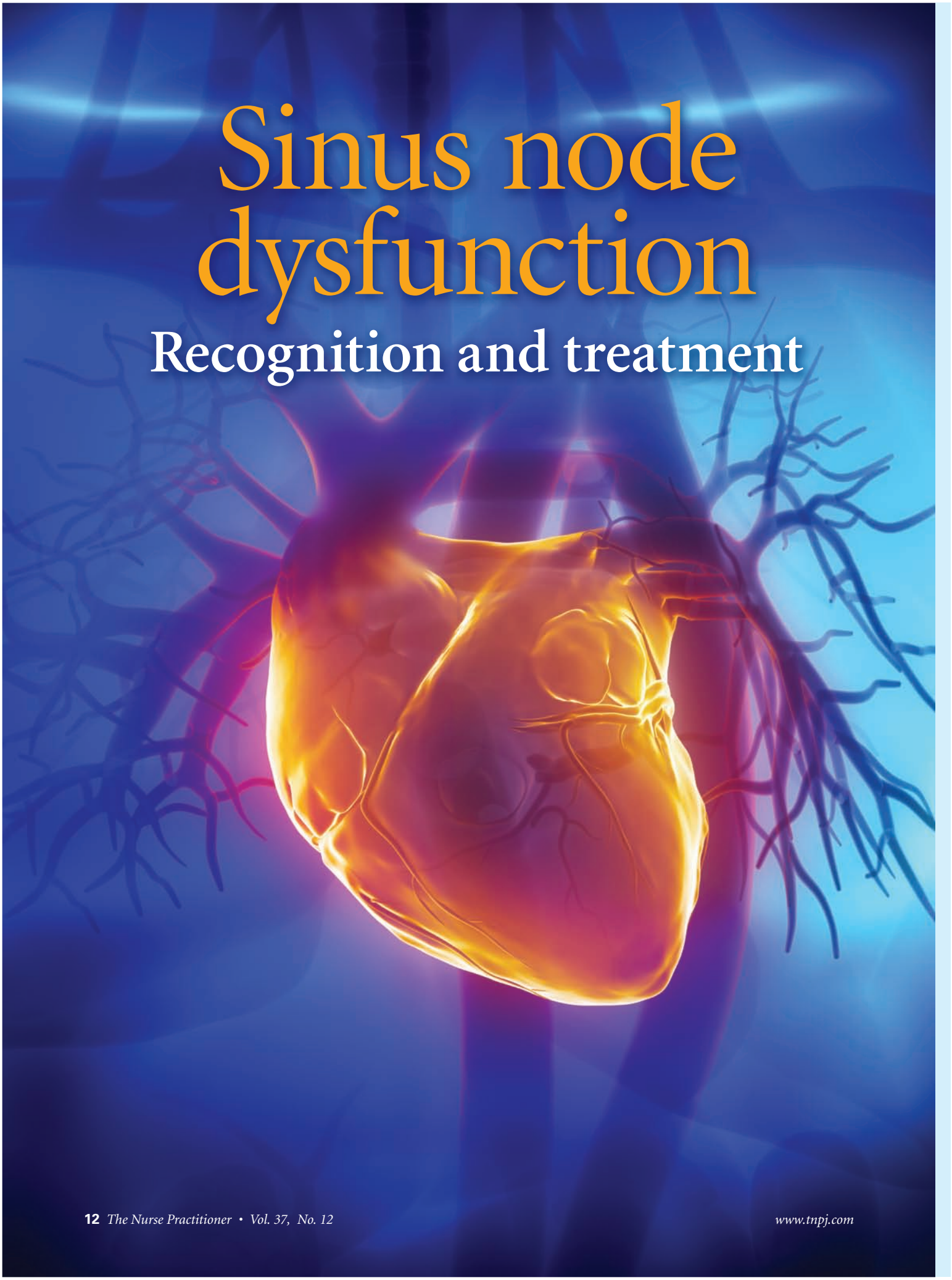
Sinus node dysfunction

Recognition and treatment



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Abstract: Sinus node dysfunction (SND) refers to a wide range of abnormalities involving sinus node and atrial impulse generation and propagation. SND occurs at any age and is commonly encountered in clinical practice. Clinicians must be able to accurately diagnose this syndrome, which can present from asymptomatic bradycardia to atrial standstill.

