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ablative energy sources have been explored over the years, includ ing cryothermy, light spectrum (laser), microwave, ultrasound, and more recently pulsed field electroporation, which injures targeted myocardium through high-energy, ultra-short pulses of electrical current that disrupts the lipid cell membrane, resulting in perma nent cell death. Recently, the well-established ablative technique of stereotactic (focused and directed) external beam ionizing radiation has been applied to the heart to treat various arrhythmias, includ ing VT and AF. This particular treatment modality holds promise given its ability to target regions of the heart that may be inacces sible to catheters, as well as the completely noninvasive nature of the procedure. A widely applied non-RF ablative energy source today is cryo thermy, where an ablative catheter tip is cooled to a temperature range (typically below –40°C) that results in permanent tissue death. Cryothermy is most widely applied to ablation of paroxysmal atrial ablation, via an expandable balloon introduced sequentially into each pulmonary vein and cooled to produce a circumferen tial ablative lesion at the ostium/antrum of each pulmonary vein. Similar catheter-based tools utilizing pulsed field ablation (PFA) have also been introduced for the purpose of electrically isolating pulmonary veins during AF ablation. IMPLANTED ELECTRICAL DEVICE THERAPY Implanted cardiac rhythm management devices are commonly utilized to manage arrhythmia. The first definitive pacemaker was implanted in 1958, and this technology has evolved to be the mainstay in the management of bradyarrhythmias. Sinus node dysfunction and AV conduction disease, particularly with symp toms, are the primary indications for most implanted pacemakers. Pacemakers are typically implanted percutaneously, with insulated wires, or leads, inserted through the upper extremity venous sys tem into the right atrial and/or ventricular myocardium, with the lead tip secured to the myocardium mechanically. The leads are connected to a pulse generator placed in the prepectoral space, which contains electronic circuitry and a battery, allowing sensing and/or delivery of pacing stimuli to maintain adequate heart rate. More recently, a completely leadless pacemaker inserted through a large femoral venous sheath directly into the RA or right ventricle endocardium has become available. Although these devices possess more limited pacing options, they likely reduce the risks associated with transvenous lead systems, including infection or lead fracture requiring

extraction. Implanted cardioverter-defibrillators (ICDs) are placed in a simi lar fashion to pacemakers. However, ICDs have the ability to sense abnormal ventricular arrhythmias and deliver either anti tachycardia pacing or defibrillation to prevent sudden death. In patients who experience a potentially lethal ventricular arrhyth mia, ICD therapy may be lifesaving. Indications for ICD therapy are considered for either primary prevention of sudden cardiac death (SCD) due to arrhythmia in an at-risk patient or as second ary prevention in a patient who has survived an SCD event. More recently, a completely subcutaneous ICD system has become available, avoiding intravenous leads that increase risk for sys temic infection, and potentially the procedure to extract a poten tially fibrosed lead in cases of lead malfunction or endovascular infection. ■ ■FURTHER READING Callans DJ: Josephson's Clinical Cardiac Electrophysiology: Techniques and Interpretations, 7th ed. Philadelphia, Wolters Kluwer, 2024. Ellenbogen K et al (eds): Clinical Cardiac Pacing, Defibrillation, and Resynchronization Therapy, 5th ed. Philadelphia, Elsevier, 2016. Jalife J, Stevenson W (eds): Zipes and Jalife's Cardiac Electrophysiology: From Cell to Bedside, 8th ed. Philadelphia, Elsevier, 2021.

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The Bradyarrhythmias:

