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Mini review on approach, teaching aspects, and common pitfalls in dealing with bradyarrhythmia

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Abstract

Bradyarrhythmias are of a wide variety and are generally considered common. While some bradyarrhythmias are benign and do not necessitate any medical intervention, some might be fatal thus require immediate emergency department actions; or otherwise, cardiac arrest and death may ensue. The key to the management of bradyarrhythmias in the emergency department lies in its early identification by a thorough history, physical examination, and ECG interpretation. Accordingly, the management plan is reflected by the presence of symptoms and their severity, vital signs status of the patients, ECG patterns, underlying causes, and possible reversibility. As medical students learn the theoretical and practical approaches to the management of bradyarrhythmias in the Emergency Department (ED), there are common pitfalls that they encounter, and those pitfalls could be serious and lethal in some instances. Knowledge about the correct way of assessing, diagnosing, and treating bradyarrhythmia patients, and avoiding the common pitfalls that they may face is critical. This paper aims to review the definition of bradyarrhythmia, its various classification, the clinical approach to the condition according to the approved guidelines, and how to subsequently manage the condition. Additionally, our study aims to guide the appropriate response on dealing with bradyarrhythmia patients, by determining the priorities of management and assessing the clinical presentation based on the stability of the patient, and by providing a training protocol for Emergency Physicians. In this review article, a literature search on bradyarrhythmia was performed in terms of methodology, diagnosis, and management. The MeSH included these keywords: “Bradycardia” OR “Bradyarrhythmia” AND “Prevalence” OR “ECG Interpretation” OR “ACLS” OR “Bradyarrhythmia Protocols”.

Keywords: Bradyarrhythmia, management, pitfalls in teaching bradyarrhythmia, ER/ED guidelines

Introduction

Bradyarrhythmia, in alignment with the National Institute of Health (NIH), is defined as a heart rate that is slower than 60 beats per minute (bpm) in adults other than well-trained athletes [1–3]. However, in routine clinical practice, the heart rate starts to raise a concern when it falls below 50 bpm. That cutoff is used to define bradycardia by American Heart Association (AHA) [1]. The symptoms vary depending on the cause of underlying arrhythmia; but generally, palpitations, skipped pulses, bouncing heart, weakness, and fatigue are all common presentations in patients. Fortunately, most of the cases are usually vanish without any medical care. However, blurred vision, chest pain, difficulty breathing, and foggy thinking, should be considered alarming signs of serious arrhythmias. Common symptoms that warrant a thorough workup also include syncope, presyncope, heart failure symptoms, or confusion that result from cerebral hypoperfusion [3]. The first goal of the physician is to stabilize the patient if he or she is found to be in an unstable hemodynamic condition, followed by identification of the type of the arrhythmia, via taking a complete and comprehensive history, and obtaining ECG readings during the episode. So that the appropriate management protocol could be applied accordingly [1].

As will be subsequently discussed, bradyarrhythmia in all its variants is quite a common presentation in the ED, hence, it is crucial for all doctors in the frontline (e.g., emergency doctors, residents, and general practitioners) to be able to identify, diagnose, and manage bradyarrhythmia [4].

Some common pitfalls in identifying the specific type of arrhythmia may lead to undesirable outcomes, ranging from misdiagnosis,

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delayed or inappropriate treatment, and sudden cardiac death [5,6]. Due to these reasons, a systematic approach and specific algorithm are always necessary to be kept in mind by caretakers to avoid those pitfalls to avoid devastating outcomes. The algorithm for the approach and management of bradyarrhythmia is discussed in a subsequent section.

Bradyarrhythmia is one of the essential presentations in the ED, as we will discuss later the incidence and prevalence of the most common types of arrhythmias. All ED physicians must know the details of the presentation, approach to patients, and management algorithms including the common pitfall or mistakes that they may experience when they first see a case. This stems from the fact that some arrhythmias, if not treated properly or identified quickly, will lead to the development of multiple complications including syncopal attacks, strokes, heart failure, cardiac arrest, and sudden death [5-7].

For this reason, in our review, we will look into the incidence, presentations, ED approach, and the steps of management for the common presentations of bradyarrhythmias. Also, we will look into the common pitfalls and mistakes that both medical students and new physicians face in the recognition, diagnosis, and management of this condition, all the while developing recommendations that may help those physicians and a checklist for skills examination for medical students to be tested with.

Methodology

In this review article, we examined contemporary research papers on bradyarrhythmia in terms of methodology, diagnosis, and management. The mesh included these terms (Bradycardia) OR (Bradyarrhythmia). The papers were chosen based on whether or not they included one of the following topics: Bradycardia, Bradyarrhythmia, Clinical characteristics, Diagnosis, and Management. All other articles that did not fulfill one of these themes as their major endpoint met the exclusion criteria.

In this review paper, we have looked through the recent literature that discussed bradyarrhythmia's in the approach, diagnosis, and step of management. We also used textbooks and articles that discussed the difficulties of this condition's approach in the ED.