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Mr. S is a 78-year-old male (previously an avid walker) with a history of hypertension and osteoarthritis. His medications include losartan (50 mg daily) and arthritis strength acetaminophen p.r.n. He presented to the clinic for evaluation at his daughter's insistence with a 6-month history of fatigue, some dyspnea on exertion (DOE), and dizziness. According to his daughter's report, he is also experiencing periods of confusion, and she expressed concern about possible dementia. He denied chest pain or pressure, palpitations, syncope, or near syncope. His resting ECG (see *12-Lead ECG: Sinus bradycardia*) indicates sinus bradycardia with a ventricular rate of 51 beats/minute (bpm). There is no evidence of ST or T wave changes, left ventricular (LV) hypertrophy, or conduction abnormalities. Physical exam is essentially unremarkable with a resting heart rate (HR) of 56 and BP of 128/78. Blood was drawn for a thyroid-stimulating hormone, complete blood count, basic metabolic profile, and a nuclear stress test was performed to rule out myocardial ischemia as a possible cause for fatigue and DOE. Nuclear stress test results revealed a peak HR of 107 bpm, only 75% of his age-predicted maximal HR. Nuclear imaging demonstrated normal LV function with an ejection fraction (EF) of 60% and no inducible ischemia or prior myocardial injury. Stress test results were consistent with chronotropic incompetence and suggested sinus node dysfunction (SND) as a source of Mr. S's symptoms.

Introduction

SND, previously known as sick sinus syndrome, was first described as a clinical entity in the late 1960s.[1] SND refers to a wide range of abnormalities involving sinus node and atrial impulse generation/propagation.[2,3] Although SND occurs at any age, the incidence increases exponentially with advanced age,[3] the mean age for diagnosis is 68 years, and both genders are equally affected.[4,5] The incidence of this disorder is difficult to establish since it may be intermittent, and patients may be free from symptoms for many years. Furthermore, when the patient is symptomatic, the symptoms may be attributed to some other cause. Available estimates are that SND occurs in 1 of every 600 cardiac patients over age 65 and accounts for approximately half of the pacemaker implants in the United States.[5] Therefore, SND can be commonly encountered in clinical practice. As such, clinicians must be able to accurately diagnose this syndrome, which can present from asymptomatic bradycardia to atrial standstill.

Etiology

Most cases of SND are idiopathic, and the cause may be multifactorial.[5] The causes of SND may be classified as intrinsic (related to pathologic changes in the sinus node and/or atrial tissue), or extrinsic (disturbance of sinus node function caused by the influences of other factors in absence of structural abnormalities).[4,6] (See *Etiology of SND*.)

Intrinsic factors

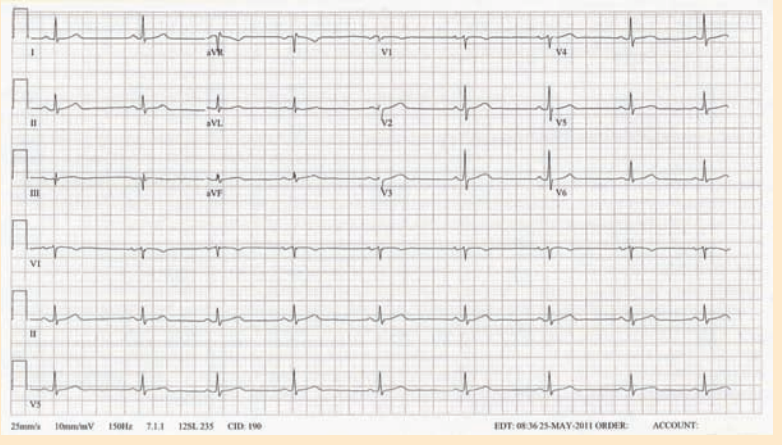
Degenerative and/or fibrotic changes in the sinoatrial (SA) node region are the predominant cause of intrinsic changes that lead to SND.[5,7,8] These changes may result from ischemia, inflammation, surgical trauma, or as part of the aging process.[6] With age, the intrinsic HR (defined as the HR in

