THE SICK SINUS SYNDROME

By Kathryn Buchanan Keller, RN, PhD, and Louis Lemberg, MD. From the Florida Atlantic University Christine E. Lynn College of Nursing, Boca Raton, Fla (KBK), and the Division of Cardiology, Department of Medicine, University of Miami Miller School of Medicine, Miami, Fla (LL).

A 20-year-old female college student was seen in the university student health center following a fainting spell while walking to class, and the third episode in 2 months. The social history was negative for recreational drugs. She was not taking any medications on a regular basis. On physical examination, her blood pressure was 110/70 mm Hg, pulse 70/min, respirations 18/min, height 5 ft 5 in (1.63 m), and weight 120 lb (54 kg). Carotid sinus pressure or massage failed to produce an attack. Chest X-rays, electrocardiogram (ECG), and echocardiogram were negative. Laboratory tests revealed sedimentation rate and high-sensitivity C-reactive protein (CRP) at normal levels. A 24-hour Holter monitor revealed no rhythm or repolarization abnormalities. The patient was reassured and told to return to the health center if symptoms recurred. Approximately 1 month later, the student was jogging on campus and again felt faint. A repeat 24-hour Holter monitor failed to reveal any arrhythmias. An event recorder was employed, but after 30 days no events had been captured. Finally, an implantable loop recorder had to be inserted and the traces shown in the figure were recorded (see Figure).

QUESTIONS

1. Which of the following interpretation(s) is/are applicable to the ECG trace in the figure?
   a. sick sinus syndrome (SSS)
   b. sino-atrial (SA) block
   c. SA arrest
   d. tachycardia-bradycardia syndrome
   e. atrial fibrillation (AF)

2. What management is indicated?
   a. observation
   b. atropine
   c. isoproterenol
   d. permanent pacemaker implantation

ANSWERS

1. a. SSS
   b. SA block
   c. SA arrest

Sick sinus syndrome is a disorder characterized by a dysfunctional sinus node. It is often idiopathic and a result of degenerative fibrosis of nodal tissue. Certain disorders such as amyloidosis, connective tissue disease, Chagas disease, and hemochromatosis are examples of intrinsic causes.1 Hypertensive heart disease and cardiomyopathies account for a smaller, but significant group that are also responsible for causing SSS.2 The extrinsic etiologies are pharmacological agents such as digitalis, calcium channel blockers, β-blockers, sympatholytic agents, and several antiarrhythmic drugs.1 The electrocardiographic presentations of SSS include (1) severe sinus bradycardia, (2) SA arrest or SA block, (3) SA arrest with an escape atrial or junctional rhythm, (4) SA arrest with failure of subsidiary pacemaker resulting in cardiac asystole, (5) bradycardia alternating with tachycardia, and (6) chronic AF with failure of sinus rhythm to return after cardioversion.2,3 Many patients with SSS may have more than 1 abnormality. In this case, the tracings show a markedly prolonged period of SA arrest or SA block. It is not possible to differentiate the 2 electrocardiographically. The sinus node may not fire at all (SA arrest) or may produce an impulse that cannot activate the atria because of SA block (block between the SA node and the SA fibers). The SA node may discharge, but if the impulse is blocked, the atrium will not be depolarized and consequently P waves will not be recorded. Electrophysiological studies with an electrode needle in the SA node can record SA nodal activity. The failure of a P wave recording under these circumstances is consistent with a block between the SA node and the atrial muscle, that is, SA block. The absence of an SA node impulse implies SA arrest.

The history and rhythm traces are characteristic of SSS. This syndrome is accompanied by disease or
Continuous trace from implantable loop recorder.
depression of the junctional tissue. The underlying pathology is observed when a junctional escape rhythm appears after a long pause. No etiology for the SSS could be identified in this patient. She was not taking any medication and had no inflammatory diseases or prolonged QT syndrome. There was no element of tachycardia, thus bradycardia-tachycardia syndrome was ruled out. However, it is important to note that the patient’s age was 20 years. Bradycardia-tachycardia is more common in the elderly with SSS. AF is the most common arrhythmia in patients with SSS, followed by the supraventricular tachycardias (eg, paroxysmal supraventricular tachycardia, atrial flutter, and atrial tachycardia).1 In patients with AF it is clinically significant to determine whether the AF is associated with SSS or with chronic AF that continues to have normal sinus function. Treating AF with cardioversion or medication can be hazardous if the sinus node is impaired.1

2. d. permanent pacemaker implantation

Generally, when a patient’s heart rate falls to less than 40 beats/min while awake and exhibits symptoms consistent with severe bradycardia (syncope, near syncope, lightheadedness, dizziness, or confused states), pacemaker implantation is indicated.2 Sinus pauses exceeding 3 seconds in symptomatic awake patients is also an indication for a pacemaker. Low heart rates or long RR pauses due to vagal influence may occur during sleep. However, a decrease in heart rate during sleep is more difficult to evaluate. Normally, atropine significantly increases the SA rate and is used in the diagnosis of sinus node dysfunction. However, when atropine 1 mg intravenously fails to stimulate the sinus node and increase the heart rate over 90 beats/min, it implies SA node dysfunction as SSS.2