The literature search was based on the available data related to bradyarrhythmias, which include Medical Subject Headings (MeSH) terminologies. The MeSH terminologies in our review included these keywords: "Bradycardia" OR "Bradyarrhythmia" AND "Prevalence" OR "ECG Interpretation" OR "ACLS" OR "Bradyarrhythmia Protocols".

Previous studies have suggested that bradyarrhythmias may be an initial presentation of underlying serious and emergent pathologies in the patients. A review of the most recent definition, etiologies, and therapeutic options were included in the literature search.

The aims and outcomes of the study are to discuss the appropriate initial evaluation and assessment of a patient with suspected bradyarrhythmias based his/her hemodynamic and clinical stability and to recognize any potentially life-threatening arrhythmias to convey life-saving algorithmic strategies. "A stable heart is timed," is the central theme here.

Bradyarrhythmia As An Emergency Phenomenon

Bradyarrhythmia is not always of pathological origin; it can occur in healthy individuals, such as during deep sleep. It is also frequently found in certain normal individuals, such as in people with an enhanced vagal tone or well-conditioned professional athletes. In those people, the arrhythmia does not manifest clinical symptoms, thus is often called asymptomatic bradyarrhythmia. On the other hand, when bradyarrhythmia occurs in a diseased heart, it may indicate a potentially life-threatening pathology. It is the most common rhythm disturbance seen in the early stages of acute myocardial infarction. Here the bradyarrhythmia is often called symptomatic bradyarrhythmia [7,8].

Bradycardia is a common presentation in the ED. Its frequency among the general population is unknown. however, a new study of about half a million individuals estimated it to be around 2%, although, in this group, it tends to be associated with a benign cause. The prevalence increases significantly in the elderly population and patients with underlying cardiac abnormalities, ranging from about 4%. in the aging group, bradycardia may be a sign of underlying pathology, especially when symptomatic [4].

Review of Heart Electrophysiology and Mechanism of Bradyarrhythmias

In healthy individuals, the heart normally fires at a rate of 60-100 bpm. This pace is achieved thanks to the sinoatrial (SA) node, which is composed of a collection of cells that initiate heartbeat called pacemaker cells. It is normally located at the right atrium adjacent to the superior vena cava (SVC). If the SA node – due to any reason – could not initiate a beat (asystole), or in case its impulse generation is less than 60 bpm (decreased automaticity), the other pacemakers, such as the atrioventricular (AV) node, may override the function of SA node to generate the heart impulse. However, the baseline impulse-generating rate of the AV node is generally lower than the SA node (40-60 bpm), resulting in clinical bradyarrhythmia. Other pacemaker cells include the bundle of His, branches of His bundle (right and left branch), and the Purkinje network, which generates an impulse rate of around 25-40 bpm (See: Table 1). The various causes underlying decreased automaticity with possible subsequent escape beat/rhythms are summarized in Table 2.

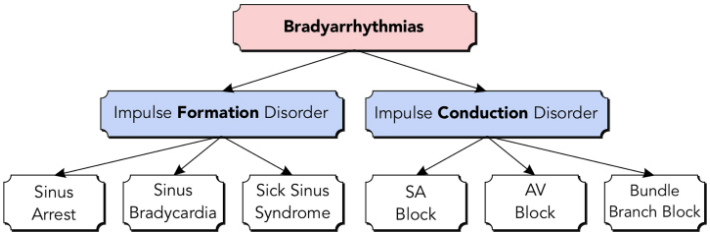

Figure 1. Classification of Bradyarrhythmias by Electrophysiologic Changes into 2 categories, i.e., those that occur due to impulse formation disorder and impulse conduction disorder. SA, sinoatrial; AV, atrioventricular

The escape beat usually ends as the SA node fires back with the start of a new beat at a normal rate. In case the SA node fires back after one escape beat, the arrhythmia is termed sinus arrest with escape beat. Escape rhythm, however, is described when the escape beat is sustained or prolonged. The occurrence of six or more consecutive junctional AV node beats defined AV junctional

escape rhythm. The reason behind the slower pace lies behind the architecture of the heart. It is designed to let the fastest pacemaker cells beat, and the rest of the cells be subsequently activated as result. The sinoatrial (SA) node is the fastest pacemaker of the heart, so, when it stops firing, a slower pacemaker will take control, but at a slower pace.

Table 1. Pacemaker Cells and Their Intrinsic Impulse-Generating Rate

Activation Sequence	Pacemaker Cells	Pacemaker Rate (bpm)
1	SA Node	60-100
2	AV Node (Junctional)	40-60
3	Bundle of His	25-40
4	Bundle Branches	25-40
5	Purkinje network	25-40

Another mechanism by which bradyarrhythmia occurs is the presence of impaired conduction of the propagating impulse through the cardiac pacemaker systems. The classical example of such electrophysiologic changes is a condition called AV block, which occurs when the conduction of electrical impulse is impaired through the atria, AV node, or proximal His-Purkinje fibers. On the appearance of ECG paper strips, these electrophysiologic changes are apparent by either prolongation of PR interval (normally range between 0.10-0.20 seconds), the absence of subsequent waves following P wave; indicating failure of propagation of electrical impulse, or even asynchrony between the P wave and QRS complex, a condition called AV dissociation. The causes of conduction disorders are numerous and are summarized in Table 2. Its classification and management plan are discussed in the subsequent section [7–9].

