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256 Paroxysmal Supraventricular Tachycardias

I aVR I II II PART 6 Disorders of the Cardiovascular System III III VI VI III III V5 V5 FIGURE 255-2 Focal atrial tachycardia. In the right panel, a surface 12-lead electrocardiogram shows focal intermittent atrial tachycardia. Note the discrete P waves, with isoelectric segments between, as well as the sinus rhythm. The left panel shows an electroanatomic map of the same focal atrial tachycardia originating from the anterior interatrial septum, as viewed in an anterior-posterior (AP) view of the left atrium obtained during electrophysiology study and ablation. The colors represent the timing of local electrical activation during each tachycardia atrial activation, showing a focal early (red) site. Additional markers of white “flecks” represent conduction direction, demonstrating activation of the atrium dispersing from this focal site. Of note, the pink and red dots represent ablation lesions, in this case, for pulmonary vein isolation. (Adapted from J Brugada et al: 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC). Eur Heart J 41:655, 2020.) every other P wave may fall coincident with the QRS. Maneuvers that increase AV block, such as carotid sinus massage, Valsalva maneuver, or administration of AV nodal–blocking agents, such as adenosine, are useful to create AV block that will expose the P wave. Acute management of sudden-onset, sustained AT is the same as for other forms of PSVT, but the response to pharmacologic therapy is variable, likely depending on the mechanism (Fig. 255-3). For AT due to reentry, administration of adenosine or vagal maneuvers may transiently increase AV block without terminating tachycardia. Some ATs terminate with a sufficient dose of adenosine, consistent with triggered activity as the mechanism. Cardioversion can be effective in some but fails in others because of immediate recur rence, suggesting automaticity as the mechanism in these cases. Beta blockers and calcium channel blockers may slow the ventricular rate by increasing AV block, which can improve tolerance of the arrhythmias, but large doses are sometimes required. Potential precipitating factors Focal atrial tachycardia Hemodynamic instability No Yes Adenosine Cardioversion Ineffective Non-DHP CCB and/or beta blocker Recurrent or incessant Ineffective Ineffective Antiarrhythmic therapy Catheter ablation (see Table 250-2) Recurrent or incessant FIGURE 255-3 Clinical approach and treatment algorithm for management

of focal atrial tachycardia. CCB, calcium channel blocker; DHP, dihydropyridine. (Adapted from J Brugada et al: 2019 ESC Guidelines for the management of patients with supraventricular tachycardia The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC) [published correction appears in Eur Heart J 41:4258, 2020]. Eur Heart J 41:655, 2020.)

aVR V4 V1 aVR aVR V1 V4 V5 V2 aVL aVL V2 V5 V6 V3 aVF aVF V3 V6 and intercurrent illness should be sought and corrected. Underlying heart disease should be considered and excluded. For patients with recurrent episodes, beta blockers, calcium chan nel blockers such as diltiazem or verapamil, and antiarrhythmic drugs such as flecainide, propafenone, disopyramide, sotalol, and amioda rone can be effective, but potential toxicities and adverse effects often warrant avoidance of long-term use. Catheter ablation targeting the AT focus is effective in >80% of patients and is recommended for recurrent symptomatic AT when drugs fail or are not desired or for incessant AT causing tachycardiainduced cardiomyopathy. Although AT is often a precursor to atrial fibrillation or atrial flutter, the associated risk for stroke and, hence, indications for long-term anticoagulation are unclear but not consid ered equivalent. ■ ■FURTHER READING Brugada J et al: 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The Task Force for the Manage ment of Patients with Supraventricular Tachycardia of the European Society of Cardiology (ESC) developed in collaboration with the Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 41:655, 2020. Callans DJ: Josephson’s Clinical Cardiac Electrophysiology: Techniques and Interpretations, 7th ed. Philadelphia, Wolters Kluwer, 2024. William H. Sauer, Paul C. Zei

Paroxysmal

Supraventricular

Tachycardias In this chapter, sustained supraventricular tachycardias (SVTs) dependent on the atrioventricular (AV) node are discussed. These include AV nodal reentry tachycardia (AVNRT), junctional tachycar dia, AV reciprocating tachycardia (AVRT) utilizing an accessory path way, and a group of additional various SVTs that involve an accessory

