

An overview of diagnosis and management of bradycardia: Literature review

Fahad Abdullah Alnajim¹, Mohammed Abdullah S Alkhidhr², Mohammed Abdullah A Alanazi³, Abdulaziz Abdullah J Bawazeer⁴, Atheer Ibrahim Shahar⁵, Basmah Mohammad Alsharif⁶, Omer Quayid Kh Alanazi⁷, Darraj Omar Qasem I⁷, Nagla Mohamed Mohamed⁸, Ahmed Ali M Alawi^{9*}, Abdulrahman Bin Suliman Alsoghayer¹⁰

¹ Faculty of Medicine, Imam Abdulrahman bin Faisal University, Dammam, KSA. ² Pediatric Department, Maternal And children Hospital Arar, KSA. ³ Internal medicine department, northern medical tower, Arar, KSA. ⁴ Department of Internal Medicine, Alnoor Specialist Hospital, Makkah, KSA. ⁵ Faculty of Medicine, Jazan University, Jazan, KSA. ⁶ Cardiac Surgery Intensive Care Unit, Madinah cardiac center, Madinah, KSA. ⁷ Faculty of Medicine, Jordan University of Yarmouk, Yarmouk, Jordan. ⁸ Imam Abdulrahman bin Faisal Hospital, National Guard, Dammam, KSA. ⁹ Obstetrics and Gynecology Department, King Abdullah Medical Complex, Jeddah, KSA. ¹⁰ Faculty of Medicine, Dar Al Uloom University, Riyadh, KSA

Abstract

Background: Bradycardia is a common finding on physical examination in symptomatic and asymptomatic patients. It can be linked to physiological changes as well as the pathological progression of an underlying disease. The symptoms of bradycardia include lightheadedness, syncope, exercise intolerance, or, in some cases, cardiac arrest. A proper history and physical exam focused on the severity assessment of bradycardia and the underlying condition is essential for the management. **Objectives:** We aimed to review the literature reviewing bradycardia, along with the possible etiologies, clinical features, diagnosis, and management in both the acute setting and definitively. **Methodology:** PubMed database was used for article selection, and papers were obtained and reviewed. **Conclusion:** Bradycardia, while is an innocent presentation in most cases, can progress rapidly into cardiac arrest and death. Proper recognition and risk assessment of which patients might develop the severe sequelae of this presentation is essential in the care process of patients. While this subject has been heavily understudying, the only effective treatment for irreversible bradycardia remains permanent pacing of the heart.

Keywords: Bradycardia, Bradyarrhythmia, Sinus node dysfunction, Sinus bradycardia, Atrioventricular conduction delay, Heart block, Sick sinus syndrome, Pacemaker

INTRODUCTION

Bradycardia is defined as a heart rate of fewer than 60 beats per minute. [1] It is not a disease per se, as it can develop physiologically while sleeping and in athletes. Also, bradycardia is more common in the elderly as a result of normal aging and other diseases progression. [2] However, what we are afraid of is symptomatic bradycardia that will develop severe and life-threatening sequelae and complications. [3] Thus, appropriate recognition of bradycardia and evaluation of the patient as a whole is essential to minimize the risk of those sequelae and complications. In this review, we will cover the causes of bradycardia, along with the signs and symptoms, diagnosis, management, and complications.

METHODOLOGY

PubMed database was used for article selection, and the following keys were used in the mesh (Bradycardia) OR (Bradyarrhythmia). In regards to the inclusion criteria, the articles were selected based on the inclusion of one of the following topics; Bradycardia, Bradyarrhythmia, Clinical features, Diagnosis, and Management. Exclusion criteria

were all other articles that did not have one of these topics as their primary endpoint.

Review

Etiology:

There are multiple causes of bradycardia. However, some are more severe than others. The three major causes of bradycardia are sinus bradycardia, sinus node dysfunction (previously called sick sinus syndrome), and atrioventricular blocks. [4]

Address for correspondence: Ahmed Ali M Alawi, Obstetrics and Gynecology Department, King Abdullah Medical Complex, Jeddah, KSA.
Email: ahmedalawi473 @ gmail.com

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 3.0 License, which allows others to remix, tweak, and build upon the work non commercially, as long as the author is credited and the new creations are licensed under the identical terms.

