery supplies most of the apex of the heart, the anterior wall of the left ventricle, and the anterior two thirds of the ventricular septum. By convention, the coronary artery—either RCA or LCX—that perfuses the posterior third of the septum is called "dominant" (even though the LAD and LCX collectively perfuse the majority of the left ventricular myocardium). In a *right dominant circulation* (present in approximately 80% of individuals), the RCA supplies the entire right ventricular free wall, the posterobasal wall of the left ventricle, and the posterior third of the ventricular septum, while the LCX generally perfuses only the lateral wall of the left ventricle. Thus, RCA occlusions can potentially lead to left ventricular damage. Although most hearts have numerous intercoronary anastomoses (collateral circulation), relatively little blood normally courses through these. However, when a coronary artery is progressively narrowed over time, blood flows via the collaterals from the high- to the low-pressure circulating causing the channels to enlarge. Through such progressive dilation and growth of collaterals, stimulated by ischemia, blood flow is provided to areas of myocardium that would otherwise be deprived of adequate perfusion. Indeed, in the setting of extensive collateralization, the normal epicardial perfusion territories may be so expanded that subsequent occlusion leads to infarction in paradoxical distributions.