Table 2. Conditions Associated with Bradycardia and Conduction Disorders [1]

Intrinsic	Cardiomyopathy Congenital Heart Disease Degenerative Fibrosis Infection/inflammation (e.g., Chagas disease, infectious endocarditis, myocarditis, etc.) Infiltrative Disorders (e.g., amyloidosis, hemochromatosis, lymphoma) Ischemia/infraction Rheumatological conditions (e.g., scleroderma, systemic lupus erythematosus) Surgical or procedural trauma
Extrinsic	Autonomic perturbation (e.g., carotid sinus hypersensitivity, physical conditioning) Sleep (with or without sleep apnea) Metabolic (e.g., acidosis, hyper/hypokalemia, hypothermia, hypothyroidism) Iatrogenic (e.g., antihypertensive, antiarrhythmic, psychoactive, others)

Clinical Approaches to Bradyarrhythmias in Emergency Department

When a patient comes to the Emergency Department with clinical features of bradyarrhythmias, the initial evaluation is dependent upon the hemodynamic stability of the patient. In a hemodynamically stable patient, the care should start with a thorough historical analysis and physical examination, which is then followed by 12-lead ECG tracing. As AHA defines symptomatic bradycardia 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 resulting from cerebral hypoperfusion attributable to slow heart rate", the physicians should be able to make it clear that the clinical features arise from underlying bradyarrhythmia, which is practically challenging [10]. The history of complaints should include the onset, frequency, timing, duration, severity, precipitating and aggravating factors, current medications are taken, association with emotional distress or certain positions, and history of any cardiac diseases. In addition to analyzing the patient's complaints, history and physical examination should also direct the physicians toward identifying the possible underlying cause of bradyarrhythmia (see: Table 2), as it might be the first clinical manifestation of systemic disease [1].

The vital signs should be assessed during physical examination, as well as a thorough cardiovascular examination. Importantly, a slow radial pulse should be correlated with precordial or carotid pulsation by palpation and auscultation, as some rhythms such as premature ventricular or atrial complexes might be mistaken as bradyarrhythmia due to inadequate stroke volume generated by ectopic beats. It may also identify possible cardiac pathologies by the presence of S3 or S4 gallops or cardiac murmurs. Endocrinologic assessment may also be appropriate in certain individuals, especially when the clinical features point toward specific endocrinologic causes, e.g., pretibial edema, cold intolerance, and weight gain despite anorexia in cases of hypothyroidism. Physical examinations should also assess the evidence of end-organ hypoperfusion, as it helps to both assess the severity of clinical presentation (impending shock, such as confusion, agitation) and clues to underlying arrhythmias, such as cardiac failure.

12-lead resting ECG should be the next initial investigation of choice once bradyarrhythmia is suspected. It is important to document the rhythms, rate, and to screen for structural heart or systemic diseases. Surprisingly, however, even in those who present with a syncopal attack with documented bradycardia by physical examination, an initial ECG only provides a diagnosis in 5% of cases. It is even lower in milder clinical features. When found, however, abnormal initial ECG is predictive of adverse prognosis, especially with documented structural abnormalities. The systematic methods to analyze the ECG tracing are discussed below [1].

Although exercise ECG has been described in some literature as the next investigation for evaluation of suspected symptomatic bradyarrhythmias and might be useful in selected patients (e.g., patients with chronotropic incompetence or those with exercise-aggravated symptoms, it is not part of the investigation performed in the emergency setting thus beyond the scope of this paper.

Imaging studies may be warranted in some cases of bradyarrhythmias, particularly newly found LBBB and second and third-degree AV blocks regardless of structural cardiac diseases or coronary artery diseases (CAD); or in any other bradyarrhythmias when structural cardiac diseases are of concern. Transthoracic echocardiography (TTE) is usually the initial imaging of choice in

the emergency setting for that purpose. The finding might include different types of cardiomyopathies, valvular heart diseases, congenital anomalies, infiltrative processes, or other structural pathologies.

TTE may also be of values to dictate the management plan. This is particularly true for instance in the case of newly-onset LBBB where the presence of cardiac failure with reduced ejection fraction will determine the need for resynchronization therapies. In addition, it can also dictate the prognosis in some cases. Further imaging investigation with CT scan or MRI may be indicated once the resuscitative measures have succeeded with the return of hemodynamic stability and symptoms improvement, and is done outside the emergency department setting, thus beyond the scope of this topic.

In hemodynamically unstable patients (altered sensorium, hypotension, syncope, chest pain, and respiratory distress), the focus should be on resuscitation and stabilization of hemodynamic parameters after primary survey assessment, followed by complete secondary surveys with complete history taking and thorough physical examinations. Primary surveys consist of rapid history and assessment of vital signs, followed by 12-lead ECG applications with immediate management by ACLS protocol for bradyarrhythmia (the management is discussed in "Basic Emergency Management of Bradyarrhythmias").

