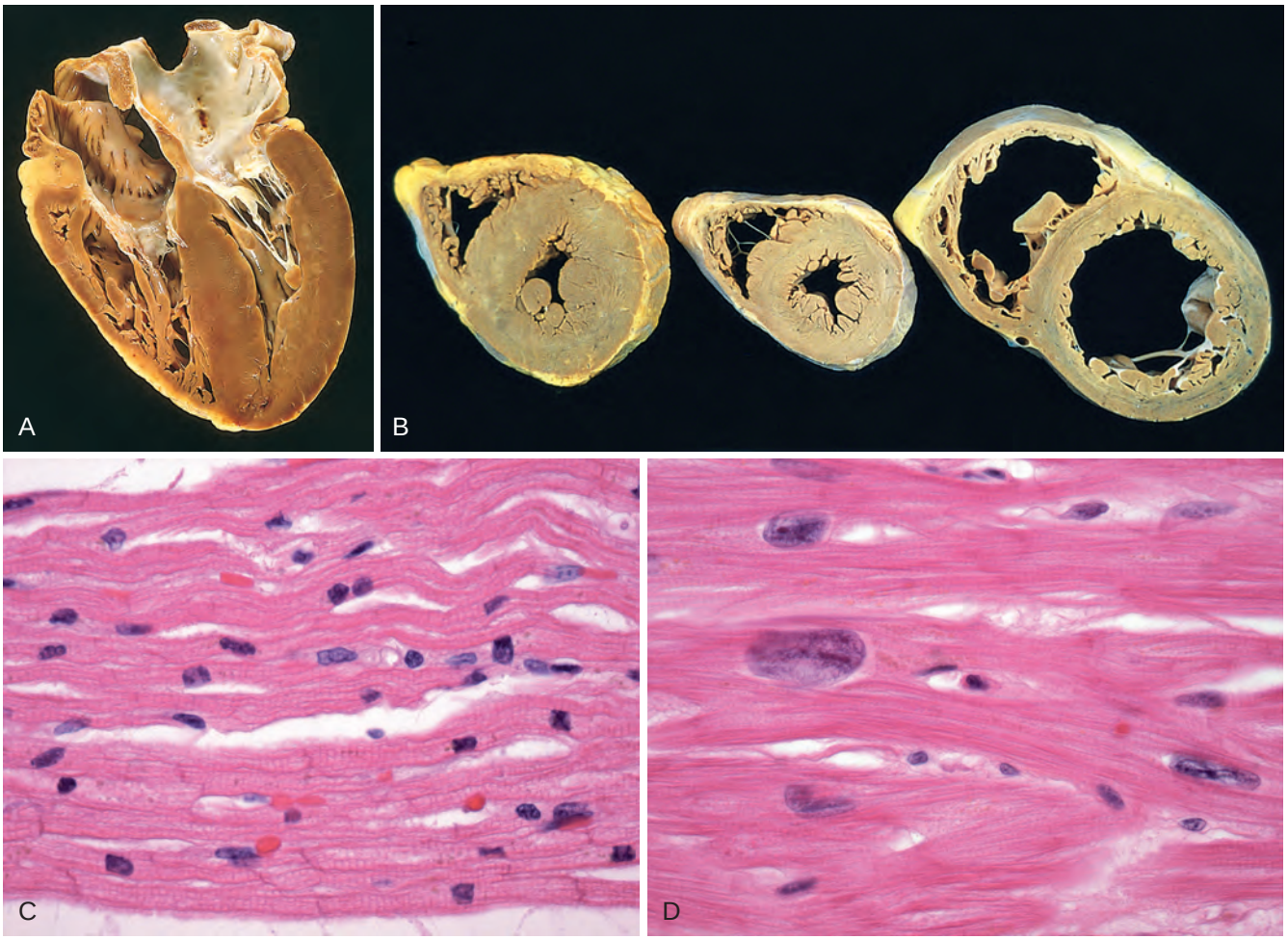

Figure 12-1 Left ventricular hypertrophy. **A,** Pressure hypertrophy due to left ventricular outflow obstruction. The left ventricle is on the lower right in this apical four-chamber view of the heart. **B,** Left ventricular hypertrophy with and without dilation, viewed in transverse heart sections. Compared with a normal heart *(center)*, the pressure-hypertrophied hearts (*left* and in **A**) have increased mass and a thick left ventricular wall, while the hypertrophied, dilated heart *(right)* has increased mass and a normal wall thickness. **C,** Normal myocardium. **D,** Hypertrophied myocardium (panels **C** and **D** are photomicrographs at the same magnification). Note the increases in both cell size and nuclear size in the hypertrophied myocytes. (**A,B,** Reproduced with permission from Edwards WD: Cardiac anatomy and examination of cardiac specimens. In Emmanouilides GC, et al [eds]: Moss and Adams Heart Disease in Infants, Children, and Adolescents: Including the Fetus and Young Adults, 5th ed. Philadelphia, Williams & Wilkins, 1995, p 86.)

At autopsy, the hearts of patients with CHF are generally heavy and dilated, and may be relatively thin-walled; they exhibit microscopic evidence of hypertrophy, but the extent of these changes is extremely variable. In hearts that have suffered myocardial infarction, loss of pumping capacity due to myocyte death leads to work-related hypertrophy of the surrounding viable myocardium. In valvular heart disease, the increased pressure or volume overloads the myocardium globally. Increased heart mass owing to disease is correlated with excess cardiac mortality and morbidity; indeed, cardiomegaly is an independent risk factor for sudden death.

In contrast to pathologic hypertrophy (which is often associated with contractile impairment), hypertrophy induced by regular strenuous exercise has varied effects on the heart depending on the type of exercise. Aerobic exercise (e.g., long distance running) tends to be associated with volume-load hypertrophy that may be accompanied by increases in capillary density (unlike other forms of hypertrophy) and decreases in resting heart rate and blood pressure—effects that are all beneficial. These changes are sometimes referred to as *physiologic hypertrophy*. Static exercise (e.g., weight lifting) is associated with pressure hypertrophy and appears more likely to be associated with deleterious changes.

Whatever its basis, CHF is characterized by variable degrees of decreased cardiac output and tissue perfusion (sometimes called forward failure), as well as pooling of blood in the venous capacitance system (backward failure); the latter may cause pulmonary edema, peripheral edema, or both. As a result, many of the significant clinical features and morphologic changes noted in CHF are actually secondary to injuries induced by hypoxia and congestion in tissues away from the heart.

The cardiovascular system is a closed circuit. Thus, although left-sided and right-sided failure can occur independently, failure of one side (particularly the left) often produces excessive strain on the other, terminating in global heart failure. Despite this interdependence, it is easiest to understand the pathology of heart failure by considering right- and left-sided heart failure separately.