II P waves V1 A B FIGURE 256-1 Atrioventricular (AV) node reentry. A. Leads II and V1 are shown. P waves are visible at the end of the QRS complex and are negative in lead II and may give the impression of S waves in the inferior limb leads II, III, and aVF and an R′ in lead V1. B. Stylized version of the AV nodal reentry circuit within the triangle of Koch (see Fig. 254-1) that involves AV node and its extensions along with perinodal atrial tissue. CS, coronary sinus. pathway, termed preexcited tachycardias. The term SVT encompasses a broad group of tachyarrhythmias based on anatomic origin and techni cally includes sinus tachycardia, atrial tachycardia (AT), atrial flutter, and atrial fibrillation; however, for the purposes of describing an orga nized approach to diagnosis and treatment of SVT, a separate discus sion for these non-AV nodal–dependent SVTs are discussed elsewhere. ATRIOVENTRICULAR NODAL REENTRY TACHYCARDIA AVNRT is the most common form of paroxysmal supraventricular tachycardia (PSVT), representing ~60% of cases referred for catheter ablation. It most commonly manifests in the second to fourth decades of life, more often in women. It is usually well tolerated, but rapid tachycardia, particularly in the elderly, may cause angina, pulmonary edema, hypotension, or syncope. It is not usually associated with

structural heart disease. In patients without associated heart disease, AVNRT is not a life-threatening arrhythmia; however, it may cause significant symptoms. The mechanism is reentry involving the AV node and the perinodal atrium, made possible by the existence of multiple pathways for con duction from the atrium into the AV node that are capable of conduc tion in two directions (Fig. 256-1). Most forms of AVNRT utilize a slowly conducting AV nodal path way (right inferior extension) that extends from the compact AV node near the His bundle, inferiorly along the tricuspid valve annulus to the floor of the coronary sinus. The reentry wavefront propagates up this slowly conducting pathway to the compact AV node and then exits from the fast pathway at the top of the AV node. The path back to the I aVR V1 V4 II aVL V2 V5 III aVF V3 V6 V1 FIGURE 256-2 Atrioventricular nodal reentry tachycardia with retrograde P waves before and after adenosine termination.

CHAPTER 256 Inferior AV node extension: Slow pathway Compact AV node: Fast pathway Paroxysmal Supraventricular Tachycardias
CS Tricuspid valve slow pathway probably involves the left atrial septum, which has con nections to the coronary sinus musculature. More unusual forms of AVNRT utilize a left inferior extension that connects to the compact AV node through the roof of the coronary sinus or, in extremely rare cases, directly from the mitral valve annulus avoiding the coronary sinus musculature altogether. In typical forms, the conduction time from the compact AV node region to the atrium is similar to that from the compact node to the His bundle and ventricles, such that atrial activation occurs at about the same time as ventricular activation. The P wave is therefore inscribed during, slightly before, or slightly after the QRS and can be difficult to discern. Often the P wave is seen at the end of the QRS complex as a pseudo-r′ in lead V1 and pseudo-S waves in leads II, III, and aVF (Fig. 256-2). More unusual forms of AVNRT have P waves falling later, any where between QRS complexes, in which case, an inverted P wave is seen in the inferior limb leads with the inverted P wave seen in the subsequent T wave. The rate can vary with sympathetic tone through its effect on the conduction time of AV nodal tissues. Simultaneous atrial and ventricular contraction results in atrial contraction against a closed tricuspid valve, producing a cannon A wave visible in the jugular venous pulse often perceived as a fluttering sensation in the neck. Elevated venous pressures may also lead to release of natriuretic peptides that cause post-tachycardia diuresis. In contrast to ATs, maneuvers or medications that produce AV nodal block terminate the arrhythmia. Acute treatment is the same as for other forms of PSVT (discussed below). Whether ongoing therapy is warranted depends on the severity of symptoms and frequency of episodes. Reassurance and instruction as to how to perform the Valsalva maneuver or other vagal I V4 V1 aVR V5 V2 aVL II V6 V3 aVF III V1

nerve–stimulating maneuvers to terminate episodes are sufficient for many patients. Administration of an oral beta blocker, verapamil, or diltiazem at the onset of an episode can be used to facilitate termina tion. Chronic therapy with these medications or flecainide is an option if prophylactic therapy is needed. Catheter ablation of the slow AV nodal pathway is recommended for patients with recurrent or severe episodes or when drug therapy is ineffective, not tolerated, or not desired by the patient. Catheter ablation is curative in >95% of patients. The major risk is AV block requiring permanent pacemaker implanta tion, which occurs in <1% of patients.