When carotid sinus stimulation, by applying pressure over the SA node, results in cardiac arrest lasting more than 3 seconds, it is an indication of sinus node dysfunction. Carotid sinus pressure is as effective as massage and has the added advantage that the pressure can be titrated easily, whereas carotid sinus massage does not allow titration; additionally, carotid sinus pressure titration for effect is a safer maneuver than massage in the elderly. Atropine use and carotid sinus pressure should be performed while the patient is being electrocardiographically monitored. Holter monitoring is commonly used in the diagnosis; however, as in this case, infrequent occurrences of the arrhythmia in question may not be documented and may require multiple days. If the symptoms are severe and intermittent, then most likely sinus node function is severe, but intermittent.1 In this patient, after a repeat 24-hour Holter monitor and an event recorder failed to reveal arrhythmias, a loop recorder was used.3 The loop recorder is a small programmable device with autoactivation that captures and records arrhythmic events. The recorder is implanted subcutaneously in the left upper quadrant of the chest, where it is placed specifically between the first and fourth ribs and between the midclavicular line and the
parasternal line. The loop recorder stores arrhythmia events and holds the recorded digital data within a memory loop. Stored data can be retrieved with a programmer to be sent by telephone to the healthcare provider. The loop recorder is designed to be used for 12 months or more, thus providing long-term continuous electrocardiographic monitoring.

The treatment of SSS depends on the basic rhythm problem, but generally involves the placement of a permanent pacemaker. Treatment of bradycardia-tachycardia syndrome usually involves the placement of a pacemaker to control the bradycardia and use of medications to treat the tachycardia. In many patients with SSS, AF develops as a consequence of pacemaker implantation. An atrially based pacemaker with ventricular leads, rather than a ventricular pacemaker, is employed in order to decrease the incidence of AF. In patients with SSS, the incidences of AF and stroke are significantly lower when either an atrial paced or atrial sensed and inhibited response to sensing (AAI) or dual chamber paced and sensed (DDD) pacemakers are used as opposed to a ventricled paced, ventricle sensed, and inhibited response to sensing (VVI) pacemaker. These improved outcomes are attributed to the AAI and DDD pacemakers, which restore function and synchronization in both atrial and ventricular chambers.

Sudden death has been reported in teenagers and young adults with SSS and marked sinus bradycardia. With a history of 3 episodes of syncope and ECG documentation of unusually long pauses, pacemaker implantation is indicated.

SUMMARY

Adams in 1827 and Stokes in 1846 initially described syncopal attacks in patients with persistent bradycardia. The alternating bradycardia-tachycardia syndrome of SA nodal dysfunction was reported in 1954 by Short. The catchy title SSS currently used was initially coined by Lown in 1967 to describe bradycardia and associated supraventricular arrhythmias following electrical cardioversion of AF. Additional terms that are used synonymously include symptomatic sinus bradycardia, SA arrest, tachycardia-bradycardia syndrome, sinus pauses, chronotropic incompetence sluggish sinus syndrome, lazy sinus syndrome, and SA syncope. The SSS is usually encountered in the elderly; however, it may be seen at any age including children and adolescents. The SSS is most often idiopathic but has been drug induced and can be a result of coronary atherosclerosis, pericardial disease, atrial arrhythmias, diffuse fibrosis infective processes, and collagen vascular disease. Profound sinus bradycardia or SA arrest can occur during an acute inferior myocardial infarction and either secondary to ischemia, local edema, or autonomic neural influence. The degree of bradycardia or absolute cycle length at which pacing should be considered is controversial. However, it is generally accepted to be less than 40 beats/min. Equally symptomatic RR cycle lengths or sinus pauses exceeding 3 seconds during waking hours may warrant pacing. DDD pacing correlated to drug therapy is generally recommended in the treatment of SSS.

The ECG manifestations that can be seen in SSS are as follows:

1. Failure of the sinus rhythm to follow termination of any supraventricular arrhythmia, whether the termination is spontaneous or electrically induced.

2. The SA node can be “remodeled” by rapid atrial rates depressing the automaticity. As a result, the sinus bradycardia may be functional in part and reversible.

3. SA block.

4. Sinus bradycardia that is severe, persistent, intermittent, or inappropriate.

5. Sinus arrest with or without a new pacemaker arising.

6. Chronic AF with persistent slow ventricular rate, in the absence of bradycardia drugs.

ACKNOWLEDGMENT

Supported in part by a grant from the Applebaum Foundation in loving memory of Mr. Joseph Applebaum.

REFERENCES


SELECTED REFERENCES


The Sick Sinus Syndrome
Kathryn Buchanan Keller and Louis Lemberg

Am J Crit Care 2006;15 226-229
Copyright © 2006 by the American Association of Critical-Care Nurses
Published online http://ajcc.aacnjournals.org/

Personal use only. For copyright permission information:
http://ajcc.aacnjournals.org/cgi/external_ref?link_type=PERMISSIONDIRECT

Subscription Information
http://ajcc.aacnjournals.org/subscriptions/

Information for authors
http://ajcc.aacnjournals.org/misc/ifora.xhtml

Submit a manuscript
http://www.editorialmanager.com/ajcc

Email alerts
http://ajcc.aacnjournals.org/subscriptions/etoc.xhtml

The American Journal of Critical Care is an official peer-reviewed journal of the American Association of Critical-Care Nurses (AACN) published bimonthly by AACN, 101 Columbia, Aliso Viejo, CA 92656. Telephone: (800) 899-1712, (949) 362-2050, ext. 532. Fax: (949) 362-2049. Copyright ©2016 by AACN. All rights reserved.

Downloaded from http://ajcc.aacnjournals.org/ by AACN on August 14, 2017