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CARDIAC ARRHYTHMIAS IN THE EMERGENCY ROOM AND ICU. BRADYARRHYTHMIAS: HOW TO IDENTIFY AND TREAT THE PATIENT WITH LOW PERFUSION

ARRITMIAS CARDÍACAS NA SALA DE EMERGÊNCIA E UTI. BRADIARRITMIAS: COMO IDENTIFICAR E TRATAR O PACIENTE COM BAIXA PERFUSÃO

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ABSTRACT

The cardiovascular system is responsible for adequate circulatory flow, which depends on systolic volume and heart rate (HR). When insufficient, it causes cerebral hypoflow and inability to perform activities. Bradycardia is caused by: a) sinus dysfunction, manifested by inappropriate HR, pauses or tachycardia-bradycardia syndrome, syncope, dizziness and intolerance to exertion, without risk to life; b) atrioventricular conduction disorder (atrioventricular (AV) blocks): first, second (Mobitz type I, Mobitz type II and advanced) and third degree (complete). First-degree and Mobitz type I AV block both have good prognosis. Mobitz type II, advanced and complete AV block, even oligosymptomatic or transient, without removable causes, have higher morbidity and mortality; c) neuromediated disorders and reflex syncope are triggered by orthostatic position or exposure to emotional stress and carotid sinus syndrome, associated with carotid stimulation. Low HR may be associated with increased risk, and signs and symptoms indicate severity. In emergency conditions the underlying causes should be treated to ensure good functioning of the airways; administer O2; monitor cardiac rhythm, HR, blood pressure, and venous access. It is important to analyze rhythm, and conduct a physical examination and clinical history, and to check for and treat contributing factors. If there are signs of low perfusion, atropine should be administered. Simulation by transcutaneous pacemaker is indicated if atropine is ineffective. Epinephrine or dopamine and transvenous stimulation should also be considered.

Keywords: Arrhythmias Cardiac; Bradycardia; Hypotention; Emergencies; Pacemaker, Artificial.

RESUMO

O sistema cardiovascular é responsável pelo fluxo circulatório adequado, o qual depende do volume sistólico e frequência cardíaca (FC). Quando insuficientes, causa hipofluxo cerebral e incapacidade de realizar atividades. A bradicardia é causada por: a) disfunção sinusal, manifestada por FC inapropriadas, pausas ou síndrome de taqui-bradicardia, síncopes, tonturas e intolerância aos esforços, sem risco à vida; b) distúrbio da condução atrioventricular (bloqueios atrioventriculares - BAV): de primeiro, segundo (Mobitz I, Mobitz II e avançado) e terceiro grau (Total). O BAV de primeiro grau e do tipo Mobitz I tem bom prognóstico. O BAV Mobitz II, avançado e total, mesmo oligossintomático ou transitório, sem causas removíveis, tem maior morbimortalidade; c) distúrbios neuromediados e a síncope reflexa são desencadeados por posição ortostática ou exposição à estresse emocional e a síndrome do seio carotídeo associada à estimulação da carótida. A FC baixa pode estar associada a um maior risco, sendo que os sinais e sintomas indicam gravidade. Na urgência, deve-se tratar as causas subjacentes assegurar o bom funcionamento das vias aéreas administrar O2 monitorar ritmo, FC, pressão arterial, e, também, o acesso venoso. É importante analisar o ritmo, exame físico e histórico, além de pesquisar e tratar os fatores contribuintes. Caso haja sinais de baixa perfusão, deve-se administrar atropina. A estimulação por marcapasso transcutâneo é indicada, caso a atropina seja ineficaz. Além disso, deve-se considerar a adrenalina ou dopamina e estimulação transvenosa.

