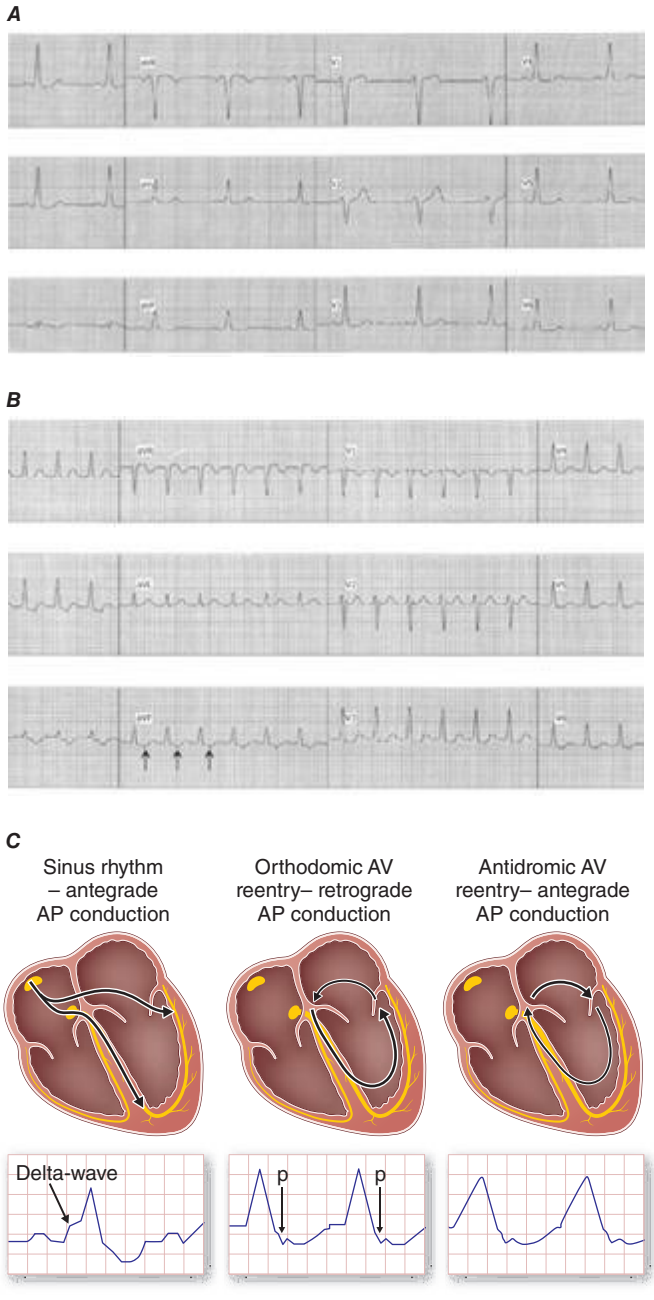

FIGURE 276-6 Wolff-Parkinson-White (WPW) syndrome. ***A.*** A 12-lead electrocardiogram in sinus rhythm (SR) of a patient with WPW demonstrating short P-R interval, delta waves, and widened QRS complex. This patient had an anteroseptal location of the AP. ***B.*** Orthodromic AV reentry in a patient with WPW syndrome using a posteroseptal AP. Note the P waves in the ST segment (*arrows*) seen in lead III and normal appearance of QRS complex. ***C.*** Three most common rhythms associated with WPW syndrome: sinus rhythm demonstrating antegrade conduction over the AP and AV node; orthodromic AVRT using retrograde conduction over the AP and antegrade conduction over the AV node; and antidromic AVRT using retrograde conduction over the AV node and antegrade conduction over the AP. AP, accessory pathway; AV, atrioventricular; AVRT, atrioventricular reentry tachycardia; WPW, Wolff-Parkinson-White.

