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■ ■FURTHER READING Jalife J, Stevenson W (eds): Zipes and Jalife’s Cardiac Electrophysiology:

From Cell to Bedside, 8th ed. Philadelphia, Elsevier, 2022. Kusumoto FM et al: 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduc tion delay: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Heart Rhythm 16:e128, 2019. Sidhu S, Marine JE: Evaluating and managing bradycardia. Trends PART 6 Disorders of the Cardiovascular System Cardiovasc Med 30:265, 2020. William H. Sauer, Paul C. Zei

Approach to

Supraventricular

Arrhythmias Supraventricular arrhythmias refer to all abnormal heart rhythms originating above the level of the ventricle including the atria and the atrioventricular (AV) junction. Supraventricular arrhythmias include premature atrial contractions (PACs), wandering atrial pacemaker, sinus arrhythmia, nonphysiologic sinus tachycardia, atrial tachycardia, junctional tachycardia, atrial flutter, and atrial fibrillation. Supraven tricular arrhythmias that result in an elevated heart rate (>100 beats/ min) are broadly defined as supraventricular tachycardias (SVTs). SVTs originate from or are dependent on conduction through the atrium or AV node to the ventricles. Most produce narrow QRS complex tachycardia (QRS duration <120 ms) characteristic of ventricular activation over the Purkinje system and, thus, are sometimes referred to as a narrow-complex tachycardias. The QRS morphology of the SVT is usually identical to the sinus rhythm QRS. Conduction block in the left or right bundle branch or activation of the ventricles from an accessory pathway produces a wide QRS complex during SVT that must be distinguished from ventricular tachycardia (VT). Mechanisms of supraventricular tachyarrhythmia can be divided into physiologic sinus tachycardia and pathologic tachycardia (Table 253-1). Pathologic tachycardia can be further subclassified by

mechanism as reentrant arrhythmias dependent on AV nodal conduction (e.g., AV nodal reentry tachycardia), large reentry circuits within the atrial tissue alone (e.g., atrial flutter), or focal atrial tachycardias that can be due to automaticity or small reentry circuits. The prognosis and treatment vary considerably depending on the mechanism and underlying heart disease. SVT can be of brief duration, termed nonsustained, or can be sustained such that an intervention, such as cardioversion, catheter ablation, or drug administration, is required for termination and main tenance of sinus rhythm. Episodes that occur with sudden onset and termination are referred to as paroxysmal. Paroxysmal supraventricular tachycardia (PSVT) refers to a family of tachycardias including AV node reentry, AV reciprocating tachycardia using an accessory pathway, and atrial tachycardia described in subsequent chapters (Fig. 253-1). Other supraventricular arrhythmias that may or may not be symp tomatic include PACs, sinus arrhythmia, ectopic atrial rhythm, and accelerated junctional rhythm. Atrial flutter and atrial fibrillation may present as a tachycardia or may be adequately rate controlled with or without AV nodal blocking agents; these entities are discussed sepa rately (see Chaps. 255, 256, 257, and 258). CLINICAL PRESENTATION Symptoms of supraventricular arrhythmia vary depending on the rate, duration, associated heart disease, and comorbidities and include palpitations, chest pain, dyspnea, diminished exertional capacity, and

