Cardiac muscle cells contract spontaneously. They do not require motor nerves to shorten. However, the intrinsic contraction rate of these cells is too slow and too unorganized for effective pumping of the heart. Happily, groups of more excitable but noncontractile cardiac cells take responsibility for initiating and conducting electrochemical impulses throughout the cardiac musculature. Such cells cause a coordinated, rhythmic sequence of cardiac muscle contractions that result in blood being moved through the cavities of the heart with appropriate volumes and pressures. These cells constitute the **cardiac conduction system**. Impulses generated at the **sinoatrial (SA) node** are distributed throughout the **atria** and to the **atrioventricular (AV) node** by way of nondiscrete **internodal pathways**. Impulses travel from the AV node, down the **AV bundle** and its **branches**, to the **Purkinje plexus** of cells embedded in the ventricular musculature.

The cardiac conduction system generates voltage changes around the heart. Some of these changes can be monitored, assessed, and measured by **electrocardiography (ECG; aka EKG)**. An ECG is essentially a voltmeter reading. It does not measure hemodynamic changes. Electrodes are placed on a number of body points on the skin. Recorded data (various waves of varying voltage over time) are displayed on an oscilloscope or a strip of moving paper. The shape and direction of wave deflections are dependent upon the spatial relationship of the electrodes (leads) on the body surface.

When the SA node fires, excitation/depolarization of the atrial musculature spreads out from the node. This is reflected in the ECG by an upward deflection of the resting (isoelectric) horizontal line (**P wave**). This deflection immediately precedes contraction of the atrial musculature and filling of the ventricles. The **P-Q interval** (**P-R interval** in the absence of a Q wave) reflects conduction of excitation from the atria to the Purkinje cell plexus in the ventricular myocardium. Prolongation of this interval beyond .20 seconds may reflect an AV conduction block. The **QRS complex** reflects depolarization of the ventricular myocardium. The term *complex* here refers to the combination of the three waves (Q, R, and S) immediately preceding ventricular contraction, wherein blood is forced into the pulmonary trunk and ascending aorta. The **S-T segment** reflects a continuing period of ventricular depolarization. Myocardial ischemia may induce a deflection of this normally horizontal segment. The **T wave** is an upward, prolonged deflection and reflects ventricular repolarization (recovery), during which the atria passively fill with blood from the vena cavae and pulmonary veins. The QT interval, corrected for heart rate (QTc), reflects ventricular depolarization and repolarization. Prolongation of this segment may suggest abnormal ventricular rhythms (arrhythmias). In a healthy heart at a low rate of beat, the P-Q, S-T, and **T-P segments** all are isoelectric (horizontal).