Bradyarrhythmias

Clinical Presentation, Diagnosis, and Management

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KEYWORDS

• Bradyarrhythmia • Sinus node dysfunction • Atrioventricular block
• Tachycardia-bradycardia syndrome • Sinus arrest

KEY POINTS

- Bradyarrhythmias can reflect normal physiologic responses, like sleeping, or reveal a number of rhythm disorders, including sinus node dysfunction and atrioventricular conduction disturbances.
- In patients with confirmed or suspected bradycardia, a thorough history and physical examination includes signs/symptoms, precipitating factors, medications, history of coronary artery disease, cardiac arrhythmia, and sudden death.
- Management is based on the severity of symptoms, underlying causes, presence of potentially reversible causes, presence of adverse signs, and risk of progression to asystole.

INTRODUCTION

Bradycardia, also known as bradyarrhythmia, is a common finding for both healthy individuals and those who are ill. This paper provides an overview on types and causes of bradyarrhythmia, the clinical presentation, diagnosis, and management.

DEFINITION OF BRADYCARDIA

In adults, bradycardia has traditionally been defined by consensus as a slow heart rate (HR) of fewer than 60 beats per minute (bpm).[1] This easily remembered HR threshold of 60 bpm for bradycardia has been challenged because it overdiagnoses bradycardia and is not consistent with published age- and sex-specific norms.[2–4] However, resting HR among the healthy, asymptomatic population varied greatly. A slow HR may be normal physiologically for some individuals, but may be inadequate for others. Classic work from Jose and Collison[5] (1970) showed that HR decreases with age. There is also

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a circadian cycle of HR, with fastest rates occurring between 1400 and 1700 hours and the slowest rates occurring between 0400 and 0600 hours.[6] During sleep, HR decreases by an average of 24 bpm in young adults[7,8] and by 14 bpm in those greater than 80 years of age.[6] Women have faster HR than men during both waking and sleeping periods, by an average of 10 bpm in young adults.[7,8] Spodick[9] reported that the "normal" range of HRs in the afternoon was 46 to 93 bpm for men and 51 to 95 bpm for women; thus, they proposed an HR of 50 bpm to be an appropriate level for defining bradycardia in adults.[9–11]

BRIEF OVERVIEW OF THE SINOATRIAL NODAL CONDUCTION SYSTEM

Cardiac rhythm is initiated and controlled by the sinoatrial (SA) node, the primary pacemaker of the heart. Current scientific knowledge indicates that the SA node structure consists of clusters of specialized cardiomyocytes enmeshed within strands of connective tissue or fibrosis.[12] In addition, there are also distinct SA conduction pathways that electrically connect the SA node to the right atrium. These SA conduction pathways play an important role in regulating the SA node automaticity and, thus, the maintenance of the HR.[12] Autonomic stimulation, ischemia, and/or structural remodeling can compromise the SA node pacemaker function and inhibit the impulse through the SA nodal conduction pathways (exit block).[12] The SA node exit block allows the impulse to originate from subsidiary pacemakers, such as the atrioventricular (AV) node and the specialized ventricular conduction system.

The HR is modulated by several factors, including the autonomic nervous system (the dynamic balance between sympathetic and parasympathetic nervous systems), the baroreceptors, the Bainbridge reflex, and the intrinsic HR.[13] The autonomic nervous system is reported to be more densely innervated in the cardiac conduction system than in the myocardium in other parts of the heart, with the SA node being the most densely innervated region of the conduction system.[14] This observation supports the central role of the autonomic nervous system in initiating and regulating the cardiac impulse. Sympathetic and parasympathetic nervous systems interact with adrenergic (α- and β-) and muscarinic receptors. In general, stimulation of β-adrenoceptors increases HR, whereas stimulation of muscarinic receptors decreases HR.[15]

The idea that the cardiac impulse originates from a very focal region in the SA node has been challenged. Boineau and associates[16] demonstrated that humans have a widely distributed physiologic pacemaker complex extending across a significantly larger area of atrial tissue. The concept of an "atrial pacemaker complex," including the SA node, SA nodal conduction pathways, and the surrounding atrial myocardium, is proposed to initiate normal atrial or sinus rhythm.[16]

TYPES OF BRADYARRHYTHMIAS

Bradyarrhythmias can reflect normal physiologic responses, as in sleeping, or reveal a number of rhythm disorders, including sinus node dysfunction and AV conduction disturbances.[17] Sinus node dysfunction is caused by a depressed automaticity or an impaired SA node and atrial impulse formation and/or propagation. Sinus node dysfunction, sometimes used interchangeably with "sick sinus syndrome,"[18,19] refers to a spectrum of heart rhythm disturbances, including sinus bradycardia (**Fig. 1**), sinus arrest, sinus exit block, and tachycardia–bradycardia syndrome.[18] Of note, the bedside monitor is sensitive to artifactual noise in the electrocardiogram (ECG) signal so that artifact can generate a false-positive bardycardiac alarm (**Fig. 2**). Chronotropic incompetence, defined as the inadequate HR response to increased activity or