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Review Article

An overview of brady dysrhythmias in the emergency department

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ABSTRACT

Bradyarrhythmias, which include physiological and pathological disorders such as sinus node dysfunction and atrioventricular conduction disturbances are frequent clinical findings in the emergency department. Although some benign bradyarrhythmias do not need treatment, acute unstable bradycardia can lead to cardiac arrest. A thorough history and physical examination should cover potential causes of sinoatrial node dysfunction or atrioventricular block in patients with confirmed or suspected bradycardia. Based on the severity of the symptoms, the underlying causes, the presence of possibly reversible causes, the presence of negative indications, and the danger of asystole progression, bradyarrhythmias are managed. Bradyarrhythmias that are unstable or symptomatic are treated with medication and/or pacing. Bradycardia is described as an adult heart rate of less than 60 beats per minute. Syncope, presyncope, momentary light-headedness or dizziness, exhaustion, dyspnea with exertion, heart failure symptoms, or disorientation brought on by cerebral hypoperfusion are typical signs of bradycardia. Atropine plays a significant role in the management of bradyarrhythmias because it counteracts the impact of the parasympathetic vagus nerve system, which can cause an increase in heart rate. Atropine administration is indicated in cases with vagus-mediated asystole, obstructions in the atrioventricular node, and sinus bradycardia. Installation of temporary pacemaker, permanent pacemaker is also among the effective management strategies of the bradyarrythmias. Diagnosis and management of bradyarrythmias in emergency department is of utmost importance since it can prevent morbidity and mortality. The purpose of this research is to review the available information about an overview of bradydysrhythmias in the emergency department.

Keywords: Bradyarrhythmia, Bradycardia, Management, Emergency, Arrythmia

INTRODUCTION

All emergency practitioners will have to assess and manage patients with bradydysrhythmias during their practice. Bradydysrhythmias are a group of cardiac conduction anomalies that can appear as anything from moderate non-life-threatening emergencies to major life-threatening emergencies. These conduction anomalies' underlying etiologies are examined, including reflex-mediated bradydysrhythmias which include trauma-

induced ones and those with metabolic, environmental, viral, and toxicologic origins. Atropine, beta agonists, and transcutaneous or transvenous pacing may be needed when there is diminished end-organ perfusion. Patients who report to the emergency department with bradydysrhythmias must be evaluated for a variety of underlying diseases that result in these disorders. Cardiovascular etiologies should be the focus of assessments, but a thorough examination also must not ignore traumatic, cerebral, or intra-abdominal processes.[1] Bradycardia is described as an adult heart rate of less than 60 beats per minute. A heart rate of less than 60 bpm is regarded as symptomatic bradycardia if it causes signs and symptoms. The following symptoms of unstable bradycardia include hypotension, abruptly altered mental status, ischemic chest pain, and heart failure.[2]

The cardiac conduction system is susceptible to momentary or chronic malfunction. As a result, there may be a drop-in heart rate and symptoms from decreased cardiac output. Any part of the cardiac conduction system, such as the sinoatrial node, atrioventricular node, His-Purkinje system, and bundle branches, might experience delay or block. Ischemia, medication toxicity, and infection are a few reversible causes of bradycardia secondary to atrioventricular obstruction. In individuals who have a dysfunctional cardiac conduction system, permanent pacemaker implantation is frequently recommended. However, some patients may need assistance with temporary transvenous pacing or similar observation in locations with a higher level of care, such as an intensive care unit, coronary care unit, or intermediate care unit. Most patients who present to the emergency room with symptomatic bradycardia and an indication for permanent pacing can be treated in cardiac telemetry units.[3]