Key words: cardiac pacemaker therapy, sick sinus syndrome, sinus node dysfunction

the absence of autonomic nerve activity) declines, and SA conduction time (SACT) increases;[9] age-related changes in ion channels have been suggested as a possible cause.[3] However, these pathologic changes may also result from concomitant conditions such as hypertension, atherosclerotic cardiovascular disease, cardiomyopathy, infiltrative disease, myocarditis, and collagen vascular diseases.[2,8,10,11] Since SND is typically diagnosed in individuals in their 70s or 80s, coexisting cardiovascular disorders are likely.[2]

Congenital heart disease and familial disorders of sinus node function can occur in younger individuals including the newborn.[3] Typically, corrective surgical procedures, such as the Mustard procedure performed for transposition of the great vessels and closure of atrial septal defects, contribute to SND.[7,8] A genetic component also appears to play a role in the development of SND in children and young adults[3,12] as does an autoimmune mechanism.[13]

12-Lead ECG: Sinus bradycardia



Etiology of SND

Intrinsic
- Idiopathic/degenerative
- Ischemic heart disease
- Hypertensive heart disease
- Cardiomyopathy
  - Infiltrative diseases
    - Sarcoidosis, amyloidosis
- Surgical trauma
  - Congenital heart disease
    - Mustard procedure
    - ASD closure
  - Cardiac transplant
- Inflammation
  - Collagen vascular disease
  - Rheumatic fever
- Infection
  - Viral myocarditis
  - Lyme disease
- Neuromuscular disorders
- Familial

Extrinsic
- Pharmacologic agents
  - Class IA, IC, III antiarrhythmic agents
  - Beta adrenergic blockers
  - Calcium channel blockers
  - Cardiac glycosides
  - Antihypertensives (such as clonidine, methyldopa)
  - Antipsychotics (such as lithium, phenothiazine derivatives)
  - Antidepressants (such as amitriptyline)
- Autonomic
  - Vasovagal syncope (cardioinhibitory)
  - Carotid sinus hypersensitivity
- Metabolic
  - Hypothyroidism
  - Hyperkalemia
  - Hypoxia
  - Intracranial hypertension

Adapted from Vijayaraman P, Ellenbogen K. Bradyarrhythmias and pacemakers. In: Fuster V, Walsh R, Harrington R, eds. *Hurst's the Heart.* 13th ed. New York, NY: McGraw-Hill; 2011.

Extrinsic factors

Numerous factors can affect sinus node function without causing structural disturbances including medications, autonomic nervous system influences, and metabolic disturbances. Cardioactive drugs may initiate/aggravate sinus bradycardia or chronotropic incompetence.[4] The medications most likely to affect sinus node function are beta-adrenergic blockers, calcium channel blockers, antiarrhythmic drugs (specifically Class IA, IC, and III), and cardiac glycosides.[8,11] (See *Classification of antiarrhythmic drugs.*) Psychotropic medications may also contribute to sinus node depression.[4,14]

Influences from the autonomic nervous system can affect normal sinus node function. Parasympathetic stimulation slows the sinus discharge rate and increases the intranodal conduction time, resulting in sinus node exit block at times.[4] Individuals with conditions such as vasovagal syncope and carotid sinus hypersensitivity frequently have associated bradycardia.[4,8] Heightened vagal tone from excessive physical training may result in syncope related to bradycardia or atrioventricular (AV) conduction abnormalities in otherwise healthy individuals.[7]

Hyperthyroidism and hypothyroidism produce changes in the cardiovascular system. Thyroid hormone affects the action potential duration and repolarization currents in the cardiac myocytes, resulting in changes in HR and development of dysrhythmias;[15] in hypothyroidism, this produces bradycardia. Electrolyte imbalances (particularly hyperkalemia) can result in bradycardia, sinus arrest, and SA exit block as well as AV block.[5]