How to cite this article: Alnajim, F. A., Alkhidhr M. A. S., Alanazi, M. A. A., Bawazeer, A. A. J., Shahar, A. I., Alsharif, B. M. and *et al.* An Overview of Diagnosis and Management of Bradycardia: Literature Review. Arch Pharma Pract 2021;12(1):13-5.

- **Sinus Bradycardia**

Sinus bradycardia is defined as bradycardia with a normal sinus rhythm on an electrocardiogram (ECG).^[5] This means that the sinus node is firing impulses at a slower rate than normal. The normal firing rate for the sinus node is around 60 to 100 beats per minute. A regular heart rate can be defined on ECG as no or minimal changes in the interval between each R wave.^[1] Sinus bradycardia can be a normal finding in healthy individuals, as it is responsible for the physiological slowing of heartbeats during sleep. However, it can also occur as a pathological response to other conditions.^[5] Common pathological causes of sinus bradycardia include myocardial infarction, obstructive sleep apnea, medications beta-blockers, and infections such as Lyme disease.^[6]

- **Sinus Node Dysfunction (SND)**

SND can be defined as the inability of the sinoatrial (SA) node to generate an adequate heart rate that meets the physiological demands of an individual.^[7] This condition may manifest either as abnormal automaticity, failure of a generation of impulse, or abnormal conduction, failure of transmission. SND is secondary to multiple etiologies such as sinus node fibrosis, medications such as antiarrhythmics, infiltrative diseases, and trauma during surgery.^[7]

- **Atrioventricular (AV) conduction delay**

AV block can be defined as a conduction delay or interruption of an impulse from the atria to the ventricle.^[8] Classically, it is divided into three degrees. In first degree AV block, there is a delay in the transmission of impulses from the atria to the ventricles without interruption. In second degree AV block, there is an interruption of impulse propagation that will lead to a “missed beat”, usually in a regular pattern. In third-degree AV block, also called complete heart block, no atrial impulses are propagated to the ventricle. Thus, the AV node works as the pacemaker of the heart.^[1, 8] The common etiologies for AV conduction delay are myocardial infarction, cardiomyopathy, congenital heart diseases, metabolic abnormalities, and in some cases it can be idiopathic.^[4]

Clinical Features

In physiological cases, bradycardia is asymptomatic. When bradycardia becomes severe or is associated with other pathologies, it may manifest as lightheadedness, presyncope, and syncope, chest pain on exertion, symptoms of heart failure, cognitive slowing, and exercise intolerance.^[1, 9] Symptoms usually appear or are increased in a situation where an increase in cardiac output is required. For example, during exercise, stress, or during an active infection.^[4, 9] It is important to know that, in many cases, patients do not usually present with the complaint of bradycardia rather the underlying condition itself, for instance, myocardial infarction. However, in some cases, patients may develop symptomatic bradycardia that mandates proper and rapid management.^[2] Symptomatic bradycardia is defined as “documented bradyarrhythmia that is directly responsible for the development of the clinical manifestations of syncope or

presyncope, transient dizziness or lightheadedness, heart failure symptoms, or confusion state, resulting from cerebral hypoperfusion attributable to slow heart rate”.^[2, 10]