Disorders of the

Sinoatrial Node CHAPTER 251 The Bradyarrhythmias: Disorders of the Sinoatrial Node
The sinoatrial (SA) node serves as the natural pacemaker of the heart and has variable rates in response to parasympathetic and sympathetic stimulation. If the sinus node is dysfunctional or suppressed, a subsid iary pacemaker in the atrioventricular node or specialized conduction system will take over, leading to a junctional or ventricular rhythm. Symptoms of sinus node dysfunction can vary but typically present as fatigue, exercise intolerance, or dyspnea. The diagnostic evaluation includes an investigation into reversible causes of sinus bradycardia, confirmation of sinus node dysfunction with outpatient telemetry monitoring or exercise testing, and possibly cardiac imaging if struc tural heart disease is suspected. Once irreversible sinus node dysfunc tion is confirmed, permanent pacemaker implantation is the only reliable long-term therapy for symptomatic bradycardia. ■ ■STRUCTURE AND PHYSIOLOGY OF THE SA NODE The SA node region is complex in structure. Clusters of myocytes with pacemaker activity are surrounded by fibroblasts, endothelial cells, and transitional cells. These clusters of small fusiform cells in the sulcus terminalis on the epicardial surface of the heart at the right atrial–supe rior vena cava junction envelop the SA nodal artery. The SA node is structurally heterogeneous, but the central prototypic nodal cells have fewer distinct myofibrils than does the surrounding atrial myocar dium, no intercalated disks visible on light microscopy, a poorly devel oped sarcoplasmic reticulum, and no T tubules. Cells in the peripheral regions of the SA node are transitional in both structure and function. The SA nodal artery arises from the right coronary artery in 55–60% and the left circumflex artery in 40–45% of persons. This feature, along with a protective extracellular matrix of connective tissue, insulates the SA node from the hyperpolarizing influence of the larger atrium. In addition, the alignment of this complex matrix is associated with nearly unidirectional electrical propagation to the atrium (Fig. 251-1). Pacemaker cells spontaneously depolarize in a continuous manner setting the natural rate of depolarization and myocardial contraction. Action potential depolarization in the SA node is normally at a rest ing rate of 60–100 beats/min. The autonomic

nervous system exhibits control over the sinus node, with a preponderance of parasympathetic innervation at baseline. Removal of parasympathetic tone or an increase in sympathetic innervation leads to an increase in rate of depolarization. In denervated hearts, the rate of electrical depolariza tion (intrinsic heart rate) is approximately 100 beats/min, reflecting the rate of automaticity of the sinus node uninhibited by parasympathetic tone. The complement of ionic currents present in nodal cells results in a less negative resting membrane potential compared with atrial or ventricular myocytes. Electrical diastole in nodal cells is characterized by slow diastolic depolarization (phase 4), which generates an action potential as the membrane voltage reaches threshold. The action potential upstrokes (phase 0) are slow compared with atrial or ventric ular myocytes, being mediated by calcium rather than sodium current. Cells with properties of SA nodal tissue are electrically connected to the remainder of the myocardium by cells with an electrophysiologic phenotype between that of nodal cells and that of atrial or ventricular myocytes. Cells in the SA node exhibit the most rapid phase 4 depo larization and thus are the dominant pacemakers in a normal heart. Myocytes within the SA node complex include specialized cells surrounded by fibrous tissue. Unlike atrial and ventricular cells, sinus node pacemaker cells have no true resting potential, but instead depo larize automatically and repetitively after the end of an action potential, and the depolarizing current in the SA node myocytes results primarily from slow calcium currents instead of fast sodium channels, which are

Sinus Node Pacemaker Cells +30 mV PART 6 Disorders of the Cardiovascular System 0 mV Phase 0 Phase 3 –30 mV Threshold Phase 4 –60 mV iK if iCa-T iCa-L FIGURE 251-1 Cellular ion currents involved in depolarization and automaticity of sinoatrial (SA) nodal pacemaker cells. Phase 4 spontaneous depolarization results from if (funny) current, along with T- and L-type calcium channels. Phase 0 is the depolarization phase of the action potential. This is followed by phase 3 repolarization, which results from the outward directed hyperpolarizing K+ currents. if, funny current; iCa-T, T-type calcium current; iCa-L, L-type calcium current; iK, potassium current. absent in SA node cells. Spontaneous phase 4 depolarization results from a combination of slow inward depolarizing sodium current (If, “funny current”), along with inward calcium current controlled by T-type and L-type calcium channels. The upstroke of depolarization in SA node myocytes is slower and lower in amplitude than in ventricular myocytes. In patients <85 years of age, the resting heart rate is strongly influ enced by parasympathetic tone at baseline. The absence or elimination of autonomic influence on the SA node (e.g., after atropine adminis tration) leads to an intrinsic heart rate that is normally 100–110 beats/ min. The myocytes within the SA node that initiate pacing will change with different rates with a superior shift at higher heart rates and an inferior shift at lower rates. This shift may lead to a slightly different