Syncope, presyncope, momentary light-headedness or dizziness, exhaustion, dyspnea with exertion, heart failure symptoms, or disorientation brought on by cerebral hypoperfusion are typical signs of bradycardia. A detailed and elaborated history, physical exam, examination of medications, and meticulous search for reversible reasons should all be done as part of the initial evaluation. A 12-lead electrocardiography should be performed on patients with bradycardia after the initial history and physical because it may detect structural heart disease, conduction abnormalities, or other cardiac disorders that could put them at risk for bradyarrhythmias. A diagnosis or symptom-rhythm connection can be made with ambulatory electrocardiographic monitoring and greater yields are obtained with longer-term observation.[4] For patients with compromised hemodynamics and life-threatening bradyarrhythmias, the temporary transvenous pacemaker insertion operation is crucial and one of the most frequent procedures carried out in the emergency room. Low heart rates in bradyarrhythmia patients result in decreased cardiac output and hypoperfusion of essential organs, which can cause symptoms like angina, syncope, dizziness, shortness of breath, angina, unstable hemodynamic state, and even sudden cardiac death. End-organ perfusion is maintained by reinstating the cardiac depolarization and guaranteeing efficient myocardial contraction with the insertion of a transvenous temporary pacemaker. Acute myocardial infarction, drug misuse and side effects, electrolyte imbalance, and idiopathic and degenerative illnesses of the conduction system are a few examples of heterogeneous situations that can result in unstable bradyarrhythmias that require temporary transvenous pacemaker placement.[5] The purpose of this research is to review the available information about an overview of bradydysrhythmias in the emergency department.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on November 16, 2022, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about an overview of bradydysrhythmias in the emergency department. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

The emergency physician must take into account a number of probable causes, including both ischemia and nonischaemic causes, while dealing with a patient who has bradycardia, while also attending to resuscitation and other care concerns. Acute myocardial infarction, profound hypoxia, overwhelming sepsis, and severe hypothermia are just a few examples of severe systemic events that should be given immediate consideration. Beyond these conditions, there is a wide range of differential diagnoses that can be divided into primary and secondary causes of bradyarrhythmias. In a wide series of unstable bradyarrhythmias treated in prehospital and emergency department settings, the fundamental causes, which account for only 15% of cases, are related to an inherent fault in the production or conduction of an impulse. Secondary causes include both ischemic and nonischaemic cardiovascular syndromes, which are caused by elements external to the heart conduction system.[6] There is always a chance that patients with bradydysrhythmias will arrive hemodynamically unstable or develop instability while undergoing evaluation and treatment. Emergency clinicians must be ready to deliver emergent interventions as necessary to stabilize these patients, even if these scenarios might not happen frequently. The key to management is having a working knowledge of operations, including the insertion of a transvenous pacemaker, as well as resuscitation methods and drugs.[1] Classification of bradyarrhythmias based on

the patient clinical characteristics is illustrated in (Figure 1).

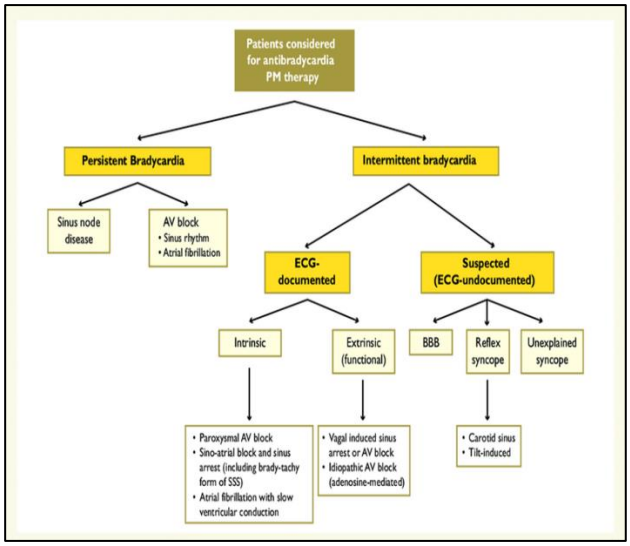

Figure 1: Classification of bradyarrhythmias based on the patient clinical characteristics.[18]