Descritores: *Arritmias Cardíacas; Bradicardia; Hipotensão; Emergências; Marca-Passo Artificial.*

INTRODUCTION

The heart and vascular system are inter-related structures whose functions include maintaining an adequate peripheral blood flow to meet the metabolic demands, pumping the blood back to the lungs for gas exchange, and allowing a new cycle to start. This involves integration between the heart's pumping function and the ability to change the resistance to the peripheral blood flow that is responsible for maintaining human life. This integration is responsible for cardiac output, which corresponds to the product of the stroke volume or systolic volume and heart rate. Any reduction in heart rate may, to some extent, be offset by variations in peripheral resistance. When this goal is not reached, cerebral hypoflow occurs, making an individual unable to perform routine activities.[1,2]

Slow pulse was recognized and described by Stokes as a cause of circulatory repercussions during a physical examination in 1946, solely by observation of the decoupling of the "a" and "c" waves of the jugular venous pulse. This is regarded the first description of atrioventricular blocks (AVB) as a possible cause of syncope.[3]

Palpation of the arterial pulse has been practiced since antiquity, but the understanding of arrhythmia and its effects began to be better understood with the advent of equipment such as the sphygmomanometer in 1854, which enabled the recording of arterial pulse, and later in 1902, with the electrocardiographic (ECG) recordings made by Einthoven with a string galvanometer.[4]

Bradycardia can have other causes. During the 60s and 70s, changes in the sinus node (SN) were described that modify the adequate generation of heartbeats.[5] Other conditions, such as metabolic changes, use of medications, and neuromediated and reflex syncope can also decrease the heart rate.[6]

BRADYARRHYTHMIAS

Bradycardia, defined as a reduction in heart rate below 60 beats per minute (bpm)[7] with regular or irregular rhythm, can impair one's ability to maintain an adequate cardiac output for daily activities or physical exercise.[8] According to the modification of the generation, conduction, and changes caused by neural and reflex situations, we can classify them as: a) sinus function disorder; b) atrioventricular conduction disorder; and c) neurally mediated reflex disorders and carotid sinus syndrome.

SINUS FUNCTION DISORDER

A sinus function disorder involves disturbances in the generation and conduction of the sinus stimulus (difficulty in its passage from the sinoatrial node to the atrial tissue) characterizing the heart's inability to perform its pacemaker function. It is more prevalent in elderly individuals (1/600 of patients > 65 years), although it can be identified in all age groups.[9,10] It can manifest as inappropriate bradycardia (heart rate incompatible with the activity performed by the patient) and sinoatrial pauses or blocks accompanied by slow heart rates. It is also commonly associated with AVB of varying degrees and atrial tachyarrhythmias, such as atrial tachycardia and flutter or paroxysmal atrial fibrillation characterizing tachycardia–bradycardia syndrome, indicating diffuse disease and significant instability of the conduction system.[11,12]

Sinus function disorders can occur secondary to intrinsic causes, i.e., related to the sinus node itself, or extrinsic, i.e., influenced by external factors on the sinus node. These causes are described in Table 1. The electrical remodeling of the atria in cardiac insufficiency and atrial fibrillation can cause dysfunction in some patients.[13-15] Drug use, electrolytic and endocrine disorders, and autonomic dysfunctions also play an important role in this condition.[10,16-19]

On resting, dynamic, and/or exercise ECG, sinus bradycardia, sinus pauses or stops, sinoatrial block, atrial tachycardia, atrial fibrillation, and chronotropic incompetence are more frequently observed.[10-12] Bradycardia–tachycardia syndrome (Figure 1) is observed in more than half of the affected patients.[20]

The clinical features vary from asymptomatic patients with ECG evidence of some degree of sinus node dysfunction to patients with intermittent symptoms. The most common manifestations are dizziness, light-headedness, pre-syncope, syncope, palpitations, exercise intolerance, and fatigue. Systemic thromboembolism and cerebral ischemic events can be found, particularly in patients who develop atrial fibrillation.[10-12]

As a general rule, this pathology generates no imminent risk to life in emergency services and usually has a post-syncope presentation of dizziness and exercise intolerance associated with bradycardia.[21]

Rest and monitoring are often sufficient when waiting for definitive treatment. If symptoms are relevant, the use of

Table 1. Conditions associated with sinus node disease.