P wave inscribed on electrocardiograms (ECGs) recorded during dif ferent rates of sinus rhythm. In addition, a progressive decline in maximum heart rate occurs with age, although the resting heart rate normally remains unchanged. Intrinsic heart rate declines 5–6 beats/min for each decade of age. However, the constancy of resting heart rate is associated with a grad ual decrease in parasympathetic tone and a transition to predominant sympathetic tone by the ninth decade. ■ ■DIAGNOSIS OF SA NODAL DISEASE Intrinsic sinus node disease is sometimes referred to as sick sinus syn drome or sinus node dysfunction (SND) and can manifest as fatigue, exercise intolerance, or syncope resulting from either reduced heart rate or pauses. Electrocardiographic recording plays a central role in the diagnosis and management of SA node dysfunction. The correla tion between symptoms and slow heart rate or pauses is essential in determining whether bradycardia may be

considered pathologic and necessitating intervention. Baseline ECG can detect baseline sinus bra dycardia but may not indicate symptom correlation in certain settings. To address the limitations of the resting ECG, longer-term recording employing mobile telemetry devices such as Holter monitors or mobile cardiac telemetry can also be helpful in correlating symptoms with rate abnormalities (Fig. 251-2). In addition, commercially available wearable devices, such as watches with ECG recording capabilities, can have electrograms with excellent fidelity that may also be utilized. Contemporary event monitors may be automatically triggered to record the ECG when certain programmed heart rate criteria are met and implantable monitors permit very longterm recording (years) in particularly challenging patients. Treadmill testing can be utilized to assess for maximum heart rate. It is worth noting, however, that standard Bruce protocol treadmill testing may

Phase 4 be helpful in detecting abnormalities in maximum heart rate, but more insidious chronotropic incompetence that manifests as abnormalities of rate increase during submaximal exercise may be more evident with treadmill protocols that have more gradual effort increases. Once there is evidence of SND, it is important to rule out reversible causes of resting sinus bradycardia or chronotropic incompetence. Table 251-1 lists the potentially reversible causes of sinus node dis ease and includes hypothyroidism and rate-slowing medications. Many patients with sleep apnea will have high vagal tone during sleep, especially during apneic events. Sinus bradycardia and sinus pauses frequently are seen if a patient is being monitored during this period. Sleep apnea, a common reversible cause, should be suspected if marked sinus bradycardia and prolonged sinus pauses are observed in a telemetry monitoring period during sleep. It is also worth noting that asymptomatic sinus bradycardia and pauses during sleep are typi cally not an indication for pacing, and it is, therefore, important when interpreting a wearable monitor to determine the timing of bradycardia events with regard to the sleep versus awake periods. If structural heart disease is suspected, transthoracic echocardiog raphy should be used to detect potential cardiac abnormalities associ ated with SND (Fig. 251-3). Advanced cardiac imaging is indicated for evaluation of possible myocardial diseases such as amyloidosis, infiltrative cardiomyopathy, or myocarditis. Invasive electrophysiology testing solely to assess sinus node function is rarely utilized beyond the noninvasive techniques mentioned. In patients who are undergoing electrophysiology studies (EPS) for other indications, evaluation of sinus node function as part of the EPS may be considered. In symp tomatic patients with suspected SND, EPS may rarely be considered when the diagnosis remains uncertain and after initial noninvasive evaluation is inconclusive. Investigation of the sinus node during EPS can consist of determination of sinus node recovery time (SNRT) and sinoatrial conduction time (SACT). In addition, the intrinsic heart rate [118.1 – (0.57 × age)] can be assessed via pharmacologic blockade of autonomic tone with intravenous propranolol and atropine. EPS is not widely used, however, as there is no evidence that abnormal SNRT or SACT alone can be used as an absolute indication for permanent pac ing (PPM). There is no indication for EPS in asymptomatic patients with sinus bradycardia. ■ ■SA NODAL DYSFUNCTION SUBTYPES SND can be categorized into problems with impulse formation and problems with impulse conduction. The term sick sinus syndrome may be used interchangeably with SND and refers to a group of related