Classification Of Bradyarrhythmias

There are different ways by which bradyarrhythmia can be classified. AHA classifies bradyarrhythmias into 3 main categories based on pathophysiologic changes in the cardiac pacemakers, i.e., sinus node dysfunction (SND), AV Block, and Conduction Tissue Disorders (with 1:1 AV Conduction). Others classify into 2 main categories, i.e., impulse conduction disorders – which include SA block, AV block, and bundle branch block (BBB) –, and impulse formation disorder – which include sinus bradycardia (see: figure 1). Regardless of the different ways of classification, each model has a resemblance to each other and somehow has overlapping subclassification entities.

For the descriptive purpose of this paper, the next following paragraphs will be structured according to 2 main classification systems based on electrophysiologic changes described above, i.e., impulse formation disorders and impulse conduction disorders.

Impulse Formation Disorders

A. Sinus Bradycardia

Sinus Bradycardia occurs when the SA nodes fire at a slower pace than normal, usually around 45-59 bpm. AHA describes sinus bradycardia under the SND category and defines its cutoff heart rate (and thus the R-R interval) as less than 50 bpm. Although it may be considered physiological, e.g., in young healthy professional athletes or those with a high vagal tone, it is necessary to exclude the alarming symptoms associated with it. It is also prudent to exclude possible underlying conditions such as hypothyroidism or the presence of cardiac pathologies (see Table 2). Keep in mind that the beating source remains from the SA node, as there is no escape beat arising from the bradycardia [1,7,8].

B. Sinus Arrest

Sometimes, the sinus node may fail to initiate a pulse. This is termed sinus arrest. On ECG, it appears as the absence of P waves with its subsequent waves. As aforementioned, when the SA node fails to initiate a pulse, cardiac tissue may compensate for the arrest by the firing of the second-fastest pacemaker, the AV node, which eventually drives the heart rhythm at a slower rhythm. This phenomenon is known as an escape beat. Escape beats are then terminated when the sinus node regains its function again, which should usually occur after 2-3 escape beats [1,7,8].

C. Sick Sinus Syndrome

Sick Sinus Syndrome, also known nowadays as sinus node dysfunction (SND), is commonly encountered in the elderly population, mostly at 70-80 years of age as the pathophysiology is due to age-dependent progressive fibrosis of the SA node and its surrounding myocardial tissue. This results in both impulse formation and conduction being impaired. Subsequently, this may create various types of arrhythmias, including sinus bradycardia, sinus arrest, or conduction disorders such as SA exit block. The conduction impairment, such as AV block, may occur due to fibrotic involvement of AV nodes or proximal His bundle, which may be clinically apparent after atrial pacing for SND. In addition to bradyarrhythmias, tachyarrhythmias may sometimes occur, including atrial fibrillation. As such, both dysrhythmias may coexist, resulting in "tachy-brady syndrome". It is possible to concurrently identify the presence of cardiac diseases, e.g., valvular heart diseases, ischemic heart diseases, atrial fibrillation, and others, as these conditions are also age-dependent [1,7,8].

Impulse Conduction Disorders

A. SA Block

The underlying mechanism of SA block, in contrast to impulse formation disorder, is a failure of the SA node impulse to propagate beyond the SA node. Histologically, the SA node consists of two main groups of cells. Pace-making (P) cells are the ones creating the electrical impulse, whereas transitional (T) cells transmit the impulse into the atria. The failure of T cells to transmit some of the impulses thus results in sinus block.

Consequently, when this occurs, the atrial tissue and beyond do not receive any signal from the sinus node.

Some literature classifies the SA node into three degrees (See: table 3). First-degree SA blocks are indistinguishable on ECG and need electrophysiologic testing to be diagnosed. Second-degree SA blocks are further divided into two subtypes, namely: Mobitz type I, and Mobitz type II. In Mobitz type I, the PP interval keeps shortening, until eventually, one P wave drops. Note that the PP interval meant here is different from than PR interval that we will discuss later on In AV blocks; in SA blocks, the PR interval remains fixed. Mobitz type II is characterized by dropped P wave preceded by a fixed PP interval. Third-degree blocks are composed of prolonged SA node pauses, in that sense, it is hard to differentiate between it and Sinus Arrest [1,7,8].