Classification of antiarrhythmic drugs

Class	Action	Medication Examples
IA	Inhibits fast sodium channel, decreases automaticity, depresses phase 0, and prolongs the action potential duration.	• Disopyramide • Procainamide • Quinidine
IB	Inhibits fast sodium channel, depresses phase 0 slightly, and shortens action potential duration.	• Lidocaine • Mexiletine
IC	Inhibits fast sodium channel, depresses phase 0 markedly, slows His-Purkinje conduction profoundly leading to a prolonged QRS duration.	• Flecainide • Propafenone
II	Depresses phase 4 depolarization, blocks sympathetic stimulation of the conduction system.	• Acebutolol • Esmolol • Propranolol
III	Blocks potassium channel, prolongs phase 3 repolarization, prolongs action potential duration.	• Amiodarone • Dofetilide • Dronedarone • Ibutilide • Sotalol (also has class II effects)
IV	Inhibits inward calcium channel, depresses phase 4 depolarization, Lengthens repolarization in phases 1 and 2.	• Diltiazem • Verapamil

Source: Adapted from Morton PG, Fontaine DK. *Critical Care Nursing A Holistic Approach.* 9th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins. 2009:333.

Pathophysiology

The pathophysiologic mechanisms for SND include failure of impulse generation and failure of the impulse to spread throughout the atria.[3] In addition, there may be failure of secondary pacemaker activity (AV junction and Bundle of His) manifested as failed escape rhythms and increased atrial vulnerability to fibrillation and other tachydysrhythmias.[8] Atrial fibrillation results in sinus node remodeling on a cellular and molecular basis that may promote SND.[16]

Clinical presentation

The clinical manifestations of SND reflect the rhythm disturbances previously described. They are also related to a reduction in cerebral or peripheral perfusion and abrupt changes in HR. Symptoms are diverse and range from the following: nonspecific complaints of fatigue, palpitations, irritability, lassitude, lack of concentration, forgetfulness to the more dramatic symptoms of syncope, recurrent dizziness, and heart failure.[14,17] In some cases, stroke may be the first indication of SND in patients with paroxysmal atrial fibrillation and thromboembolism.[4] Furthermore, unrecognized SND may manifest as a perioperative complication that can cause various dysrhythmias—even cardiac arrest.[18]

Syncope in patients with SND is believed to be associated with dysrhythmias producing long periods of bradycardia (sinus pauses and SA block) occurring spontaneously or after termination of a tachydysrhythmia, resulting in inadequate cerebral blood flow.[2,8] Sinus bradycardia, even if severe, is rarely a cause of syncope; however, presyncopal symptoms such as lightheadedness, dizziness, and other nonspecific complaints may be observed.[8] The degree of bradycardia that may produce symptoms will vary based on the individual's physiologic condition, age, and functional ability.[14]

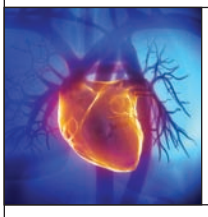
In some patients, the clinical manifestations may be more subtle and correspond to an inadequate HR response to daily activities. Most patients with chronotropic incompetence will exhibit symptoms of fatigue or shortness of breath with exertion.[14] A number of these complaints (especially fatigue, dizziness, and lightheadedness) are relatively common in many patients (older adults) who represent a large proportion of the SND population.[8]

Electrocardiographic manifestations

Abnormalities related to impulse formation within the sinus node and conduction through surrounding atrial tissue produce a variety of electrocardiographic manifestations. These include persistent sinus bradycardia, sinus arrest, sinoatrial exit block, chronotropic incompetence, and atrial or junctional tachydysrhythmias alternating between periods of bradycardia and asystole.[14]

Persistent sinus bradycardia is the most frequent ECG manifestation in SND.[17] This sinus bradycardia is persistent

(not caused by drugs) and is inappropriate for the physiologic circumstances.[7] In chronotropic incompetence, the sinus rate does not adequately increase in response to physical stress or exertion.[10] Chronotropic incompetence is present in 20% to 60% of individuals with SND.[4] In SA block, the SA node discharges at regular intervals, but some impulses are blocked from reaching the atria, and it is recognized by the dropping of sinus beats in a regular pattern. Sinus arrest is due to a failure of impulse formation in the sinus node itself. Sinus arrest is distinguished from SA block by the absence of sinus P waves that occur without any discernible pattern.[14] Bradycardia–tachycardia syndrome manifests as alternating periods of sinus bradycardia and tachycardia—most commonly atrial fibrillation or flutter.[7,14] Conversion from the tachycardia may be manifested by bradycardia or periods of sinus arrest,[11] which is referred to as postconversion pause or asystole.