Intrinsic	Age
	Arterial hypertension
	Ico
	Rheumatic disease
	Cardiomyopathies
	Pericarditis
	Congenital heart defects
	Collagen vascular diseases
	Amyloidosis
	Surgical trauma
	- Interatrial communication
	- Mustard
	Cardiac tumors
	Irradiation
Extrinsic	Hypothermia
	Electrolyte imbalance
	Hypothyroidism
	Dist. SNA
	Hyperbilirubinemia
	Drugs
	- Digitalis
	- Beta-blockers
	- Calcium channel blockers
	- Methyldopa
	- Reserpine
	- Lithium carbonate
	- Cimetidine
	- Phenothiazines

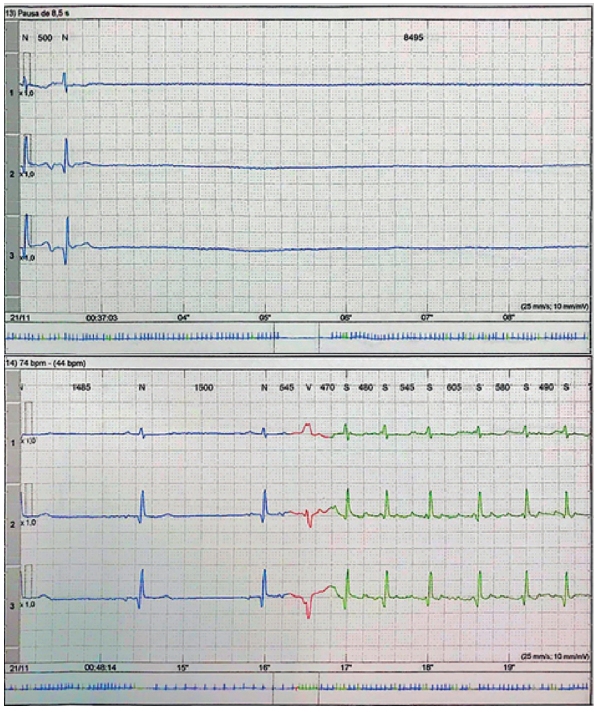

Figure 1. Holter clipping, initially showing sinus rhythm, sinus pause, and a period of atrial tachycardia (bradycardia–tachycardia syndrome).

sympathomimetic drugs, such as atropine (0.5 mg IV repeated 3–5 min up to 3 mg or six doses), epinephrine (2–10 μg/min), or dopamine (2–20 μg/kg per minute) can be used.

ATRIOVENTRICULAR CONDUCTION DISORDERS

The delay or failure in stimulus conduction between the atria and the ventricles characterize an AVB, which may be asymptomatic or cause symptoms of low cardiac cerebral and systemic output such as dizziness, pre-syncope, syncope, fatigue, dyspnea, edema, and palpitations due to bradycardia.

AVB are divided into first-degree, second-degree (Mobitz I, Mobitz II, fixed 2:1 and advanced), and third-degree. They can also be classified according to their electrophysiological location of supra-, intra-, or infra-Hisian, the last two being of greater severity. AVB may be congenital or acquired. Their duration may be permanent or transitory; depending on their etiology (such as electrolytic alterations or drug action), if the causal factor is removed, the stimulation may be waived.[22]

The first-degree AVB is characterized by a prolongation of PR interval beyond 200 ms. It commonly has a supra-Hisian location and good prognosis and is asymptomatic (Figure 2).

The second-degree AVB is subdivided into:

I - Type I (Wenckebach or Mobitz I), in which there is a progressive increase in the PR interval until an atrial stimulus (P wave) is not conducted to the ventricles and the subsequent beat is accompanied by a PR interval similar to the beginning of the cycle (shorter). It usually has a supra-Hisian location, usually has a good prognosis and may be asymptomatic (Figure 2).