Table 3. Degrees of SA Block and their ECG Appearance

	ECG appearance
1st degree	• Unidentifiable by ECG. • Electrophysiology is needed • Note: in the 1st degree, there is a delay between impulse generation and transmission to the atrium
2nd degree (Mobitz type 1)	• The progressive lengthening of PP interval • Accompanied by one lost P wave at the end of each group • Before dropped P wave, there is a progressive shortening of the P-P interval
2nd degree (Mobitz type 2)	• One dropped P wave with no lengthening of P-P lengthening • The lost P wavelength is twice the length of the preceding P-P interval
3rd degree	• Complete absence of P waves, a possible junctional escape rhythm • May produce long sinus pauses or sinus arrest • Difficult to distinguish it from sinus arrest
Note: There is no conducted impulse from the SA node to the right atrium	

B. AV Block

Analogous to the SA block, AV block is the presence of impaired conduction of electrical impulse, but in this case at any level from the SA node up to the terminal Purkinje fibers. It is also classified similarly to SA blocks into 3 degrees (see: Table 4). Their identification and diagnosis principally depend on the relationship between the P wave and the QRS complex (P-R interval). First-degree AV block is characterized by a prolonged delay in conduction in the AV node, and it is diagnosed by a PR interval of longer than 0.20 seconds (normal 0.12-0.20 sec). First-degree AV blocks may be a common finding in healthy hearts; however, it can be an early sign of a plethora of heart problems, such as degenerative diseases of the conduction system or drug toxicity. In the second-degree AV blocks, not every P wave is followed by a subsequent QRS complex, thus P wave to QRS complex ratio is greater than 1:1. There are two types of second-degree AV blocks; Mobitz type I (also known as the Wenckebach phenomenon) and Mobitz type II. Wenckebach phenomenon is unique in that it presents progressively increasing delays between the P wave and the QRS complex until one impulse fails to conduct (usually the third or fourth beat). On the contrary, in Mobitz type II AV block, there is no progressive lengthening of the PR interval, but one impulse fails to conduct (sudden dropped beat). Lastly, the third-degree AV block resembles complete independency (dissociation) between the P wave and QRS complex, in that a P wave will conduct by itself but will not activate a QRS complex, and QRS complexes will be self-activated at a slower pace [1,7,8].

Table 4. Degrees of AV Block and Their Characteristic ECG Appearance

	Description and ECG Appearance
st degree	P-R interval of longer than 0.2 second
2nd degree (Mobitz type 1)	The progressive lengthening of the PR interval, accompanied by one lost P wave, or P to QRS complex ratio of higher than 1:1
2nd degree (Mobitz type 2)	One dropped P wave without progressive P-R interval lengthening
3rd degree	Complete AV dissociation (P and QRS interval beat independent of each other

C. Bundle-Branch Block

Another type of serious bradyarrhythmia is bundle branch blocks (BBB). It refers to a conduction block in either the left or right bundle branches. In AHA Guidelines, it is described under the category of conduction disorder with 1:1 AV conduction. The common types include right BBB (RBBB) and left BBB (LBBB). With RBBB, conduction through the right bundle is obstructed, so it will not be activated by the normal pathway (SA node to terminal Purkinje). However, it will be activated as soon as the left bundle gets activated, the left bundle will then transmit a signal that will activate the right bundle. Because of this, the QRS complex will assume a different, rather diagnostic shape in leads V1 and V2. In lead V1, the QRS complex forms the pattern of rSR', while in lead V2 it forms take the shape of broad and deep S waves shown as (qRs) (see: Table 5)

LBBB follows a similar pathophysiological mechanism as the right bundle, i.e., blockage of the bundle branch; but this time, it is the left bundle that is blocked. QRS complexes make the shape of 'M' in lead V1 representing (sR), and the shape of 'W' representing (RR) in lead V6.

In contrast to RBBBs which might be normal in some individuals, LBBBs rarely occur in normal hearts. It usually represents significant cardiac disease, such as ischemic coronary artery disease (CAD) [1,8].

Table 5. Summary of classical QRS pattern of bundle branch block on lead V1 and V6. LBBB, left bundle branch block; RBBB, right bundle branch block

	Lead V1	Lead V6
Normal	rS	qR
RBBB	rSR'	qRs
LBBB	rS	R

Basics Emergency Management Of Bradyarrhythmias

After an initial assessment in the primary survey, management should be governed by the presence of clinical instability (see: figure 2 and 3), i.e., the presence of hypotension, altered sensorium, chest pain, signs of shock, and acute heart failure (e.g., dyspnea and peripheral edema) [11]. Other considerations may include severe bradycardia and ventricular rates less than 30 bpm as they may be at increased risk of torsade de pointes, thus necessitating management and close monitoring closely. Continued syncope accompanied with bradycardia or syncope on exertion, and a family history of hereditary arrhythmia are all possible grounds for hospitalization [12].

The first step in the management is admission to the emergency department followed by resuscitation according to ACLS protocols, initially by maintaining a patent airway with breathing assistance measures if necessary. Oxygen may be administered if the patient is dyspneic. 12-lead ECGs should be applied if applicable as long as it does not interfere with the therapeutic plan. Importantly, IV access should be established early before progression to shock as it may render the patient difficult to obtain IV access, which necessitates more invasive access, such as by venous cut-down or intraosseous approach [13,14].