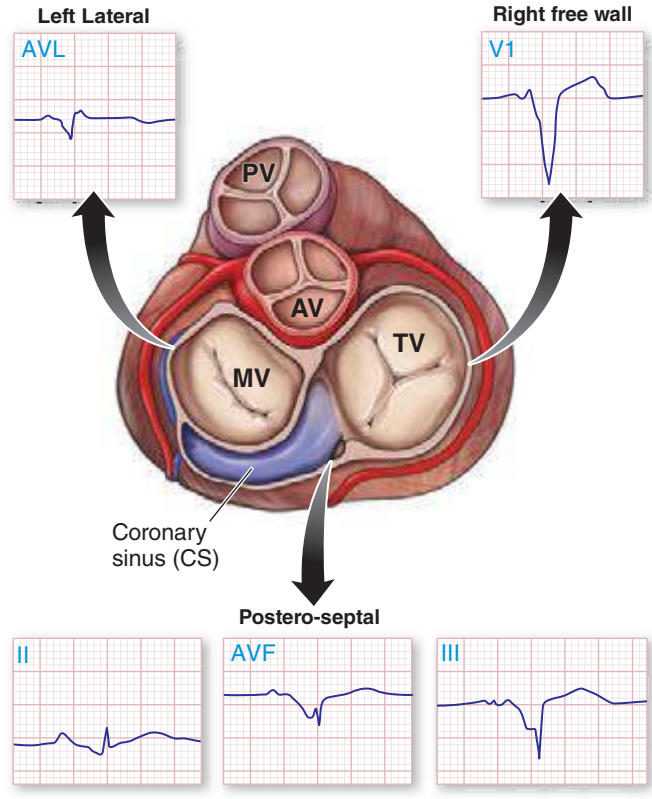

FIGURE 276-7 Potential locations for accessory pathways in patients with Wolff-Parkinson-White Syndrome and typical QRS appearance of delta waves that can mimic underlying structural heart disease such as myocardial infraction of bundle branch block. AV, aortic valve; MV, mitral valve; PV, pulmonary valve; TV, tricuspid valve.

Preexcited Tachycardias Preexcitated tachycardia occurs when the ventricles are activated by antegrade conduction over the AP (Fig. 276-6*C*). The most common is *antidromic AV reentry* in which activation propagates from atrium to ventricle via the AP and then conducts retrogradely to the atria via the His-Purkinje system and the AV node (or rarely a second AP). The wide QRS complex is produced entirely via ventricular excitation over the AP because there is no contribution of ventricular activation over more rapidly conducting specialized His-Purkinje fibers. This tachycardia is often indistinguishable from monomorphic ventricular tachycardia. The presence of preexcitation in sinus rhythm suggests the diagnosis.

Preexcitated tachycardia also occurs if an AP allows antegrade conduction to the ventricles during AT, atrial flutter, atrial fibrillation (Fig. 276-8), or AV nodal reentry. Atrial fibrillation and atrial flutter are potentially life threatening if the AP allows very rapid repetitive conduction. Approximately 25% of APs causing preexcitation allow minimum R-to-R intervals of less than 250 ms during atrial fibrillation are therefore associated with a risk of inducing ventricular fibrillation and sudden death. Preexcited atrial fibrillation presents as a wide-complex, very irregular rhythm. During atrial fibrillation, the ventricular rate is determined by the conduction properties of the AP and AV node. The QRS complex can appear quite bizarre and change on a beat-to-beat basis due to the variability in the degree of fusion from activation over the AV node and AP, or all beats may be due to conduction over the AP (Fig. 276-8). Ventricular activation from the Purkinje system may depolarize the ventricular end of the AP and prevent 1:1 atrial wavefront conduction over the AP. Slowing AV nodal conduction can thereby facilitate AP conduction and dangerously accelerate the ventricular rate. Administration of AV nodal–blocking agents including oral or intravenous verapamil, diltiazem, beta blockers, intravenous adenosine, and intravenous amiodarone are contraindicated. Preexcited tachycardias should be treated with electrical cardioversion or intravenous procainamide or ibutilide, which may terminate or slow the ventricular rate.

Management of Patients with Accessory Pathways Acute management of orthodromic AV reentry is discussed below for PSVT. Patients