TABLE 253-1 Mechanisms of Supraventricular Arrhythmias Physiologic Sinus Tachycardia Defining feature: normal sinus mechanism precipitated by exertion, stress, exogenous or endogenous stimulants, concurrent illness Pathologic Supraventricular Tachycardia (SVT) A. Tachycardias originating from the atrium Defining feature: tachycardia may continue despite beats that fail to conduct to the ventricles, indicating that the atrioventricular (AV) node is not participating in the tachycardia circuit 1. Inappropriate sinus tachycardia Defining feature: tachycardia from the normal sinus node area that occurs without an identifiable precipitating factor as a result of dysfunctional autonomic regulation 2. Focal atrial tachycardia (AT) Defining feature: regular atrial tachycardia with defined P wave; may be sustained, nonsustained, paroxysmal, or incessant; frequent sites of origin occur along the valve annuli of left or right atrium, pulmonary veins, coronary sinus musculature, superior vena cava 3. Atrial flutter and macroreentrant atrial tachycardia Defining feature: macroreentry reflected as organized atrial activity on an electrocardiogram (ECG), commonly seen as sawtooth flutter waves at rates typically faster than 200 beats/min 4. Atrial fibrillation Defining feature: chaotic rapid atrial electrical activity with variable ventricular rate; the most common sustained cardiac arrhythmia in older adults 5. Multifocal atrial tachycardia Defining feature: multiple discrete P waves often seen in patients with pulmonary disease during acute exacerbations of pulmonary insufficiency B. AV nodal reentry tachycardia (AVNRT) Defining feature: paroxysmal regular tachycardia with P waves visible at the end of the QRS complex or not visible at all; the most common paroxysmal sustained tachycardia in healthy young adults; more common in women C. Tachycardias associated with accessory atrioventricular pathways 1. Orthodromic AV reciprocating tachycardia (AVRT) Defining feature: paroxysmal sustained tachycardia similar to AV nodal reentry; during sinus rhythm, evidence of ventricular preexcitation may be present (Wolff-Parkinson-White syndrome) or absent (concealed accessory pathway) 2. Preexcited tachycardia Defining feature: wide QRS tachycardia with QRS morphology similar to ventricular tachycardia a. Antidromic AV reciprocating tachycardia—regular paroxysmal tachycardia b. Atrial fibrillation with preexcitation—irregular wide-complex or intermittently wide-complex tachycardia, some with dangerously rapid rates faster than 250/min c. Atrial tachycardia or flutter with preexcitation Other Supraventricular Arrhythmias A. Premature atrial contractions (PACs) Defining feature: sinus rhythm with an early atrial complex distinct from

the sinus P wave resulting in an irregular rhythm. A pattern of ectopy (i.e., trigeminy, bigeminy) is sometimes seen with PACs B. Sinus arrhythmia Defining feature: irregular rhythm with a sinus P wave and with P-P intervals varying with respiration C. Accelerated junctional rhythm Defining feature: paroxysmal regular rhythm with P waves visible at the end of the QRS complex or not visible at all D. Ectopic atrial rhythm Defining feature: regular rhythm with a rate <100 beats/min but usually faster than sinus rhythm and with a P wave distinct from sinus that may or may not sustain occasionally syncope. Rarely, a supraventricular arrhythmia precipi tates cardiac arrest in patients with Wolff-Parkinson-White (WPW) syndrome or severe heart disease, such as hypertrophic cardiomyopa thy. Asymptomatic supraventricular arrhythmias are often captured on routine electrocardiographic (ECG) recordings and sometimes prompt

NARROW-COMPLEX TACHYCARDIA – OBTAIN FULL 12-LEAD ECG WITH LONG RHYTHM STRIP Regular atrial rate Atrial fibrillation VA block: more V’s than A’s AV block: more A’s than V’s 1:1 AV response • Junctional tachycardia • AVNRT • ORT • AT • Rarely atrial flutter • Atrial flutter • Atrial tachycardia • Rarely AVNRT with 2:1 block below the His bundle FIGURE 253-1 Diagnostic possibilities based on the appearance of the 12-lead electrocardiogram (ECG) recorded during an episode of supraventricular tachycardia (SVT). AT, focal atrial tachycardia; AVNRT, atrioventricular (AV) nodal reentry tachycardia; ORT, orthodromic AV reentry tachycardia. referral to a cardiologist. Most asymptomatic supraventricular arrhyth mias do not require treatment or further evaluation. ■ ■INITIAL EVALUATION The diagnosis of SVT is most often entertained when evaluating a patient for arrhythmia-related symptoms or when evidence of ven tricular preexcitation is seen on an ECG as an outpatient. Diagnosis of SVT requires obtaining an ECG at the time of symptoms (Fig. 253-2). Ventricular preexcitation on the resting ECG suggests AV reciprocat ing tachycardia using an accessory pathway. When the arrhythmia is ongoing at the time of recording, the ECG usually establishes or sug gests the diagnosis. In the urgent care or inpatient setting, treatment of SVT will often involve vagal maneuvers or carotid sinus massage (CSM) to achieve AV block (Table 253-2). In the appropriate patient, CSM should be used cautiously, if at all, if there is concern for carotid atherosclerosis that may be embolized during manipulation. If this is unsuccessful, the administration of 6 or 12 mg of adenosine to cause transient AV block is usually successful in terminating an AV nodal– dependent SVT or diagnosing a non-AV nodal–dependent SVT such as atrial tachycardia or atrial flutter. There are some atrial tachycardias AV nodal blockade (Adenosine or vagal reflex maneuver) Atrial rate continues with AV block SVT terminated SVT slows No effect • Fascicular VT • Inadequate dose/effect • Sinus tachycardia • Junctional tachycardia FIGURE 253-2 Diagnostic effect of increasing atrioventricular (AV) node blockade with vagal maneuvers, carotid sinus massage, adenosine, verapamil, or beta blockers. AT, focal atrial tachycardia; AVNRT, atrioventricular nodal reentry tachycardia; AVRT, atrioventricular reciprocating tachycardia; SVT, supraventricular tachycardia.