PART 6 Disorders of the Cardiovascular System JUNCTIONAL TACHYCARDIA Junctional ectopic tachycardia (JET) is due to automaticity within the AV node. It is rare in adults and more frequently encountered as an incessant tachycardia in children, often in the perioperative period of surgery

for congenital heart disease. It presents as a narrow QRS tachycardia, often with ventriculoatrial (VA) block, such that AV dis sociation is present. JET can occur as a manifestation of increased adrenergic tone and may be seen after administration of isoproterenol, particularly after catheter ablation in the perinodal region. It may also occur for a short period of time after ablation for AVNRT. Accelerated junctional rhythm is a junctional automatic rhythm between 50 and 100 beats/min. Initiation may occur with gradual acceleration in rate, suggesting an automatic focus, or after a premature ventricular con traction, suggesting a focus of triggered automaticity. VA conduction is usually present, with P-wave morphology and timing such that it resembles AVNRT at a slow rate. It can be related to increased sympa thetic tone and may produce palpitations. It usually does not require specific therapy. ACCESSORY PATHWAYS AND THE

WOLFF-PARKINSON-WHITE SYNDROME Accessory pathways (APs) occur in 1 in 1500–2000 people and are associated with a variety of arrhythmias including narrow-complex PSVT, wide-complex tachycardias, and, rarely, sudden death. Most patients have structurally normal hearts, but APs are associated with Ebstein's anomaly of the tricuspid valve and forms of hypertrophic cardiomyopathy including PRKAG2 mutations, Danon's disease, and Fabry's disease (Fig. 256-3). APs are abnormal connections that allow conduction between the atrium and ventricles across the AV ring. They are present from birth and are due to failure of complete partitioning of atrium and ventricle by the fibrous AV rings. They occur across either an AV valve annulus or the septum, most frequently between the left atrium and free wall of the left ventricle, followed by posteroseptal, right free wall, and antero septal locations. If the impulse from the sinus node conducts through the AP to the ventricle (antegrade) before the impulse conducts through the AV node and His bundle, then the ventricles are preexcited during sinus rhythm, and the electrocardiogram (ECG) shows a short P-R interval (<0.12 s), slurred initial portion of the QRS (delta wave), and prolonged QRS duration produced by slow conduction through direct activation of ventricular myocardium over the AP. The mor phology of the QRS and delta wave is determined by the AP location and associated site of earliest ventricular activation, and the degree of fusion between the excitation wavefronts from conduction over the AV node and conduction over the AP (Fig. 256-4). Right-sided pathways preexcite the right ventricle, producing a left bundle branch block–like configuration in lead V1, and often create marked preexcitation because of their relatively close proximity of the AP to the sinus node (Fig. 256-4). Left-sided pathways preexcite the left ventricle and may produce a right bundle branch–like configuration in lead V1 and a negative delta wave in aVL, indicating initial depolariza tion of the lateral portion of the left ventricle that can mimic Q waves of lateral wall infarction (Fig. 256-4). Because of the relatively large distance between the sinus node and left free wall APs, preexcitation may be minimal or absent on 12-lead ECG. Preexcitation due to an AP at the diaphragmatic surface of the heart, typically in the paraseptal region, produces delta waves that are negative in leads III and aVF,

A B Sinus rhythm— antegrade AP conduction Orthodomic AV reentry—retrograde AP conduction Antidromic AV reentry—antegrade AP conduction p p Delta-wave C FIGURE 256-3 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 accessory pathway (AP). B. Orthodromic atrioventricular (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 AV reentry tachycardia (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. mimicking the Q waves of inferior wall infarction (Fig. 256-4). Preexci tation can be intermittent and disappear during exercise as conduction over the AV node accelerates and may take over ventricular activation completely. Wolff-Parkinson-White (WPW) syndrome is defined as a preexcited QRS during sinus rhythm and episodes of PSVT. There are a number of variations of APs that may not cause preexcitation and/or arrhythmias. Concealed APs allow only retrograde conduction, from ventricle to atrium, so no preexcitation is present during sinus rhythm, but SVT