Sinus (55 bpm), pause (3.4 seconds) 400 ms FIGURE 251-2 Sinoatrial exit block. A pause in the heart rhythm is seen that results from a sinus pause. On the second line of the tracing, there is a pause that results from the absence of a sinus beat (absent P wave) and no subsequent QRS. This

is followed by a junctional escape beat and eventually recovery of the presence of sinus rhythm P waves. conditions comprising problems of both impulse formation and impulse conduction. Sinus Node Exit Block (See Fig. 251-4) “Sinus arrest” results from failure of impulse formation within the sinus node. Sinoatrial exit block results from failure of sinus node activity to propagate to the atrium. Sinoatrial exit block can have similar pattern characteristics of types of atrioventricular (AV) node block. It can manifest as complete SA block. Type I SA block involves fixed delay out of the sinus node. Type II SA block can occur with either progressive delay and then intermittent failure to propagate to the atrium (Mobitz I type) or fixed delay with intermittent failure to conduct (Mobitz II). The mass of the sinus node is not large enough to have an appearance on the ECG. Instead, the P waves that result from atrial depolarization can provide information that reflects the health of the sinus node. Type II seconddegree SA block can be inferred on the ECG if the sinus rate abruptly transitions to a sinus rate that is half the previous rate (every other sinus depolarization is blocked from exiting to the atrium). Sinoatrial Wenckebach can be inferred from the ECG in the setting of progres sive shortening of the P-P interval leading up to a sinus pause. This is due to progressive prolongation of SA conduction, but to a lesser extent with each successive prolongation. This is similar to the typical progressive shortening of the R-R interval that is observed with AV nodal Wenckebach. Other types of SA block require invasive EPS to decipher. The exercise of determining the type of SA block with inva sive electrophysiology testing is typically not necessary because it does not alter management. Tachy-Brady Syndrome Tachycardia-bradycardia (tachy-brady) syndrome is a subset of sick sinus syndrome/sinus node disease that consists of high heart rates (most commonly atrial fibrillation) with alternating symptomatic bradycardia or offset pauses (Fig. 251-5). Commonly, medications that are needed for rate control of tachycardia exacerbate bradycardia episodes, and thus the presence of tachy-brady syndrome is often a reason to consider pacemaker implantation.

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Chronotropic Incompetence Chronotropic incompetence (CI) is broadly defined as the inability of the heart to increase its rate to meet activity or demand. Compared to an increased stroke volume, the increase in heart rate is a stronger contributor to the increase in oxygen uptake (VO2) during aerobic exercise. Therefore, CI can be the primary cause of severe exercise intolerance and increased cardiovas cular events and overall morality. Unfortunately, CI lacks a consistent definition, leading to a lack of clarity on its overall prevalence. CI can take many forms, including failure to achieve a percentage (e.g., 85%) of age-predicted maximal heart rate [(220 – age) × 0.85] during stress testing. Other definitions that have been used include an overall a max imum heart rate [208 – (0.7 × age)], heart rate instability with exercise, or failure to achieve submaximal heart rate. Due to this latter category, standard exercise testing can, at times, fail to recognize a patient with CI because some patients can achieve an appropriate maximum heart rate but may exhibit heart rate instability or inadequate heart rate during activities of daily living (ADLs). Ambulatory heart rate moni toring along with a diary can be helpful to correlate symptoms with abnormally slow heart rates. Because CI can be insidious and multiple definitions exist, it can be easily overlooked. Sinus Node Fibrosis Clinical SND is most common in older adults. This is due to normally occurring age-associated increase in fibrotic tissue in the SA node, which can exacerbate any degree of SND. A loss of pacemaker cells in the sinus node is also seen with age. It is worth noting, however, that while increased fibrosis in the SA node and decreased numbers of pacemaker myocytes are part of a normal process of aging, SND is pathologic and there are many elderly patients with extensive fibrosis and normal heart rate. SA Nodal Ischemia and Infarction Sinus

bradycardia is com mon in patients with acute inferior or posterior myocardial infarction (MI) and can be exacerbated by increased vagal tone (Bezold-Jarisch reflex) or with the use of drugs such as morphine and beta blockers. Ischemia of the SA nodal artery occurs in acute coronary syndromes