Chronotropic incompetence is present in 20% to 60% of individuals with sinus node dysfunction.

More than one of these ECG manifestations may be recorded in the same individual at different times and may be associated with abnormal AV conduction as well.[7] This can be represented by atrial fibrillation—a common expression of SND—accompanied by a slow ventricular response in the absence of medication that depress AV conduction.[14]

Diagnostic evaluation

If the patient presents with typical symptoms, and the ECG findings are straightforward, the diagnosis may be simple. In many cases, however, the diagnosis of SND can be difficult. Many of the symptoms are nonspecific and are present in other disorders, especially among older adults; symptoms may be variable and intermittent. If the patient is asymptomatic at the time of evaluation, no ECG manifestations may be apparent. The history surrounding symptoms warrants a thorough investigation. Physical exam may reveal concomitant medical problems, evidence of structural heart disease, or extrinsic causes of SND. Carotid sinus massage (CSM), which is usually performed during autonomic testing with continuous ECG and BP monitoring, may reveal sinus pause of more than 3 seconds and/or hypotension in patients with carotid sinus hypersensitivity. Making the diagnosis of SND requires correlation of the symptoms with bradydysrhythmias,[14] which may be accomplished through ambulatory ECG monitoring. The ambulatory electrocardiography device is a continuous ECG recorder that records and stores data on two to three channels for 24- or 48-hour periods.[19] During monitoring, the patient maintains a diary of activities and symptoms, marking the events by pressing a button, so they can be correlated with ECG changes.[20] When symptoms are transient or infrequent, the brief snapshot of ECG activity (provided by the ambulatory electrocardiography device) is often inadequate in documenting an ECG-related cause, making long-term monitoring necessary.

Intermittent patient or event-activated recorders (also referred to as event monitors) allow for extended monitoring. Event monitors are typically worn for 30 days, and devices vary from those that save data only when activated by the patient to those with automatic triggers that transmit data transtelephonically to a central monitoring station for analysis.[20] These devices are highly effective in documenting infrequent events, although the quality of recording is not as consistent as that of the ambulatory electrocardiography device.[19] This type of event monitoring requires more effort on the part of the patient and some technical skill to transmit the data.

Real-time continuous monitoring systems automatically record and transmit event data from ambulatory patients to an attended monitoring station. Data can also be recorded through patient-triggered activation;[20] this type of device may be useful for patients unable to manage the technical requirements of the standard type of event monitor.

For patients with infrequent syncopal or presyncopal events suspicious for a dysrhythmic origin, an implantable loop recorder may be indicated.[20] The small, leadless device is implanted under the skin at about the second rib on the left front of the chest.[19] The device can be triggered by the patient when they place a special magnet over it or automatically, saving the data for retrieval at a later time.[20]

Invasive electrophysiologic testing

When noninvasive methods fail to provide an explanation for symptoms like syncope or presyncope that are suggestive of bradycardia or asystole, invasive electrophysiologic (EP) testing should be considered.[19] Sinus node recovery time (SNRT) and SACT are two tests that may be used as adjuncts in the evaluation of sinus node function.[21] SNRT is a measure of sinus node automaticity[4] and is the time required for sinus rhythm to resume once multiple stimuli of overdrive atrial pacing is terminated.[10] Since the spontaneous sinus rate influences the SNRT, a corrected sinus node recovery time (CSNRT) is calculated by subtracting the sinus node cycle length from the SNRT value;[21] a prolonged

CSNRT has been found in patients suspected of having SND.[19] SACT is the time from sinus activation to local atrial activation in the region surrounding the sinus node.[21]

The primary limitations of EP assessment in SND are the low sensitivity of the tests and the variable clinical significance of the abnormalities revealed.[6] The sensitivity of the SNRT and SACT are about 50% alone and approximately 65% combined.[6,19] The combined specificity is better at approximately 88%.[19]

If abnormalities are found, EP testing may support the diagnosis of SND but cannot be used to exclude sinus node disease in view of the limited sensitivity.[6] The EP study may also offer insight into other dysrhythmias (supraventricular or ventricular tachycardia) that may be responsible for the symptoms.