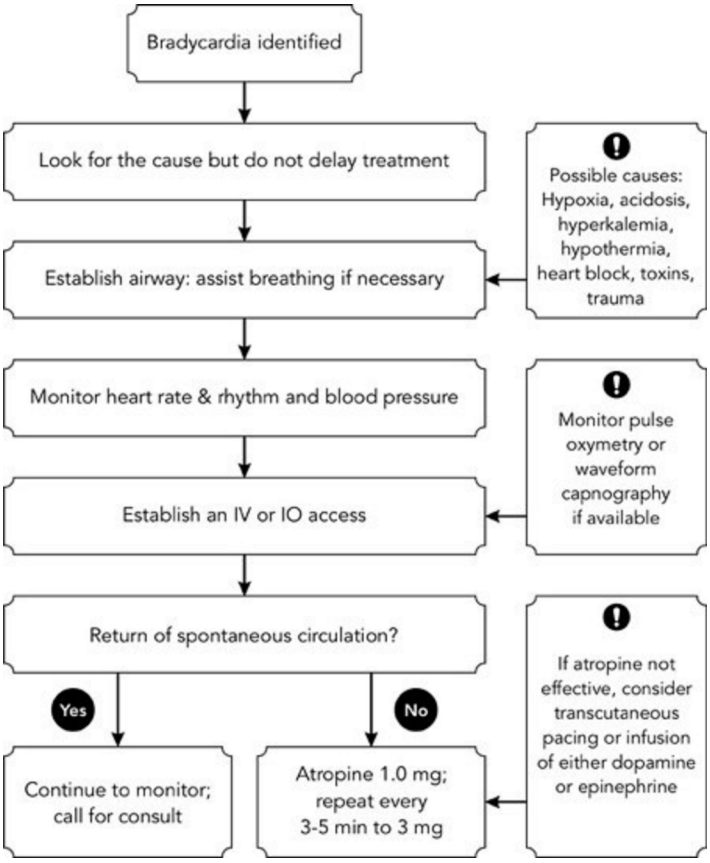

Figure 2. Algorithm of Emergency Management of Bradyarrhythmias. Modified from ACLS Guidelines 2020

The first medical therapy is IV atropine, an anticholinergic agent that blocks muscarinic acetylcholine receptors. It has a half-life of approximately 2 hours when administered intravenously. In the adult, the first dose of 1 mg IV bolus is initially administered. The maximum dose should not exceed 3mg and each 1mg dose should be given in 3-5 minutes intervals. If atropine is not proven effective by the reversal of hemodynamic parameters, mainly heart rate, the next step should be taken by either transcutaneous pacing or medical therapy.

The next medical therapy after a 3 mg dose of atropine is either dopamine or epinephrine. Dopamine is administered by IV infusion at a rate of 5-20 mcg/kg per minute. In contrast to its vasodilatory effects when given in a low dose (1-2 mcg/kg/min), a higher dose of 5-20 mcg/kg/min promotes chronotropy and inotropy of the heart. Despite its effectiveness, judicious use should be considered as it is associated with vasoconstriction and proarrhythmic effects, notably when it exceeds 20 mcg/kg/min. Alternatively, epinephrine may be administered by infusion of 2-10 mcg per minute. This catecholamine acts by stimulating both alpha- and beta-adrenergic receptors, with resultant effects of increased chronotropy and inotropy, as well as blood pressure and myocardial oxygen consumption. It needs to be titrated according to the hemodynamic parameter.

Temporary pacemaker by either transcutaneous, transesophageal, or transvenous route electrodes may be indicated in select cases. ACLS protocols support the use of transcutaneous pacing as initial management after atropine administration has failed to improve the condition. AHA stated that transcutaneous pacing should be considered to improve heart rate until either the bradycardia episode is resolved or a permanent pacemaker or transvenous pacemaker is placed (see Figure 3). Its use in SND is, however, infrequent, due to its low association with hemodynamic instability. The need for anxiolytic and analgesia may be considered in those who are conscious before placement of a transcutaneous pacemaker [1].

Transvenous pacing or cardiologic expert consultation should be done if neither of the aforementioned measures improves the patient's clinical presentation. Temporary pacemaker indication is generally similar to permanent pacemaker placement, with transvenous largely used to bridge the need for the placement of the permanent one. The possible complications (see: Table 6) may be immediate such as pneumothorax and cardiac perforation, intermediate such as dislodgement, or late such as pacemaker-related infections or conduction fracture.