Reflection from literature

Understanding the hemodynamic effects of life-threatening arrhythmias is important for emergency management. In every situation, the patient's needs should come first and not just the electrical rhythm. When it is required, asymptomatic bradycardia should be referred for definitive care rather than addressed urgently. Bradycardia brought on by end-organ hypoperfusion should be treated with tests of atropine, isoproterenol, or emergency pacemaker implantation, and the stabilized patient should then be referred as needed to the cardiac procedure laboratory or cardiac care unit. Regardless of the anatomic and electrophysiologic causes of the reduced heart rate, bradyarrhythmias are treated in the same way. Given these few care guidelines, it is abundantly evident that the hemodynamic consequences of any arrhythmia rather than the arrhythmia itself should be given top priority.[7]

The patient's medication history should be examined for any potential bradycardia-causing medicines, and those medications should ideally be stopped while care decisions for a patient with sinus bradycardia are being determined. A patient may qualify for a permanent pacemaker if he or she has coexisting diseases that make it necessary for him or her to take specific medications, which may be the cause of the sinus bradycardia. When it is possible to stop using a medication, the patient may be assessed for a permanent pacemaker if symptoms and heart rate do not improve.[8] Electrocardiographic monitoring should be used on patients who have bradycardia and adequate perfusion at the time of their initial presentation. Patients with bradyarrhythmias and systemic hypoperfusion should be given standard dosages of atropine intravenously and 0.6 mg to 1.0 mg intravenously should be administered for such bradyarrhythmias with systemic hypoperfusion, with repeat bolus as necessary up to the vagolytic dose. In cases of unstable bradycardia where atropine eventually succeeds in bringing the vital signs back to normal.[9]

Results of a retrospective study showed that syncope, fainting, collapsing, angina, and dyspnea/heart failure were the main symptoms of bradyarrhythmia. The initial electrocardiograph revealed pacemaker failure, high grade atrioventricular block, sinus bradycardia/ atrioventricular block, sinoatrial arrest, and bradycardic atrial fibrillation. Primary disturbances of cardiac automaticity and/or conduction, negative medication effects, acute myocardial infarction, pacemaker failure, drunkenness, and electrolyte imbalance were the underlying reasons. Bed rest helped 39%patients to get rid of their ailments, 61% patients received intravenous medications to raise heart rate; 20% needed extra temporary transvenous/transcutaneous pacing. Cardiac bypass was needed in two individuals. In 50% patients, permanent pacemakers were installed. At 30 days, reported mortality rate was 5%. For initial stabilization, roughly 20% of patients who presented with compromising bradycardia required temporary emergency pacing; in 50% of cases, permanent pacing had to be instituted.[10] Results of another retrospective study showed that a partial or full response to treatment was seen in almost half of the patients who received atropine for compromised rhythms in the prehospital setting. Adverse reactions were rare. When compared to patients with atrioventricular block, those who reported to emergency care staff with hemodynamically unstable bradycardia responded more frequently to a single dosage and a lower overall dose of atropine. Patients who return to normal sinus rhythm by emergency department discharge were probably able to do so before they arrived at the hospital.[11]

The patient should be sent to the emergency room if 12-lead ECG reveals second-degree atrioventricular block, type I or type II, or third-degree atrioventricular block, especially if the patient just experienced a syncopal episode. Once in the emergency room, secondary reasons such hypoxemia, irregular electrolytes, hypothyroidism, or drug toxicity should be tested out. However, in elderly individuals, these reasons hardly account for sinus node dysfunction or atrioventricular block. The principal electrical system process that causes this disease is often age and conduction system fibrosis. If the patient is on digoxin. It is obvious that older patients with bradycardia may have high digoxin levels.[12] Various bradyarrhythmias can manifest in patients. It is critical to recognize and address those that can be reversed. Many patients need permanent pacemaker implantation because of the permanent malfunction, and others may also need transient transvenous pacing. When medically possible, permanent pacemaker implantation can be completed in the majority of patients within 2 days. However, in some clinical circumstances, delaying permanent pacemaker

implantation may be fair and required, which is not linked to an increase in adverse events. Weekend permanent pacemaker implantation should be taken into consideration in certain circumstances to lessen the requirement for temporary transvenous pacing, decrease duration of stay, and lower hospital costs even though most hospitals may not offer 24/7 electrophysiology services coverage.[3]