Patients with sinus bradycardia and syncope (but no other symptoms attributable to SND) should undergo autonomic testing such as carotid massage and tilt table testing, as well as EP testing, to uncover the mechanism of syncope.[17]

Exercise stress testing

Patients with symptoms such as fatigue, shortness of breath with exercise, or daily activities may have chronotropic incompetence. This is defined as the inability of the sinus node to achieve at least 80% of the age-predicted HR.[4] Exercise testing may be useful in determining the response of the sinus node to physiologic demands. If there is little or no increase in HR with exercise, the diagnosis of chronotropic incompetence is evident.[14] In other cases, the diagnosis is not so clear. Definitions of chronotropic incompetence rely on the HR response to maximal exercise.[22] Failure to achieve 80% of the maximum predicted HR (220 minus age) at peak exercise is considered evidence of a blunted HR response.[2] However, many patients are unable to perform significant exercise on the treadmill—especially older adults. The Chronotropic Assessment Exercise Protocol (CAEP) is an easily performed treadmill test that utilizes gradual increases in both elevation and speed, allowing assessment of chronotropic response during submaximal exercise.[22,23]

Treatment

Treatment of SND is directed toward alleviating symptoms, and management should begin with a search for reversible causes of sinus node depression; this includes consideration of medications, autonomic dysfunction, and ischemia. Antiarrhythmic agents (including beta-adrenergic blockers and calcium channel blockers) and other medications can suppress sinus node function.[14] Whenever possible, an alternative medication that may be equally effective without slowing the HR should be selected. Although theophylline and beta-adrenergic agonists may still be used in isolated cases to treat symptomatic bradycardia, they do not prevent syncope[4] and are not commonly employed in the treatment of SND.

Indications for pacemaker implantation

The only effective treatment for symptomatic bradycardia is permanent cardiac pacing.[2] According to the most recent American College of Cardiology (ACC), American Heart Association (AHA), and Heart Rhythm Society (HRS) device guidelines (see *Recommendations for permanent pacing in SND*), the Class I indications for permanent pacemaker implantation in SND include documented symptomatic bradycardia, symptomatic chronotropic incompetence, and symptomatic bradycardia that result from required drug therapy for medical conditions.[2] Pacemaker placement is reasonable (Class IIa indication) for SND in individuals with an HR less than 40 bpm when a clear association between significant symptoms are consistent with bradycardia, but the actual presence of bradycardia has not been documented. Pacemaker placement is also reasonable for syncope of unexplained origin when clinically significant abnormalities of sinus node function are discovered or provoked during EP studies.[2] Implantation may be considered in minimally symptomatic patients with chronic HR less than 40 bpm while awake (Class IIb indication).[2] Permanent pacemaker implantation is not indicated for SND in asymptomatic patients, for those whom the symptoms suggestive of bradycardia have been clearly documented to occur in the absence of bradycardia, and when symptomatic bradycardia is due to nonessential drug therapy (Class III indication).[2]

Single vs. dual-chamber pacing

Cardiac pacemaker therapy has been found to be highly effective in SND when bradydysrhythmias account for the symptoms.[8] Current practice has moved away from the use of single-chamber ventricular pacing (VVI and VVIR) in patients with SND unless permanent atrial fibrillation or other atrial disease prohibits the use of atrial pacing modes.[8] In SND, dual chamber (AV pacing) (see *Dual-chamber pacing leads*) has been found to reduce the risk of atrial fibrillation, reduce signs and symptoms of heart failure, and slightly improve the quality of life compared to single-chamber ventricular pacing.[24]

Dual-chamber pacing is able to maintain AV synchrony, prevent syncope during AV block, and slightly reduce the risk of atrial fibrillation. However, it may result in a high percentage of right ventricular (RV) pacing, which causes ventricular dysynchrony and is linked to increased risk of atrial fibrillation in patients with sinus node disease.[25] Pacing in the RV changes electrical activation and contraction of the ventricles resulting in ventricular remodeling, decreased

LV EF, and left atrial dilatation.[26] Ventricular dysynchrony caused by ventricular pacing (even when AV synchrony is preserved) has been found to increase the risk of heart failure and atrial fibrillation in patients with SND.[27]

Recommendations for permanent pacing in SND

Class I Pacemaer indicated
- SND with documented symptomatic bradycardia.
- SND as resulting from essential long-term drug therapy.
- Symptomatic chronotropic incompetence.