Diagnosis

A thorough history and physical examination are required not only to diagnose bradyarrhythmia but also to discover the underlying etiology and comorbid condition of the patient. It is essential to ask about the details of the episode of bradycardia, the frequency, timing, duration, triggers, and alleviating factors.^[1, 2] Since medications are a common cause of bradycardia, one must always ask about both prescription and over-the-counter medication usage. Asking about previous episodes and how closely matched those were to the recent episode is also important as it may indicate different pathologies or similar ones. In the end, physicians should end their history with a comprehensive review of systems, family history of comorbid conditions, and cardiovascular risk assessment.^[2] During the physical exam, physicians must focus on the examination of the cardiovascular system. However, other systems must also be examined to determine whether a mixed pathology is present or not. One important note during a physical exam is carotid sinus massage to exclude carotid sinus hypersensitivity syndrome that may present with features similar to bradycardia.^[1, 3] However, due to the risk of stroke precipitated by carotid sinus massage^[11, 12], it must be done in a safe environment with the patient’s blood pressure and ECG monitoring.^[2]

After the history and physical examination, the most essential tool for evaluation is a 12-lead ECG. It can detect the abnormality whether it is a conductive or generative error. However, for it to be effective, it must be done during the attack.^[2] If the symptoms are resolved, the ECG will not be of value in the diagnosis of bradyarrhythmia as it will show normal heart rhythm in most cases, unless it is due to an AV conduction defect.^[1, 4] However, it remains essential to rule out other pathologies that may be found. For example, myocardial infarction and cardiomyopathies. If the ECG reading is normal and the patient reports that symptoms appear or increase in severity with exercise, exercise ECG is indicated to determine the abnormality. However, in most cases, it can be difficult to obtain useful data from exercise ECG because of the comorbid condition of most patients.^[2, 9] If suspicion remains high for bradycardia, an ambulatory monitor, such as holter, can be used to diagnose bradyarrhythmia.^[4, 6] Other tests that can be done are laboratory testing and invasive testing. These tests should never be done routinely and only when high suspicion of an underlying pathology be present. Examples of lab tests include thyroid screening and Lyme titers. An example of invasive testing is the implantable cardiac monitor.^[2, 4]

Management

Bradycardia management depends on multiple factors. These include the severity of the symptoms, the underlying etiology, the reversibility of the condition, the presence of signs

indicating unstable condition, and the risk of progression into asystole, also called cardiac flatline. ^[1, 4] The signs that indicate an unstable patient are signs of shock, syncope, heart failure, and myocardial infarction. Those patients are at risk of cardiac arrest and death and must be treated initially in accordance with the advanced cardiac life support (ACLS) guidelines and principles. ^[10, 13]

In the acute setting, increasing the heart rate is the top priority of the management team. Initial pharmacological therapy is by using atropine sulfate. ^[10, 13] Atropine is an antimuscarinic drug. It acts by reversing the cholinergic effect on the heart that reduces the heart rate. The recommended dose in adults is 0.5 mg intravenously, repeated 3-5 times to a maximum dose of 3 mg if necessary. ^[10, 13] In cases where atropine is ineffective, β -adrenergic agonists can be used. Examples of these include dopamine and epinephrine. ^[10] If the patient remains unstable, temporary pacing should be initiated. There are multiple types of temporary pacing. However, the most commonly used one in the acute setting is transvenous pacing. ^[4, 14, 15] Furthermore, this type requires sedatives or analgesics as the procedure is painful.

Treatment of the underlying cause is essential. For instance, if the patient is suffering from hypothyroidism, proper management of the patient's condition is all that is needed to manage his bradycardia. ^[1] However, in some patients, the underlying cause cannot be reversible such as in cases where sinus node fibrosis has set in secondary to myocardial infarction. Permanent pacemaker implantation is the only effective treatment for those patients. ^[4] Furthermore, it should be done based on the severity and frequency of the symptoms, the risk of symptoms progression, and the type of bradyarrhythmia the patient suffers from. ^[4] Generally, patients with third-degree and second-degree type 2 heart block must be permanently paced as there is a risk of severe complications and rapid destabilization of their condition. ^[2]

CONCLUSION

Bradycardia is a common clinical finding. It can be related to various physiologic and pathologic conditions. It can be benign, requiring no treatment. However, it can lead to cardiac arrest and death in select patients. A thorough history and physical examination should include the chief complaint of bradycardia in addition to the possible causes of this condition. Management of bradycardia is based on whether the condition is life-threatening in the acute attack or the future. Pharmacologic therapy and/or pacing are used to manage unstable or symptomatic bradycardia. Permanent pacing remains the only effective therapeutic option to manage patients with an irreversible cause of bradycardia.