Transcutaneous cardiac pacing is a fast, convenient, non-invasive ventricular stimulation that conscious patients can tolerate better than other methods as compared to patients who are contrasted with invasive treatments for unstable bradycardias. Intravenous infusions of beta-adrenergic agonists with rate-accelerating effects are used to treat unstable bradycardia that is not responsive to atropine. While the patient is set up for emergency transvenous temporary pacing, if necessary, dopamine, epinephrine or transcutaneous cardiac pacing can be successful. Transcutaneous cardiac pacing should also be initiated immediately for unstable patients, especially those with a strong block such as Mobitz type II, second degree or third degree. Transcutaneous cardiac pacing is a clinically viable therapeutic option for individuals with atropine-resistant unstable bradycardia.[13]

Inoue et al reported that there have been three cases of bradyarrhythmia with significant extracardiac organ disease. In first case, the sinuses were ill. He arrived at the hospital with a syncope complaint and afterwards developed apnea while second case was admitted with severe liver failure and a full atrioventricular block. Paroxysmal atrioventricular block was present in the third case. He reported experiencing syncope after having convulsions. Their symptoms could be the result of circulatory shock brought on by the ventricle's lax lower pacemaker. The malfunction of the extracardiac organs was satisfactorily addressed by emergency temporary pacing. Permanent pacemakers were installed despite the fact that their bradyarrhythmias were temporary to prevent recurrence. In patients with critical bradyarrhythmia, a rapid temporary pacemaker should be recommended for survival.[14] White et al revealed in their study findings that prehospital cardiac arrest patients who had bradyasystolic rhythms about 25% of the time have very poor long-term prognoses. The study included 20 individuals who presented with bradyasystolic prehospital cardiac arrest. All of them were given the standard advanced cardiac life support treatment in addition to being externally paced right away with a pacemaker and automated external defibrillator, 2 out of the 20 patients had signs of electrical capture, and no one else experienced pacing-induced pulses. During resuscitation, 4 out of the 20 patients experienced sinus rhythm and blood pressure development. Three people made it out of the emergency room, but no one made it out of the hospital. On some occasions, bradycardia and pulseless idioventricular rhythms increased in frequency without being affected by electrical capture or medication. Although the automated external defibrillator and pacemaker did not improve survival, they were dependable, simple to operate, and error-free.[15]

Results of a prospective cohort study showed that chest compressions were administered to a total of 1853 patients for bradycardia/poor perfusion as opposed to 1489 patients for asystole/pulseless electrical activity. Overall, 755 of 1353 patients with bradycardia and 365 of 1489 patients with asystole/pulseless electrical activity were able to leave the hospital. Cardiopulmonary resuscitation for bradycardia with inadequate perfusion was linked to an improved survival to hospital discharge after adjusting for known variables. Children who had chest compressions started for bradycardia and poor perfusion before to the onset of pulselessness had a higher chance of surviving until discharge than children who had chest compressions started for asystole or pulseless electrical activity.[16] Since the parasympathetic vagus nerve system's primary effect on the heart is to slow it down atropine helps majorly in bradyarrythmias management as it prevents this effect, which could lead to an increase in heart rate. Vagus-mediated sinus bradycardia, blockages in the atrioventricular node, and vagus-mediated asystole are indications for atropine administration. The typical atropine dosage is 0.5-1.0 mg intravenously every 3-5 minutes, with a maximum dose of 0.04 mg/kg (3 mg), for symptomatic bradycardia or when there is asystole. Additionally, atropine can be used to treat third-degree heart blocks with a strong Purkinje or atrioventricular-nodal escape rhythm as well as second-degree atrioventricular blocks. However, with a low Purkinje or ventricular escape rhythm and third-degree heart block, it is typically ineffective.[17] Despite of being a significant prevalent condition in the emergency department studies addressing the management of bradydysrhythmias in literature are quite limited and scarce hence advocating strong need of further clinical research which can also aid in development of effective management strategies of bradyarrythmias generating optimal outcomes.