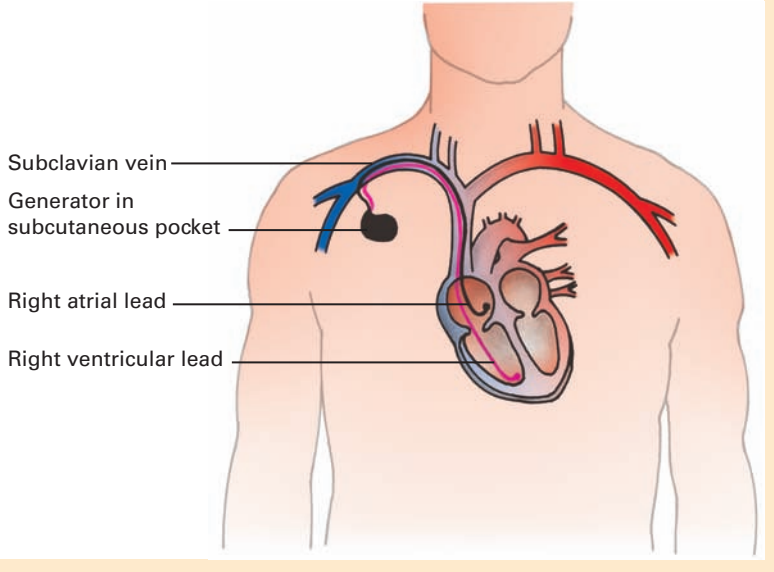
Class IIa Pacemaker reasonable
- SND with heart rate <40 bpm when a clear association between symptoms consistent with bradycardia and actual presence of bradycardia has not been documented.
- Syncope of unexplained origin when clinically significant abnormalities of sinus node function are found during EP studies.

Class IIb Pacemaker considered
- Minimally symptomatic with chronic heart rate <40 bpm while awake.

Class III Pacemaker not indicated
- Asymptomatic patients.
- Symptoms suggestive of bradycardia clearly documented to occur in the absence of bradycardia.
- Symptomatic bradycardia due to non-essential drug therapy.

Adapted from ACC/AHA/HRS. 2008 Guideline for device-based therapy for cardiac rhythm abnormalities. *Hert Rhythm.* 2008;5:e1-e62.

Dual-chamber pacing leads



Source: *Interpreting difficult ECGs. A rapid reference.* Philadelphia, PA: Lippincott Williams & Wilkins. 2006:200.

Pacing the RV from sites such as the Bundle of His, para Hisian tissues, right ventricular outflow tract (RVOT), and the RV septum has been explored in an effort to find a more physiologic alternative to apical pacing.[23,28] The sites most studied are the RVOT and septum, and to date, the results have failed to substantiate the perceived theoretical advantage.[28,29] Since lead placement in the RV apex remains the standard, various pacing strategies are employed to reduce the percentage of RV pacing.

Pacing modalities in SND

Efforts to reduce frequent and unnecessary RV apical pacing may be accomplished through various pacing strategies. Most patients with SND have intrinsic AV and normal intraventricular conduction.[30] Single-lead atrial pacing (AAIR) (see *The pacemaker code*) has been evaluated as an option to reduce unnecessary RV pacing. AAIR pacing requires intact conduction through the AV node and does not protect against bradycardia in the event AV block develops. When compared to dual-chamber (DDDR) pacing, AAIR pacing was associated with a higher incidence of paroxysmal atrial fibrillation, and a two-fold increased risk of pacemaker reoperation. DDDR pacing, programmed with a moderately prolonged AV interval, was identified as a better choice for patients with SND.[26] Pacemakers equipped with managed ventricular pacing mode, which automatically changes modes between AAI/R and DDD/R, can reduce unnecessary RV pacing as well.[30]