TABLE 251-1 Reversible Causes of Sinus Node Dysfunction Medical Conditions Associated with Sinus Bradycardia • Hypothyroidism • Sleep apnea • Hypoxia • Hypothermia • Increased intracranial pressure • Lyme disease • Myocarditis • COVID-19 • Vagal reflex (cough, pain, etc.) PART 6 Disorders of the Cardiovascular System Medications Associated with Sinus Node Dysfunction Antihypertensive Medications • Beta-adrenergic receptor blockers • Clonidine • Methyldopa • Nondihydropyridine calcium channel blockers Antiarrhythmic Medications • Amiodarone • Dronedarone • Flecainide • Procainamide • Propafenone • Quinidine • Sotalol • Ivabradine Psychiatric Medications • Donepezil • Lithium • Opioid analgesics • Phenothiazine antiemetics and antipsychotics • Phenytoin • Selective serotonin reuptake inhibitors • Tricyclic antidepressants Other • Anesthetic drugs (propofol) • Cannabis • Digoxin • Muscle relaxants more typically with involvement with the right coronary artery, and even with infarction, the effect on SA node function most often is transient. However, there are rare cases where SA infarction can affect sinus node function. One potential rare complication of atrial fibrilla tion catheter ablation is the inadvertent injury to the SA nodal artery that may be coursing over a targeted ablation region in the right and left atrium. SND and arrest have been described following ablation of atrial fibrillation and flutter. Carotid Sinus Hypersensitivity and Neurally Mediated Bradycardia Sinus bradycardia is a prominent feature of carotid sinus hypersensitivity and neurally mediated bradycardia associ ated with the cardioinhibitory variant of vasovagal syncope. Carotid hypersensitivity with recurrent syncope or presyncope associated with a predominant cardioinhibitory component responds to pacemaker implantation. Although the vasodepressor effect of the enhanced vagal tone may be unaffected by the pacing support, the lack of bradycardia often prevents injury with this subtype of vasovagal syncope. Several randomized trials have investigated the efficacy of permanent pac ing in patients with drug-refractory vasovagal syncope, with mixed results. Although initial trials suggested that patients undergoing pacemaker implantation have fewer recurrences and a longer time to recurrence of symptoms, at least one follow-up study did not confirm these results.

TREATMENT SA Nodal Disease TEMPORARY PACING FOR TRANSIENT SUPPORT In symptomatic patients presenting with sinus node disease, remov ing any possible reversible cause remains the initial strategy. Acute myocardial infarction, electrolyte abnormalities, medications, and hypothyroidism should all be considered as potentially reversible causes. Unnecessary medications that may be causing bradycardia should be eliminated. Beta blockers, calcium channel blockers, and digoxin are some of the more common medications in use that may cause bradycardia. These drugs may have a wide range of indica tions in patients after MI and with chronic systolic dysfunction. If stopping the medication or decreasing the dose is an option, this should be tried first. If the medication is felt to be unavoidable, a pacemaker may be indicated. In patients with tachy-brady syndrome, alleviation of the tachy cardia, whether it is atrial fibrillation or other forms of supra ventricular tachyarrhythmias, can prevent bradycardia events. Treatment of the tachycardia can sometimes be accomplished with antiarrhythmic drug therapy or catheter ablation. If arrhythmia control cannot be achieved, permanent pacing may be necessary. Hypoxia from decrease in blood flow to the SA node, which can occur with cardiac ischemia or MI, can lead to slowing of phase 4 depolarization and resultant bradycardia. Further ischemia and necrosis of pacemaker cells can cause irreversible sinus node dis ease. On occasion, reversal of ischemia with