Irregular atrial and ventricular rates CHAPTER 253 Multifocal atrial tachycardia Approach to Supraventricular Arrhythmias
that are adenosine sensitive, and thus, termination of an SVT with adenosine does not exclude this potential diagnosis. For transient arrhythmias, ambulatory ECG recording is warranted. Patients will often have access to ECG recording devices, such as a watch or smartphone-enabled ECG recording electrode pair. Therefore, a patient may have an ECG diagnosis before seeing a physician (Fig. 253-3). Exercise testing is useful for assessing exercise-related symptoms and potentially evoking the arrhythmia. Additional evaluation for underly ing cardiac disease and to exclude

potentially dangerous arrhythmias should be performed based on the clinical scenario. Occasionally, an invasive electrophysiology study is warranted to provoke the arrhyth mia with pacing, confirm the mechanism, and risk stratify the patient, but most commonly, this is performed at the time of intended catheter ablation to treat the arrhythmia. Paroxysmal SVT is most commonly encountered in patients who do not have structural heart disease. Other supraventricular arrhythmias, particularly atrial fibrillation, are often associated with a variety of heart diseases. At initial evaluation, history and examination should assess possible underlying heart disease. Any abnormal findings may warrant further cardiac evaluation. • AVNRT • AVRT • Adenosine sensitive Focal AT • Atrial flutter • Atrial tachycardia

TABLE 253-2 Vagal Maneuvers Larynx Chest muscles PART 6 Disorders of the Cardiovascular System Lungs 15s Diaphragm Abdominal muscles Abdominal cavity Rectus muscles Holding breath while bearing down to increase intrathoracic pressure Breathing hard into a syringe against pressure to increase intrathoracic pressure Sternocleidomastoid muscle Cardiac plexus Submerge face into cold water (diver’s reflex) Carotid sinus massage Heart Rate Over 120 — 200 BPM Average This ECG was not checked for AFib because your heart rate was over 120 BPM. Reported Symptoms • Rapid pounding, or fluttering heartbeat • Chest tightness or pain • Fainting If you repeatedly get this result or you’re not feeling well, you should talk to your doctor. 0s 1s 2s 3s 4s 5s 6s 7s 8s 9s 10s 11s 12s 13s 14s 15s 16s 17s 18s 19s 20s 21s 22s 23s 24s 25s 26s 27s 28s 29s 25 mm/s, 10 mm/mV, Lead I, 511Hz, iOS 12.1.4, watchOS 5.1.3, Watch4,2 — The waveform is similar to a Lead I ECG. For more information, see Instructions for Use. FIGURE 253-3 Narrow-complex tachycardia recorded by a consumer wearable monitor (Apple watch). Afib, atrial fibrillation; ECG, electrocardiogram.

Raise legs abruptly to increase venous return Carotid sinus Vagus nerve Right common carotid artery Adenosine Adenosine

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