REFERENCES

1. Sidhu S, Marine JE. Evaluating and managing bradycardia. *Trends in cardiovascular medicine*. 2020 Jul 1;30(5):265-72. doi:10.1016/j.tcm.2019.07.001.
2. Kusumoto FM, Schoenfeld MH, Barrett C, Edgerton JR, Ellenbogen KA, Gold MR, Goldschlager NF, Hamilton RM, Joglar JA, Kim RJ, Lee R. 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Journal of the American College of Cardiology*. 2019;74(7):e51-e156.
3. Hasan F, Bogossian H, Lemke B. Akute Bradykardien. *Herzschrittmachertherapie+ Elektrophysiologie*. 2020 Mar;31(1):3-9. doi:10.1007/s00399-020-00665-z.
4. Wung SF. Bradyarrhythmias: clinical presentation, diagnosis, and management. *Critical Care Nursing Clinics*. 2016 Sep 1;28(3):297-308.
5. Wilders R, Verkerk AO. Long QT Syndrome and Sinus Bradycardia—A Mini Review. *Frontiers in cardiovascular medicine*. 2018 Aug 3;5:106.
6. Alboni P, Stucci N, Parisi C. Sinus bradycardia and syncope: what pathophysiological mechanism and what management of the patient?. *Giornale italiano di cardiologia* (2006). 2017 Nov;18(11):774.
7. De Ponti R, Marazzato J, Bagliani G, Leonelli FM, Padeletti L. Sick sinus syndrome. *Cardiac electrophysiology clinics*. 2018 Jun 1;10(2):183-95.
8. Leonelli FM, Bagliani G, De Ponti R, Padeletti L. Intraventricular delay and blocks. *Cardiac Electrophysiology Clinics*. 2018 Jun 1;10(2):211-31.
9. Guasch E, Mont L. Diagnosis, pathophysiology, and management of exercise-induced arrhythmias. *Nature Reviews Cardiology*. 2017 Feb;14(2):88.
10. Panchal AR, Berg KM, Hirsch KG, Kudenchuk PJ, Del Rios M, Cabañas JG, Link MS, Kurz MC, Chan PS, Morley PT, Hazinski MF. 2019 American Heart Association focused update on advanced cardiovascular life support: use of advanced airways, vasopressors, and extracorporeal cardiopulmonary resuscitation during cardiac arrest: an update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2019 Dec 10;140(24):e881-94.
11. Van den Brink RB, de Lange FJ. Carotid sinus massage is not a benign intervention. *Nederlands Tijdschrift Voor Geneeskunde*. 2017 Jan 1;161:D1312-.
12. van Munster CE, van Ballegoij WJ, Schroeder-Tanka JM, van den Berg-Vos RM. A severe stroke following carotid sinus massage. *Nederlands Tijdschrift Voor Geneeskunde*. 2017 Jan 1;161:D826-.
13. Soar J, Nolan JP, Böttiger BW, Perkins GD, Lott C, Carli P, Pellis T, Sandroni C, Skrifvars MB, Smith GB, Sunde K. European resuscitation council guidelines for resuscitation 2015: section 3. Adult advanced life support. *Resuscitation*. 2015 Oct 1;95:100-47.
14. Heckman L, Vijayaraman P, Luermans J, Stipdonk AM, Salden F, Maass AH, Prinzen FW, Vernooij K. Novel bradycardia pacing strategies. *Heart*. 2020 Dec 1;106(24):1883-9.
15. Sullivan BL, Bartels K, Hamilton N. Insertion and management of temporary pacemakers. In *Seminars in cardiothoracic and vascular anesthesia* 2016 Mar (Vol. 20, No. 1, pp. 52-62). Sage CA: Los Angeles, CA: SAGE Publications.