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PART 6 Disorders of the Cardiovascular System FIGURE 249-8 Fractional flow reserve. The fractional flow reserve is measured using a coronary pressure-sensor guidewire that measures the ratio of the pressure in the coronary artery distal to the stenosis (P_d , green) divided by the pressure in the artery proximal to the stenosis (P_a , red) at maximal hyperemia following the injection of adenosine. A fractional flow reserve of <0.80 indicates that revascularization would be beneficial. ■ ■ FURTHER READING Bangalore S et al: Evidence-based practices in the cardiac catheterization laboratory. *Circulation* 144:e107, 2021. Moscucci M (ed): Grossman & Baim's Cardiac Catheterization, Angiography, and Intervention, 9th ed. Philadelphia, Lippincott Williams & Wilkins, 2020. Nishimura R et al: Hemodynamics in the cardiac catheterization laboratory of the 21st century. *Circulation* 125:2138, 2012. Räber L et al: Clinical use of intracoronary imaging. Part 1: guidance and optimization of coronary interventions. An expert consensus document of the European Association of Percutaneous Cardiovascular Interventions. *Eur Heart J* 39:3281, 2018. Samuels BA et al: Comprehensive management of angina with nonobstructive coronary artery disease (ANOCA), Part 1 definition, patient population, and diagnosis. *J Am Coll Cardiol* 82:1245, 2023. Principles of Clinical Cardiac Electrophysiology William H. Sauer, Bruce A. Koplan, Paul C. Zei

HISTORICAL PERSPECTIVE Clinical cardiac electrophysiology is the subspecialty of cardiology that focuses on the study and management of heart rhythm disorders. The development of the modern surface electrocardiogram (ECG) by Willem Einthoven more than 100 years ago enabled an understanding of the relationship between cardiac electrical potentials, mechanical cardiac function, and pathophysiology of cardiac arrhythmias. In the mid-twentieth century, the recording of cellular membrane currents enabled the understanding that the surface ECG represents the sum of cellular cardiac electrical activity. An understanding of cellular electrophysiology also ushered in the development of antiarrhythmic drugs utilized by cardiac electrophysiologists. The modern era of clinical cardiac electrophysiology began with the first recordings of human intracardiac electrograms in the 1960s. Initially, invasive electrophysiology studies were limited to diagnostic tools. This included serial electrophysiologic testing to evaluate arrhythmia mechanisms and evaluate arrhythmia suppression by antiarrhythmic drugs, and programmed stimulation of the ventricle for risk stratification of sudden cardiac death. In the 1960s and 1970s, cardiac surgery was the only available invasive treatment for cardiac arrhythmias. The subsequent development of radiofrequency catheter ablation in the 1980s ushered in the era of interventional cardiac electrophysiology. In addition, with the development of implanted cardiac rhythm management

devices including pacemakers and defibrillators, clinical cardiac electrophysiology became a distinct medical subspecialty. Developments in catheter ablation techniques, cardiac resynchronization and conduction system pacing, subcutaneous defibrillator implantation, leadless pacemaker implantation, left atrial appendage closure, and laser-assisted lead extraction have broadened the procedural aspects of the specialty over the past 30 years; however, the principles of arrhythmia patient management have remained the same.

CELLULAR ELECTROPHYSIOLOGY

The cardiac action potential (AP) drives the electrophysiologic behavior of all cardiac myocytes. The AP is characterized morphologically by five distinct phases, termed phases 0–4, as shown in Fig. 250-1. Moreover, as ventricular electrophysiologic activity accounts for the QRS and T complexes of the surface ECG, each AP phase in ventricular tissues corresponds to distinct phases in the surface ECG: Phase 0, the rapid upstroke, corresponds to the QRS deflection; phases 1–2 account for the ST segment; phase 3 accounts for the T wave; while phase 4 corresponds to the segment between the end of the T wave and the subsequent QRS deflection. In addition, the P wave corresponds to atrial depolarization, while the PR interval corresponds to the time between

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