revascularization can alleviate bradycardia. Sinus pauses in the setting of tachy-brady syndrome may be eliminated if atrial tachyarrhythmias can be suc cessfully treated. It is also important to recognize when bradycardia may be transient. Acute illness associated with episodes of extreme vagal tone may lead to transient SA node abnormalities. Typically, this may be observed as sinus slowing, followed by transient sinus arrest and/or AV block. Although a pacemaker may be needed in extreme instances of prolonged arrest, recovery from the acute ill ness may make the pacemaker unnecessary in follow-up. Sinus bradycardia may also be observed after heart transplanta tion and cardiac surgery. Due to cardiac denervation, a normal resting heart rate in heart transplant recipients is generally 90–110 beats/min. Therefore, a heart rate that may be normal in a non transplant patient may represent CI in a transplanted patient. In the case of heart transplantation, sinus bradycardia may be due to accumulated drugs such as amiodarone that affect the donor heart or ischemic injury to the SA node upon transplantation. If the SA nodal artery is injured at the time of right atriotomy during cardiac surgery, sinus arrest with junctional rhythm may be observed. Tem porary pacing or pharmacologic support with beta-1 adrenergic agonists may be needed in these circumstances while awaiting SA nodal recovery. In addition, sinus bradycardia and sinus pauses are common after spinal cord injury. The mechanism of bradycardia is enhanced parasympathetic tone and autonomic dysreflexia. Common trig gers can be tracheal suctioning and turning the patient. Atropine and inotropes have shown mixed success. Adenosine blockade with theophylline or aminophylline can sometimes be successful. Temporary and sometimes permanent pacing may be necessary in extreme circumstances. PERMANENT PACEMAKER IMPLANTATION Pacing in SA nodal disease is indicated to alleviate symptoms of bradycardia. Consensus guidelines published by the American Heart Association (AHA)/American College of Cardiology (ACC)/ Heart Rhythm Society (HRS) outline the indications for the use of pacemakers and categorize them by class based on levels of evi dence (Fig. 251-6). Since the first implementation of permanent pacing in the 1950s, many advances in technology have resulted in miniaturization, increased longevity of pulse generators, improve ment in leads, and increased functionality. To better understand

Treat underlying cause as needed, e.g., sleep apnea (Class I) Treatment effective or unnecessary Yes Observe Yes Transthoracic echocardiography (Class IIa) Suspicion for infiltrative CM, endocarditis, ACHD Yes No Advanced imaging (Class IIa) Treat identified abnormalities If not already performed: Exercise ECG testing (Class IIa) FIGURE 251-3 Evaluation of bradycardia and conduction disease. In patients with sinus node dysfunction, reversible causes should be identified and eliminated when possible. If no reversible cause can be identified, structural heart disease should be considered and evaluated for, if appropriate. If no symptoms are present, an observation strategy is appropriate. In patients who are symptomatic, further evaluation with ambulatory monitoring or exercise testing to identify symptom-rhythm correlation should be considered. ACHD, adult congenital heart disease; CM, cardiomyopathy. (Reproduced with permission from FM Kusumoto et al: 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. Heart Rhythm 16:e128, 2019.)

Evidence for sinus node dysfunction CHAPTER 251 Reversible or physiologic cause Yes No The Bradyarrhythmias: Disorders of the Sinoatrial Node
No Suspicion for structural heart disease No Symptoms Observe No Yes Exercise related Yes No Diagnostic If not already performed: Ambulatory ECG monitoring (Class I) No Yes Electrophysiology study (if performed for other reasons) (Class IIb) Sinus node dysfunction treatment algorithm

SAN EG VI PART 6 Disorders of the Cardiovascular System A III V B FIGURE 251-4 A. Mobitz type I sinoatrial (SA) nodal exit block. A theoretical SA node electrogram (SAN EG) is shown. Note that there is grouped beating producing a regularly irregular heart rhythm. The SAN EG rate is constant with progressive delay in exit from the node and activation of the atria, inscribing the P wave. This produces subtly decreasing P-P intervals before the pause, and the pause is less than twice the cycle length of the last sinus interval. B. Mobitz type II SA nodal exit block. This panel shows sinus rhythm in the first four beats followed by a sinus pause with the absence of a P wave. The interval comprising the absent P wave is exactly twice as long as the preceding P-P interval consistent with type II SA exit block. pacemaker therapy for bradycardias, it is important to be familiar with the fundamentals of pacemaker function. There is no established heart rate below which pacemaker treat ment is indicated (Table 251-2). Well-conditioned athletes can have resting sinus rates below 40 beats/min, and some individuals can have similar levels of bradycardia during sleep. Permanent pacing is typically not indicated for sleep-related pauses felt secondary to high vagal tone in the absence of other symptoms. Asymptomatic sinus bradycardia has not been associated with adverse outcomes and does not typically warrant permanent pacing. In situations such as asymptomatic sinus bradycardia, sinus pauses secondary to physiologically elevated parasympathetic tone, transient pauses during sleep, or asymptomatic SND where symptoms have been documented to occur in the absence of bradycardia, a pacemaker is generally not indicated. Medications to improve heart rate in order to avoid PPM are very rarely utilized. Medications such as methylxanthines (e.g., theoph ylline) or beta agonists (e.g., terbutaline) are sometimes utilized on Termination of Atrial Fibrillation (90-105 bpm), Pause (7.4 seconds) 400 ms FIGURE 251-5 Offset pause and tachy-brady syndrome. An offset pause after termination of atrial fibrillation is seen and is consistent with tachy-brady syndrome.