Table 6. Complications of Transvenous Catheter Placement [15]

Timeline	Complications (Rate %)
Immediate	• Pneumothorax (0.6-0.9%) • Cardiac Perforation (0.1-0.3%) • Hematoma (0.2-0.7%)
Intermediate	• Lead dislodgement (0.4-1.7%) • Pocket revision due to pain complaints (0.4%)
Late	• Pacemaker infections (1.8-1.9 per 1000 pacemaker years) • Lead-related reintervention – conductor fracture, insulation break (1.7-.4%)

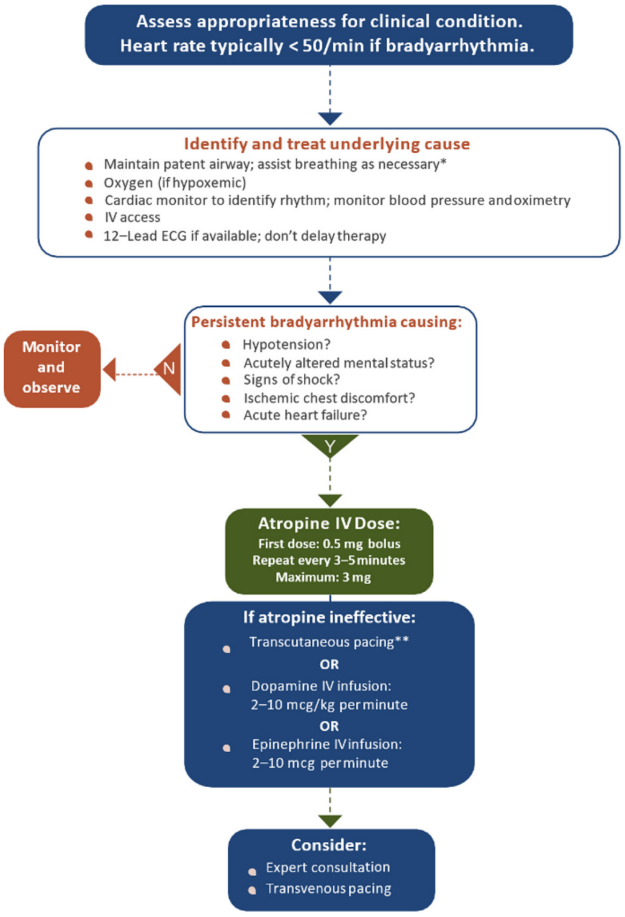

Figure 3. Emergency Management of Bradyarrhythmias

Postresuscitation Care

The mainstay of bradyarrhythmia therapy is the installation of a pacemaker. Before a pacemaker is implanted, every reversible cause of bradyarrhythmia must be addressed. Patients with clinical or electrocardiographic evidence of ischemia should be treated accordingly before getting a permanent pacemaker because the bradycardia may go away. The prognosis for AV block is determined by its severity and its location. Permanent cardiac pacing is typically used to treat complete heart block. Because the probability of full heart block in second-degree AV block type 2 is significant, pacemaker insertion is strongly recommended [16]. If the patient is symptomatic or the level of block is intra- or infra-His, a permanent pacemaker may be considered in second-degree AV block type 1 [16]. Pacemakers should be implanted in patients with 2:1 infranodal block.

Permanent pacing is used to treat sinus node disease, which is the second most common reason. It's a safe rhythm, and there's no evidence that cardiac pacing improves survival in this patient group. Pacing is commonly used to treat symptoms associated with sinus node disease (chronotropic incompetence, sinus pauses of more than 3 seconds, or sinus bradycardia of fewer than 40 beats per minute). According to current European Society of Cardiology (ESC) guidelines, individuals with a history of syncope and asymptomatic sinus pauses >6 seconds should be offered pacemaker implantation because there is insufficient evidence that pacing can prevent syncopal occurrences [16]. It can be difficult to tell if a patient has chronotropic incompetence. If the patient's heart rate rises above 100 beats per minute during a treadmill test or ambulatory Holter recording, pacemaker insertion is unlikely to help. The indications for permanent pacing are listed in Table 7.

Table 7. Recommendations for pacemaker implantation according to reference [16]

Recommendations in sinus node disease
Pacing is indicated when symptoms can be attributed to ¬bradycardia in sinus node disease.
Pacing is indicated in patients affected by sinus node disease who have documented symptomatic bradycardia due to sinus arrest or ¬sinoatrial block.
Sinus node dysfunction after cardiac surgery and heart transplantation. A period of clinical observation from 5 days to some weeks is indicated to assess if the rhythm disturbance resolves.
Recommendations in AV conduction disorder
Pacing is indicated in patients with third- or second-degree type 2 AV block irrespective of symptoms.
Intermittent/paroxysmal AV block (including atrial fibrillation with slow ventricular ¬conduction). Pacing is indicated in patients with intermittent/paroxysmal intrinsic third- or second-degree AV block.
Pacing is indicated in patients with an alternating bundle-branch block with or without ¬symptoms.
Pacing is indicated in patients with syncope, bundle-branch block, and ¬positive electrophysiology studies defined as an HV interval of ≥70 ms, or ¬second- or third-degree His-Purkinje block demonstrated during ¬incremental atrial pacing or with the pharmacological challenge.
High degree or complete AV block after cardiac surgery and transcatheter aortic valve implantation. A period of clinical observation of up to 7 days is indicated to assess whether the rhythm disturbance is transient and resolves. In the case of a complete AV block with a low rate of escape rhythm, this observation period can be shortened since the resolution is ¬unlikely.
Pacing should be considered in patients with a history of syncope and ¬documentation of asymptomatic pauses >6 s due to sinus arrest, ¬sinoatrial block, or AV block.
Pacing should be considered in patients with second-degree type 1 AV block which causes symptoms or is found to be located at intra- or ¬infra-His levels in electrophysiology studies.
Pacing should be considered in patients ≥40 years with syncope and ¬documented symptomatic pause/s due to sinus arrest or AV block or the combination of the two.