II – Type II (Mobitz II), in which a sudden blocking of the P wave occurs, keeping a fixed PR interval. This type of block usually occurs due to a more severe disease of the conduction system. In the 2:1 AVB, an alternating conduction occurs in

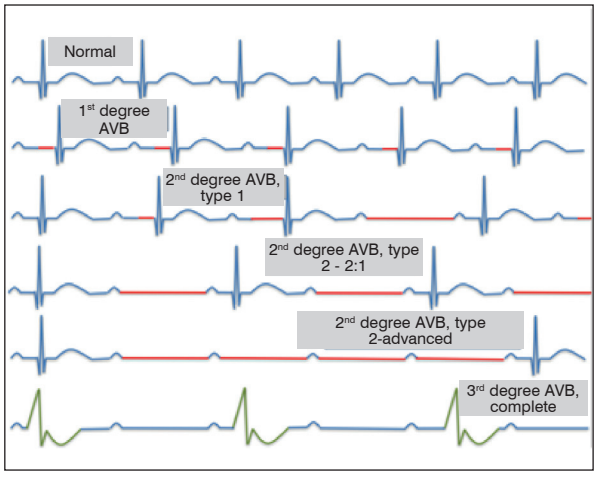

Figure 2. Models of the electrocardiographic manifestations of the atrioventricular blocks. Normal tracing is shown in blue, prolonged follow-ups are shown in red, and ventricular escape is shown in green.

the atrial impulse to the ventricles with the P-waves blocked in a proportion of 2:1 (two P-waves for each QRS) and can be supra- or infra-Hisian (Figure 2).

III – Advanced AVB, in which where there are more than two P-waves blocked in sequence, making the P:QRS ratio greater than 2:1 (i.e., 3:1, 4:1) (Figure 2).

IV – Third-degree AVB, or a complete AVB (CAVB), in which there is no atrioventricular conduction, causing dissociation between P waves and QRS. The ventricular rhythm is maintained by junctional or ventricular escape (Figure 2).

The second-degree atrioventricular type II, advanced AVB, and CAVB are most commonly located intra- or infra-Hisian following severe disease of the cardiac conduction system and featuring a higher risk of morbidity and mortality.

In Brazil, the most common acquired causes are: myocardial ischemia, Chagas disease, chronic degenerative fibrosis, coronary artery disease, and other cardiovascular diseases such as aortic stenosis, hypertension, or pulmonary embolism.[23]

In this situation, the transitory or established lesion of AV conduction is the determinant of symptoms of disease severity and the need for instrumentation.[20] First-degree blocks rarely require treatment.[24] The second-degree block type I[25] also rarely requires treatment in special care units. However, it should be noted that, when the block is total, survival is maintained by an ectopic focus, which can be unstable, especially if the QRS is very wide and the escape frequency is slow.[26]

Special attention should be given to second-degree type II and advanced blocks, which in addition to the symptoms caused by bradycardia may indicate the imminence of a total block, in which the escape rhythm is not guaranteed.[27]

Due to the characteristics of the pathology, the ventricular atrial blockade in acute myocardial infarction is a marker of poor prognosis, and the appearance of branch block and first degree or higher AV block indicates the need for a transvenous pacemaker.

Another recommendation of note is the transient block; if there is a removable causal factor, it should be treated as an established lesion.[20]

In the emergency services, an approach is needed whenever there are signs of transient or definitive low flow related to second-degree or greater AVB.

Even if oligosymptomatic, second-degree type II or higher AVB should be evaluated for a definitive pacemaker; if it cannot be performed soon enough, a transvenous temporary pacemaker should be installed. Temporary cardiac pacing is recommended for patients with acute myocardial infarction and the emergence of bundle branch blocks and a first-degree or greater AVB.

A transthoracic pacemaker is indicated as a bridge for transvenous or definitive stimulation, especially in hemodynamically unstable individuals.

Neurally mediated or reflex syndromes and carotid sinus hypersensitivity disease

Neurally mediated reflex syncope (NMRS), a result of neural reflex, usually presents as dizziness and syncope. Vasovagal syncope, a type of NMRS, is the most prevalent type and is characterized by hypotension and/or a bradycardia reflex as a result of sudden failure of the self-regulation blood pressure system. It can be triggered by a prolonged orthostatic position or exposure to emotional stress, fear, pain, medical procedures, hemorrhage, or heat, besides other stimuli. Common signs and symptoms include pallor, diaphoresis, dizziness, tinnitus, and blurred vision.[28,29]

Syncope that, besides the neurally mediated component, presents a triggering factor (cough, urination, gastrointestinal stimulus, or post-exercise) is known as situational syncope.[30]

These can be classified into three types:[31] Type I or mixed, characterized by the association of vasodepression and cardioinhibition; Type 2 or cardioinhibitory, which has a longer period of asystole; and Type 3 or vasodepressor, which presents as a marked predominance of hypotension.