CONCLUSION

For individuals with an unstable bradyarrhythmia, diagnosis and management in the emergency department is essential. By minimizing the amount of time that essential organs are exposed to hypoperfusion, timely diagnosis and management of bradyarrythmias is able to lower mortality, especially in critical patients at the time of admission in the emergency department.

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REFERENCES

1. Deal N. Evaluation and management of bradydysrhythmias in the emergency department. Emergency Med Pract. 2013;15(9):1-15.

2. Rujichanuntagul S, Sri-On J, Traiwanatham M. Bradycardia in Older Patients in a Single-Center Emergency Department: Incidence, Characteristics and Outcomes. Open access emergency medicine : OAEM. 2022;14:147-53.
3. Abrich VA, Le RJ, Mulpuru SK. Clinical Outcomes of Various Management Strategies for Symptomatic Bradycardia. Clin Med Res. 2020;18(2-3):75-81.
4. Sidhu S, Marine JE. Evaluating and managing bradycardia. Trends Cardiovascular Med. 2020;30(5):265-72.
5. Senturk B, Kucuk S, Vural S, Demirtas E, Coskun F. Bedside Temporary Transvenous Pacemaker Insertion in the Emergency Department: A Single-Center Experience. Sisli Etfal Hastanesi tip bulteni. 2021;55(3):359-65.
6. Brady WJ, Harrigan RA. Evaluation and management of bradyarrhythmias in the emergency department. Emergency Med Clin N Am. 1998;16(2):361-88.
7. Hoffman JR. Emergency department management of life-threatening arrhythmias. Emergency Med Clin N Am. 1986;4(4):761-74.
8. Neumar RW, Otto CW, Link MS. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010;122(18-3):S729-67.
9. Brady WJ, Harrigan RA. Diagnosis and management of bradycardia and atrioventricular block associated with acute coronary ischemia. Emerg Med Clin N Am. 2001;19(2):371-84.
10. Sodeck GH, Domanovits H, Meron G. Compromising bradycardia: management in the emergency department. Resuscitation. 2007;73(1):96-102.
11. Brady WJ, Swart G, DeBehnke DJ, Ma OJ, Aufderheide TP. The efficacy of atropine in the treatment of hemodynamically unstable bradycardia and atrioventricular block: prehospital and emergency department considerations. Resuscitation. 1999;41(1):47-55.
12. Byrnes TJ, Costantini O. Tachyarrhythmias and Bradyarrhythmias: Differential Diagnosis and Initial Management in the Primary Care Office. Med Clin N Am. 2017;101(3):495-506.
13. Bektas F, Soyuncu S. The efficacy of transcutaneous cardiac pacing in ED. Am J Emergency Med. 2016;34(11):2090-3.
14. Inoue T, Takayanagi K, Sakai Y, Hayashi T, Morooka S. Cardiac pacing as emergency care for serious bradyarrhythmia with circulatory shock. J Med. 2001;32(5-6):321-31.
15. White JM, Nowak RM, Martin GB, Best R, Carden DL, Tomlanovich MC. Immediate emergency department external cardiac pacing for prehospital bradyasystolic arrest. Ann Emergency Med. 1985;14(4):298-302.
16. Donoghue A, Berg RA, Hazinski MF, Praestgaard AH, Roberts K, Nadkarni VM. Cardiopulmonary resuscitation for bradycardia with poor perfusion versus pulseless cardiac arrest. Pediatrics. 2009;124(6):1541-8.
17. Trappe HJ. Concept of the five 'A's for treating emergency arrhythmias. J Emergencies, trauma, shock. 2010;3(2):129-36.
18. Brignole MA, Barón-Esquivias G. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: The Task Force on Cardiac Pacing and Resynchronization Therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Europace. 2013;15.

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