the His bundle affecting fibers which will ultimately extend to the left bundle branch. Regardless of the exact anatomical level of clinical bundle branch block, it remains a good clinical rule that most patients exhibiting Mobitz II AV Block will also exhibit a full bundle branch block pattern during periods of conduction between episodes of second degree AV block.

In ambiguous cases, other clues may be helpful. Because infranodal function improves relatively little with exercise, infranodal block tends to worsen with the increasing heart rates associated with exercise or stress. Atropine is not helpful for infranodal block, and because it may accelerate sinus rates, it may cause a patient to progress to higher degrees of AV block with a consequent decrease in the conducted ventricular rate. Exogenous catecholamines such as isoproterenol infusion may be helpful acutely but should not be relied on. Because of its malignant potential, hemodynamically significant Mobitz II AV block should be addressed with early temporary or permanent pacing.

2:1 and High-Degree Atrioventricular Blocks

2:1 AV block is a failure of conduction of every other P wave (see Fig. 9-4D). This pattern is most commonly seen with an infranodal block in the His bundle or bundle branches. However, 2:1 AV block may also be observed in advanced AV nodal disease. It can be distinguished from the more common infranodal form of 2:1 block by the typical Mobitz I periodicity accompanied by a usually narrow QRS complex at other times in the same patient. Because two consecutive conducted P waves are not available to assess the Mobitz pattern, a 2:1 AV block is neither Mobitz I nor Mobitz II.

High-degree AV block is second-degree AV block with conduction failure of two or more consecutive P waves. High-degree AV block is neither Mobitz I nor Mobitz II. Although Mobitz periodicity cannot be assigned, like other forms of second-degree AV blocks, the level of block must be established to assess prognosis and guide therapy. In this case, the ancillary clues described for Mobitz blocks remain useful.

Third-Degree Atrioventricular Block

Third-degree AV block or equivalently complete heart block is a complete failure of AV conduction. In the setting of underlying sinus rhythm, this is an atrial rate faster than the ventricular rate associated with AV dissociation (see Fig. 9-4E). However, when the underlying rhythm is AF, the definition of complete heart block cannot rely on the demonstration of AV dissociation. Because conducted AF always results in an *irregular* ventricular response, the finding of a *regular and slow* ventricular response during AF implies an associated complete heart block.

As is the case for second-degree AV block, the level of the third-degree block determines the clinical behavior and prognosis of complete heart block. Complete heart block at the level of the AV node is associated with a generally stable junctional escape with rates between 40 and 50 beats per minute and usually with a narrow QRS complex. If the patient had a bundle branch block before the development of complete heart block, a block at the level of the AV node is associated with a wide QRS escape, identical to the conducted QRS before the development of a block.

Complete heart block at an infranodal level is associated with a wide and slow ventricular escape rhythm, which often is slower than 40 beats per minute with a QRS different from the antecedent conducted morphology. Unfortunately, infranodal escape rhythms may be absent entirely, leading to asystole and loss of consciousness. When infranodal complete heart block is suspected, regardless of tolerance of the ventricular escape rhythm, prompt institution of temporary or permanent ventricular pacing is appropriate.

TACHYCARDIAS

Overview and Classification

Tachyarrhythmias are categorized as supraventricular and ventricular arrhythmias. SVT relies mechanistically on the atrium or the AV node, or both. During SVT, normal depolarization of the ventricles by the His-Purkinje system produces a narrow complex tachycardia. SVT can manifest as a wide complex tachycardia in the setting of aberrancy or antegrade conduction down an accessory pathway, producing an abnormal sequence of ventricular activation. Ventricular tachyarrhythmias do not depend on the atrium or AV node; they originate in the ventricles, generating a wide complex tachycardia.

Supraventricular Tachycardias

SVTs can be categorized as PSVT, focal atrial tachycardia, atrial flutter and related organized reentrant atrial tachycardias, and AF. This classification scheme, which addresses the underlying arrhythmic mechanism, clinical presentation, and prognosis, guides evaluation and therapy.

PSVT typically manifests in young patients without structural heart disease. The PSVT syndrome is characterized by recurrent tachypalpitations with abrupt onset and offset. Focal atrial tachycardia is more often observed in patients with underlying atrial enlargement and valvular heart disease. AF and atrial flutter are associated with advancing age, hypertension, structural heart disease, diabetes, obstructive sleep apnea, and pulmonary disease. Unlike PSVT, AF carries an increased risk of stroke, heart failure, and death.

Paroxysmal Supraventricular Tachycardia

The incidence of PSVT is 35 cases per 100,000 person-years, with a prevalence of 2.25 per 1000 person-years. Patients report recurrent tachypalpitations. Associated symptoms may include shortness of breath, lightheadedness, chest pain, and syncope. Anginal chest pain and ischemic ST-segment depression are common and related to increased myocardial oxygen demand coupled with the loss of normal diastolic coronary perfusion time. These findings do not necessarily indicate underlying coronary artery disease and typically resolve with tachycardia termination.

PSVT typically occurs independent of structural heart disease and may manifest at any point from infancy to advanced age. PSVT relies on reentry, which is localized in the AV node in approximately 60% of cases and uses a concealed or manifest accessory pathway in 40%. Unless a delta wave indicative of WPW is identified, the underlying mechanism of PSVT is not usually apparent on initial clinical presentation.

An ECG obtained during PSVT can provide useful clues to establish the diagnosis and guide management. The AV relationship should be assessed during tachycardia. By ascertaining the