In the emergency room, the post-syncopal presentation is more common and is usually associated with a prolonged orthostatic position or exposure to emotional stress, fear, or pain. Pallor, diaphoresis, dizziness, tinnitus, and blurred vision are common.[28]

Specific therapy in such cases is usually unnecessary. Dorsal decubitus, limb elevation, and hydration often improves symptoms, but the use of atropine may be necessary.

Carotid sinus hypersensitivity syndrome

Carotid sinus hypersensitivity syndrome is an abnormal reflex condition that is more common in men after 40 years of age attributed to baroreceptor dysfunction and possibly spinal cord dysfunction. It is associated with mechanical stimulation of the carotid sinus, either spontaneously or by massage, with a cardioinhibitory response of asystole or AVB > 3 seconds, vasodepressor behavior with a drop ≥ 50 mmHg systolic blood pressure, or a mixed response (Figure 3).

Carotid sinus hypersensitivity can manifest as syncope in older individuals associated with the mechanical stimulation of the carotid sinus, spontaneously or by massage, with cardioinhibitory response of asystole or AVB.[29]

As a general rule, the approach can be postponed for permanent cardiac pacing. Medication or temporary pacemaker placement are rarely necessary.

Emergency bradycardia care

Bradycardia can be a marker of or associated with the risk of death and involves several mechanisms, including severe cerebral hypoflow followed by stroke, coronary hypoflow accompanied by myocardial infarction, or, in cases of bradycardia secondary to AVB with a very slow ventricular response, the risk of potentially malignant ventricular tachycardia.

The signs and symptoms that usually present in situations of low perfusion indicate greater severity and should call for for prompt action in emergency rooms and intensive therapies.[32] In these situations, monitoring, the guarantee of venous access, besides having easy availability to medication, the use of a transvenous and transthoracic pacemaker is imperative. Except in cases in which the causal factor can be removed, the definitive treatment is artificial cardiac stimulation;[32] in emergency situations, one should guarantee the safety conditions up to issue resolution. The steps that should be observed (Figure 4) are available in the Advanced Cardiology Life Support (ACLS) standards.[33]

When the heart rate is <60 beats per minute, the clinical conditions and symptoms should be questioned. Clinicians must identify and treat the underlying causes:

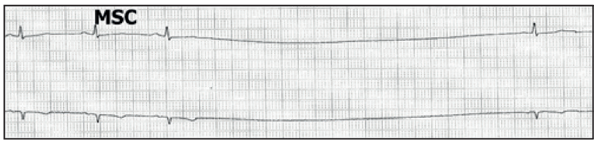

Figure 3. Electrocardiogram image indicating the beginning of the carotid sinus massage (CSM) and showing a sinus pause.

Maintain patent airways (respiratory support if necessary);

Administer O2 if the saturation is less than 94% or dyspnea is present;

Monitor pace, heart rate, and blood pressure;

- Establish a venous access;
- Obtain 12-lead ECG (if the equipment is accessible) and never delay therapy;
- Analyze the patient's rhythm, perform a physical examination, collect a history with a focus on the problem, and search for and treat contributory factors; and
- If bradycardia persists, observe if it is causing signs of low perfusion:
- Hypotension;
- Change in consciousness level;
- Signs of shock (pulmonary congestion, dizziness, low peripheral perfusion, cold skin, sweating);
- Symptoms of myocardial ischemia (chest pain); and
- Congestive heart failure (dyspnea, congestion weakness, fatigue)

There may be no other reason for the patient's symptoms. The adequacy of peripheral perfusion should be checked. The patient should be monitored and observed. However, if the infusion is low, the following steps should be taken: Transcutaneous pacemaker pacing (without delay) and the administration of atropine 0.5 mg IV, which may be repeated every 3–5 min up to 3 mg or six doses.

If the atropine is ineffective, start pacing. Consider administering adrenaline (2–10 μg/min) or dopamine (2–20 μg/kg/min) while awaiting pacemaker placement or if pacing is ineffective.