Left lateral Right free wall aVL V1 PV PV AV TV MV Coronary sinus (CS) Postero septal II aVF III FIGURE 256-4 Potential locations for accessory pathways in patients with WolffParkinson-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. can occur. Other unusual forms of APs exist. Fasciculoventricular connections between the His bundle and ventricular septum produce preexcitation but do not cause arrhythmia, probably because the circuit is too short to promote reentry. Atriofascicular pathways, also known as Mahaim fibers, probably represent a duplicate AV node and HisPurkinje system that connect the right atrium to fascicles of the right bundle branch and produce a wide-complex tachycardia having a left bundle branch block configuration. ATRIOVENTRICULAR RECIPROCATING TACHYCARDIA The most common tachycardia caused by an AP is the PSVT desig nated orthodromic AV reciprocating tachycardia. The circulating reentry wavefront propagates from the atrium anterogradely over the AV node and His-Purkinje system to the ventricles and then reenters the atria via retrograde conduction over the AP. The QRS is narrow or may have typical right or left bundle branch block, but without preexcita tion during tachycardia. Because excitation through the AV node and AP are necessary, AV or VA block results in tachycardia termination. During sinus rhythm, preexcitation is seen if the pathway also allows anterograde conduction. Most commonly, during tachycardia, the R-P interval is shorter than the P-R interval and can resemble AVNRT. Unlike typical AVNRT, P waves always follow the QRS and are never simultaneous with a narrow QRS complex because the ventricles must be activated before the reentry wavefront reaches the AP and conducts back to the atrium. The morphology of the P wave during tachycardia is determined by the pathway location, but it can be difficult to assess because it is usually inscribed during the ST segment. The P wave in posteroseptal APs is negative in leads II, III, and aVF, similar to that in AV nodal reentry, but P-wave morphology will differ from AV nodal reentry for pathways in other locations. Occasionally, an AP conducts extremely slowly in the retrograde direction, resulting in tachycardia with a long R-P interval, similar to most ATs. These pathways are

usually located in the septal region and have negative P waves in leads II, III, and aVF. Slow AP conduction facilitates reentry, often leading to nearly incessant tachycardia, known as permanent junctional recip rocating tachycardia (PJRT). Tachycardia-induced cardiomyopathy can occur. Without an invasive electrophysiology study, it may be difficult to distinguish this form of orthodromic AV reentry from atypical AV nodal reentry or AT.

CHAPTER 256 PREEXCITED TACHYCARDIAS Preexcited tachycardia occurs when the ventricles are activated by antegrade conduction over the AP. The most common mechanism is antidromic AV

reciprocating tachycardia 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 sec ond 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. Paroxysmal Supraventricular Tachycardias

Preexcited tachycardia also occurs if an AP allows antegrade con duction to the ventricles during AT, atrial flutter, atrial fibrillation (AF), or AV nodal reentry, otherwise known as bystander AP conduc tion. AF and atrial flutter are potentially life-threatening if the AP allows very rapid repetitive conduction (Fig. 256-5). Approximately 25% of APs causing preexcitation allow minimum R-to-R intervals of <250 ms during AF and are associated with a higher risk of inducing ventricular fibrillation and sudden death. Preexcited AF presents as a wide-complex, very irregular rhythm. During AF, 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. Ventricular activation from the Purkinje system may depolarize the ventricular aspect of the AP and prevent atrial wavefront conduction over the AP. Slowing AV nodal conduction without slowing AP 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, is contraindicated during preexcited AF. Rapid preexcited tachycardia should be treated with electrical cardioversion or intrave nous procainamide or ibutilide, which may terminate the arrhythmia or slow the ventricular rate. MANAGEMENT OF PATIENTS WITH ACCESSORY PATHWAYS Acute management of orthodromic AV reentry is discussed below for PSVT. Patients with WPW syndrome may have wide-complex tachy cardia due to antidromic AV reentry, orthodromic AV with bundle branch block, or a preexcited tachycardia, and treatment depends on the underlying rhythm. Initial patient evaluation should include assess ment for aggravating factors, including intercurrent illness and factors that increase sympathetic tone. Examination should focus on excluding underlying heart disease. An echocardiogram is reasonable to exclude Ebstein’s anomaly, forms of hypertrophic cardiomyopathy that can be associated with APs, or tachycardia-mediated cardiomyopathy. Patients with preexcitation who have symptoms of arrhythmia are at risk for developing AF and sudden death if they have an AP that allows rapid antegrade conduction. The risk of cardiac arrest is in the range of 2 per 1000 patients in adults but is likely greater in children. An invasive electrophysiology study is recommended to assess whether the pathway can support dangerously rapid heart rates if AF were to occur, and it is usually combined with potentially curative catheter ablation. Catheter ablation is warranted for recurrent arrhythmias when drugs are ineffective, not tolerated, or not desired by the patient. Efficacy is in the range of 95% depending on the location of the AP. Serious complications occur in <3% of patients but can include AV