a temporary basis when a pacemaker may need to be delayed due to unique circumstances such as active infection. In addition, oral theophylline may be considered to determine if an increase in heart rate is associated with improvement in symptoms in a patient with sinus bradycardia to suggest that a PPM may be beneficial. This lat ter strategy is rarely utilized in more equivocal situations. PPM is the principal treatment for SND, and the decision to pursue this treatment is largely driven by a correlation between symptoms and bradycardia. The stronger the correlation between symptoms and bradycardia, the greater is the likelihood of improve ment. PPM is most commonly achieved through transvenous implantation of one or more leads through the left or right sub clavian veins into the cardiac chambers. The leads are attached to a pacemaker generator that is placed subcutaneously in the pectoral chest region. Less commonly, pacing leads can be placed in the epicardium via surgical approaches including sternotomy or thoracotomy. This latter approach can be accomplished as a standalone procedure but is more commonly performed concomitantly 10 mm/mV, 24 s

Due to required GDMT (no reasonable alternative) No Yes No (or asymptomatic) Yes Permanent pacing (Class I) Infrequent pacing? Significant comorbidities? Yes No No Single chamber ventricular pacing (Class IIa) Normal AV conduction and reason to avoid an RV lead? No Yes Dual chamber pacing (Class I) Single chamber atrial pacing (Class I) Program to minimize ventricular pacing (Class IIa) FIGURE 251-6 Management of sinus node dysfunction. Management of sinus node dysfunction begins with eliminating reversible causes and confirming whether symptoms correlate with bradycardia. If symptoms are clearly correlated, permanent pacing should be offered. If it is unclear, a trial of oral theophylline can be considered diagnostically. If there is no correlation

between symptoms and bradycardia, then observation is appropriate. Class I recommendations should be performed or are indicated. Class IIa recommendations are considered reasonable to perform. Class IIb recommendations may be considered. Class III recommendations are associated with harm more than benefit. AV, atrioventricular; GDMT, guideline-directed management and therapy; PPM, permanent pacemaker; RV, right ventricular. (Reproduced with permission from FM Kusumoto et al: 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. Heart Rhythm 16:e128, 2019.) TABLE 251-2 Indications for Permanent Pacing in Sinus Node Dysfunction (SND) • Symptoms that are directly attributable to SND • Symptomatic sinus bradycardia because of essential medication therapy for which there is no alternative treatment • Tachy-brady syndrome and symptoms attributable to bradycardia • Symptomatic chronotropic incompetence • In patients with symptoms that are possibly attributable to SND, a trial of oral theophylline may be considered to increase heart rate and determine if permanent pacing may be beneficial Source: FM Kusumoto et al: Heart Rhythm 16:e128, 2019.

Sinus node dysfunction Confirm symptoms Rule out reversible causes CHAPTER 251 The Bradyarrhythmias: Disorders of the Sinoatrial Node
Symptoms correlate with bradycardia Likely/uncertain Observation Oral theophylline (Class IIb) Permanent pacing (Class III: Harm) Response suggests symptomatic sinus node dysfunction? Yes Willing to have a PPM? No Yes Oral theophylline (Class IIb) during another primary cardiac surgery. Leadless pacemakers that are totally self-contained pacing devices can also be placed in the right atrium and right ventricle to provide dual chamber pacing. Some leadless pacemakers can also incorporate technology to sense atrial activity to attempt to coordinate atrial sensing with ventricu lar pacing. A standard nomenclature for pacing mode programming uti lizes a four-letter code. The first letter indicates the chamber(s) paced (O, none; A, atrium; V, ventricular; D, dual; S, single). The second letter indicates the chamber(s) sensed. The third letter is the response to a sensed event (O, none; I, inhibited; T, triggered;

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