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

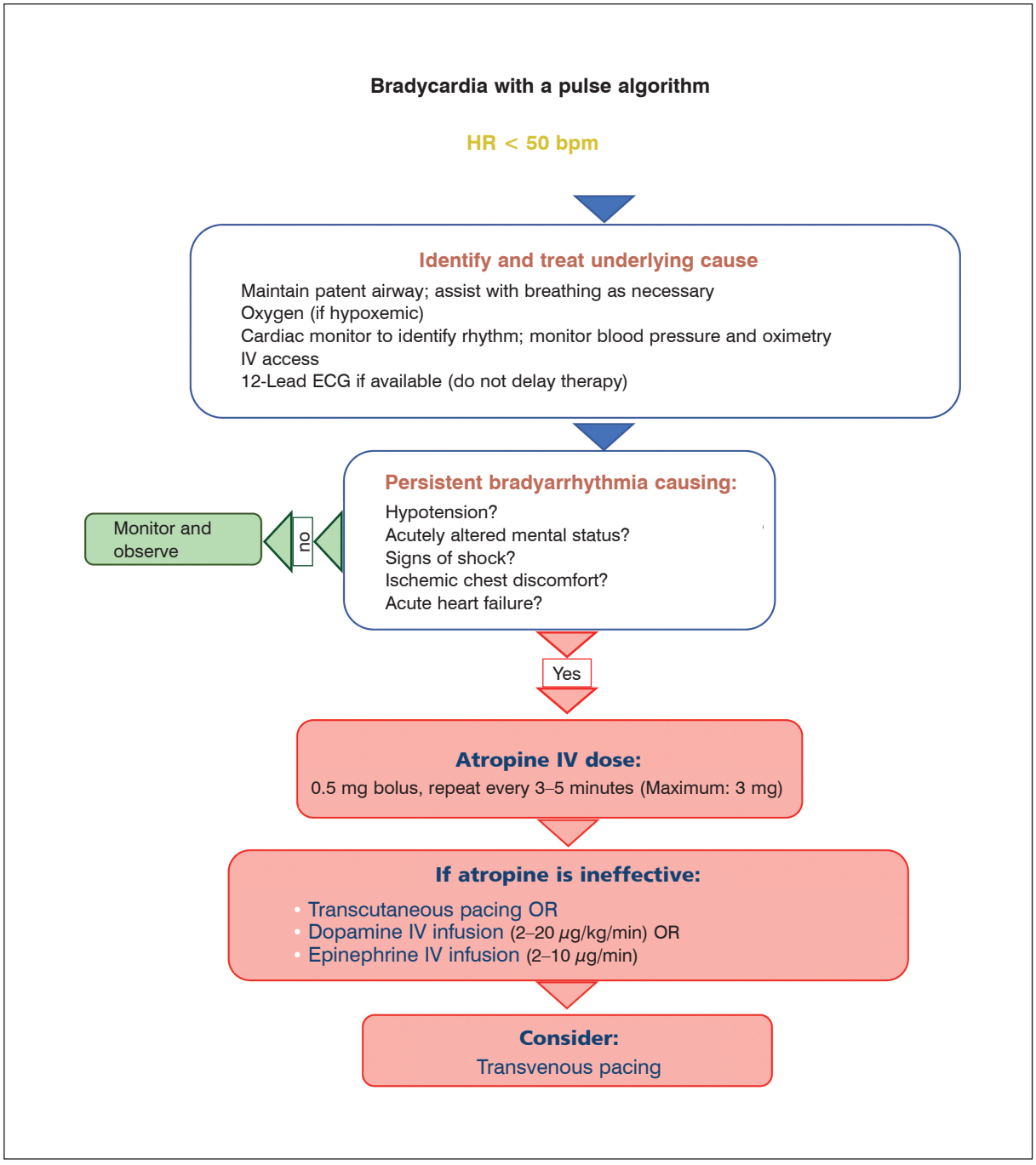

Figure 4. Bradycardia with a pulse algorithm modified from the Advanced Cardiology Life Support.[34]

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