PART 6 Disorders of the Cardiovascular System I I aVR aVR II aVL II aVL III aVF III aVF V1 V1 II II V5 V5 25mm/s 10mm/mV 150Hz 9.0.9 12SL 243 CID: 0 FIGURE 256-5 Preexcited atrial fibrillation (AF) due to conduction over a left free wall accessory pathway (AP). The electrocardiogram shows rapid irregular QRS complexes that represent fusion between conduction over the atrioventricular node and left free wall AP. Shortest R-R intervals between preexcited QRS complexes of <250 ms, as in

this case, indicate a risk of sudden death with this arrhythmia. block, cardiac tamponade, thromboembolism, coronary artery injury, and vascular access complications. The risk of AV block is higher when the AP is located near the AV node and/or His bundle, in the so-called anteroseptal or mid-septal locations. Procedure mortality is <1 in 1000 patients. Ambulatory monitoring or exercise testing is often used to gain reassurance that the AP is not high risk, evaluating for abrupt loss of conduction (preexcitation) at physiologic heart rates consistent with a low-risk pathway, but this is not completely reliable. Gradual loss of AP conduction with increased sympathetic tone does not reliably indicate low risk since this can occur as AV nodal conduction time shortens, and therefore, the possibility of rapid antegrade AP conduc tion is not excluded definitively. For patients with concealed APs or known low-risk APs causing orthodromic AVRT, chronic therapy is guided by symptoms and fre quency of events. Vagal maneuvers may terminate episodes, as may a dose of beta blocker, verapamil, or diltiazem taken at the onset of an episode. Chronic therapy with these agents or flecainide can reduce the frequency of episodes in some patients. Adults who have preexcitation but no arrhythmia symptoms have a risk of sudden death estimated to be 1 per 1000 patient-years. Electro physiology study is usually advised for people in occupations for which an arrhythmia occurrence would place them or others at risk, such as police, military, and pilots, or for individuals who desire evaluation for risk. Routine follow-up without therapy is reasonable in others. Children are at greater risk of sudden death, ~2 per 1000 patient-years.

V1 V4 V1 V4 V2 V5 V2 V5 V3 V6 V3 V6 TREATMENT Paroxysmal Supraventricular Tachycardia Acute management of narrow QRS PSVT is guided by the clinical presentation. Continuous ECG monitoring should be implemented, and a 12-lead ECG should always be obtained when possible, since this may be useful in determining the mechanism. In the presence of hypotension with unconsciousness or respiratory distress, QRSsynchronous direct current cardioversion is warranted, but this is rarely needed, because intravenous adenosine works promptly in most situations (see below). For stable individuals, initial therapy takes advantage of the fact that most PSVTs are dependent on AV nodal conduction (AV nodal reentry or orthodromic AV reentry) and, therefore, likely to respond to sympatholytic and vagotonic maneuvers and drugs. As these are administered, the ECG should be continuously recorded because the response can establish the diagnosis. AV block with only transient slowing of tachycardia may expose ongoing P waves, indicating AT or atrial flutter as the mechanism (Fig. 256-6). Carotid sinus massage is reasonable provided the risk of carotid vascular disease is low, as indicated by absence of carotid bruits and no prior history of stroke. A Valsalva maneuver should be attempted in cooperative individuals, and if effective, the patient can be taught to perform this maneuver as needed. If vagal maneuvers fail or

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