SND is associated with an inappropriate chronotropic response, the use of rate adaptive pacing, devices that incorporate one or more sensors to detect changes in activity, and is indicated to optimize tolerance with exertion.[8,14] The CAEP treadmill test is also used to assess optimal rate response in pacemaker patients with any type sensor.[23]

Stroke prevention

Atrial fibrillation is present in around 8% of patients with SND at the time of diagnosis, and the possibility of developing atrial fibrillation is approximately 5% per year.[4] Thromboembolic complications associated with atrial fibrillation contribute to the mortality and morbidity in patients with SND.[8] Reducing the risk of stroke by monitoring for atrial fibrillation and treatment with appropriate anticoagulation is also an essential part of treatment.

The pacemaker code

The capabilities of a permanent pacemaker can be described by a five-letter coding system (indicated by position I to V). For example, an AAIR pacemaker is atrium paced, atrium sensed, inhibited, and rate-adaptive. A DDDR pacemaker is dual-chamber paced, dual-chamber sensed, dual-response to sensing, and rate-adaptive.

Position I	Position II	Position III	Position IV	Position V
Chamber paced	Chamber sensed	Response to sensing	Programmability, rate modulation	Anti-tachycardia functions
A = Atrium	A = Atrium	T = Triggered	P = Simple	P = Pacing
V = Ventricle	V = Ventricle	I = Inhibited	M = Multi-programmable	S = Shock
D = Dual (both A&V)	D = Dual (both A&V)	D = Dual	R = Rate Adaptive	D = Dual (Shock/Pace)
O = None	O = None	O = None	C = Communicating O = None	O = None

Adapted from: Vijayaraman P & Ellenbogen K. Bradyarrhythmias and pacemakers. In: Fuster V, Walsh R, Harrington R., eds. Hurst's The Heart. 13th ed. McGraw-Hill; 2011. http://www/accessmedicine.com/content.aspx?aID+7814709. Reproduced with permission of the McGraw-Hill Companies.

Implications for practice

Sinus node dysfunction is commonly encountered in clinical practice. The clinical presentation can range from asymptomatic bradycardia to atrial standstill with a wide variety of symptoms (many of which are vague and intermittent). SND is commonly seen in older adults and is associated with a low incidence of sudden death. The survival of patients with SND appears to depend primarily on the severity of underlying cardiac disease and is not significantly changed by pacemaker therapy.[31] Unrecognized SND can lead to perioperative complications. With the increasing numbers of older adult patients undergoing surgery, it is important to consider SND in any patient that may present with dizziness, forgetfulness, or decreased exercise tolerance. If SND is suspected, elimination of reversible causes should be undertaken with immediate attention to medications. The patient's physical/cognitive abilities and available support system should be considered when selecting an ambulatory monitoring system to ensure optimal data collection and transmission. Patients with physical limitations such as severe arthritis or cognitive impairments may require a monitoring system that involves minimal patient participation.

Referral to an appropriate specialist will be required if autonomic/invasive EP testing or implantation of a cardiac pacemaker is indicated. Once a pacemaker is implanted, patients must be aware of the importance for regular device follow-up. This will help to insure optimal device function and allow for detection of atrial fibrillation, which may require anticoagulation for stroke prevention.

A favorable outcome

After reviewing the stress test results with Mr. S and his daughter, he was referred to a cardiac electrophysiologist for further evaluation of chronotropic incompetence. A dual-chamber pacemaker was successfully implanted without complications and was programmed DDDR with a moderately prolonged AV interval to minimize unnecessary RV pacing. He returned to the clinic for a follow-up visit 3 months after pacemaker implantation. Mr. S was without complaints and reported he had resumed his daily walking routine. He denied any further fatigue, dizziness, or DOE. His physical exam revealed a well-healed left pectoral pacemaker site, a resting BP of 122/66, and HR of 60 bpm. His daughter expressed that although Mr. S continued to experience an occasional episode of "forgetfulness," she was reassured by the improvement in his condition. NP

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