How To Read An ECG (all capital)

When an ECG suffices is and interpreted correctly, any cardiac abnormalities that a patient may have can be pinpointed with more precision. Because every patient report symptom differently and heart rhythms differ from one person to the next, we provide the following guidelines for interpreting an ECG [7].

1. Is the rhythm regular? Examine the ECG's QRS segment to see if the depolarization in the ventricles is regular. Within a set amount of time, often six to ten seconds, measuring the distance between one R and the next can determine if that baseline measurement matches all other R-to-R distances. If any irregularities are discovered, inquire with the patient about their persistence. If this is the case, search for C.H.A.P.S. symptoms, which stands for Chest discomfort, Hypotension, Altered mental state, Poor perfusion, or Shortness of breath.
2. Determine the heart rate. To get a one-minute reading, multiply a six- or ten-second radial pulse by six or 10. Determine whether the patient has bradycardia, tachycardia, supraventricular tachycardia, or ventricular tachycardia with a pulse based on the reading.
3. Identify the P waves. Examine the heart monitor to see if the P waves are present, upright, and followed by the QRS section.

If all three are normal, the electrical impulse most likely started in the SA node, as it should.

4. Take a P-R interval measurement. Time the P wave to the start of the QRS segment. A P-R interval of 0.12 to 0.20 seconds is usual, with a longer P-R interval indicating an AV node blockage or delay.

5. Count the number of QRS segments. The QRS section should last 0.04 to 0.10 seconds in normal circumstances. A bundle branch block may be indicated by a long QRS segment. Bundle branch blocks aren't always harmful, but when they're paired with other conditions, they can signal cardiac disease.

6. Examine the T wave. The T wave should follow the QRS segment and be vertical. Inverted T waves could indicate a lack of oxygen to the heart, whereas peaked T waves could indicate hyperkalemia, flat T waves could indicate low potassium, and an elevated ST-segment could signify a heart attack.

7. Take note of any errant beats. Premature atrial contractions, premature junctional contractions, and premature ventricular contractions are caused by fibers outside the SA node that stimulate the heart to beat. Count any ectopic beats to assess their interval, form, and whether they happen singly or in groups.

8. Determine the source of the problem. Look for these elements after you've gathered all of the aforementioned information.

- **Sinus:** regular rhythm with 60-100 bpm; P waves upright, round, and occurring before the QRS segment; normal P-R interval; normal QRS duration.

- **Atria:** Rhythm may or may not be regular; the QRS segment is normal with abnormal P waves (premature, flat, notched, peaked, inverted, or hidden).

- **Junctional:** Is the P wave junctional, inverted before, during, or after the typical QRS segment?

- **Ventricular:** If the rhythm originates below the SA node, the QRS segment will be wide and unusual with no P waves.

- **Paced rhythm:** Low voltage pacer spikes before the QRS should be reviewed.

9. Identify the rhythm with accuracy. Compare the ECG data to the symptoms and vital signs of the patient. This will help you understand how to get started with treatment.

Conclusion

Bradyarrhythmia and conduction blocks are typical clinical findings that can be both a normal and pathologic response (for example, in healthy, athletic people). A heart rate of fewer than 60 beats per minute is characterized as bradyarrhythmia. It may manifest as symptoms such as palpitation, dizziness, confusion, signs of shock, or without any apparent clinical features. The level of disruptions in the hierarchy of the normal cardiac conduction system can be used to further categorize them. Sinus node dysfunction (SND) and atrioventricular (AV) conduction abnormalities or blocks are the two most common types of bradyarrhythmias. The clinical assessment starts with the assessment of vital signs, which also demonstrate slow heart rate and signs of impending or apparent shock. Emergency management is dictated by the clinical stability of the patient, followed by specific types of bradyarrhythmias. In certain types of bradyarrhythmias, cardiac permanent pacemakers are necessary after clinical stability is reached to prevent future recurrence.

Teaching Aspects [17]

Checklist of Clinical Knowledge				
	Level			
	Poor	**Acceptable**	**Qualified**	**Inapplicable**
Pre-Management				
Spotting Clinical Features				
Triage				
Preliminary				
Assessing Consciousness & vitals				
Assessing Airway				
Assessing Breathing & O2				
Assessing Circulation & IV access & BP & Oximetry				
Assessing Disabilities & Exposure				
Placing Cardiac Monitor				
Assess clinical status & identify reversible causes				
Correct Method of Application				
Confidence & Hesitation				
Initial Intervention				
Doesn't Manifest Clinical signs of Poor Perfusion				
Preliminary Management & Monitoring				
Manifest Clinical signs of Poor Perfusion				
Transcutaneous Pacing				
Atropine				
Epinephrine				
Consider Transvenous Pacing & look for reversible causes				

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