APPROACH TO PEDIATRIC BRADYCARDIA – PART 1

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Introduction

Hello team,
My name is Damian Feldman-Kiss, and I am a medical student at the University of British Columbia. Today, we are going to discuss part 1 of an approach to pediatric bradycardia. This podcast was developed under the supervision of Dr. Aisling Young, a pediatric cardiologist at Victoria General Hospital, and Dr. Karen Forbes, a pediatrician and medical educator at the University of Alberta.

Learning Objectives

This first podcast will discuss the general approach to bradycardia in the pediatric population, including the etiology and initial evaluation. We will start with a stable patient. Stay tuned for part 2 that will focus on management of both the stable and unstable patient with bradycardia. In terms of objectives, at the end of this podcast you will be able to:

1. Differentiate cardiogenic syncope from seizure and other causes of syncope.
2. Define bradycardia in the pediatric population.
3. List at least five clinical features of pediatric bradycardia.
4. List four rhythms that cause bradycardia.
5. List the three most common causes of pediatric bradycardia.

Case: Introduction

Let's start with a case. You're on an emergency medicine rotation in Victoria. Your next patient is on the ambulatory side of the emergency department (ED). Your attending, Dr. Li, tells you that she is a 16-year-old female who presented to triage for a syncopal episode. Her name is Adrianne van Beek. She is stable and does not need immediate resuscitation. Dr. Li asks you to see Adrianne and then find her when you're ready to present. Before you head off, she reminds you to take a set of vitals.

Case: History [1,2,3]

You find Adrianne in her room. Adrianne tells you that she suddenly 'blacked-out' playing soccer in gym class three hours ago. She woke up on the floor with two friends by her side. Adrianne lost consciousness only briefly and her friend noticed some mild twitching. When she regained consciousness, she felt somewhat fatigued but wasn't confused or disorientated. She had been well all day and had eaten lunch before gym class. She's never experienced anything like this before.

Adrianne has some mild soreness of her left shoulder but denies a head injury and headache. There was no chest pain, palpitations, or dyspnea. There was no preceding dizziness, loss of balance, visual or auditory changes, diaphoresis, warmth, nausea, pallor, emotional stress, confusion, or irritability. There was also no loss of bowel or bladder function and no apparent rhythmic movements seen while she was unconscious.

She has been vaguely fatigued over the last few months and has felt a little short of breath while exercising. However, she denies cold intolerance, dry skin, weight gain, heavy periods, and historic chest pain and presyncope.

Adrianne's past medical history is unremarkable. Her immunizations are up to date. She doesn't take any medications or supplements and has no drug allergies. On a brief HEADS history, Adrianne has a boyfriend but is not yet sexually active. She has one to two drinks per month with friends but doesn't smoke cigarettes or consume illicit drugs. There is no family history of congenital heart disease, arrhythmia, need for pacemakers, heart muscle problems, cardiac transplant, or sudden unexplained death.

Case: Vitals

You start the physical exam by taking a set of vitals.

- Blood pressure is 112/68 and there is no orthostatic hypotension
- Radial pulses are regular and equal bilaterally at 30
- Respiratory rate is 16
- Temperature is 37.1ºC

Adrianne's vitals are all within normal limits, except for her heart rate. Now, let's discuss how bradycardia is defined in the pediatric population.

Definition of Pediatric Bradycardia

Unlike in adults, the normal range of heart rate in pediatrics depends on age. Therefore, bradycardia occurs when the heart rate falls below the normal range for the age of the patient. Normative values for bradycardia are different depending on the source. A highly cited evidence-based description of the normal ranges of heart rate in pediatrics was published in *The Lancet* in 2011. Their age-based definitions of bradycardia in beats per minute (bpm) are as follows [4]:

- term neonate: < 90
- 0 - 3 months: < 107
- 3 - 6 months: < 104
- 6 - 9 months: < 98
- 9 - 12 months: < 93
- 12 - 18 months: < 88
- 18 - 24 months: < 82
- 2 - 3 years: < 76
- 3 - 4 years: < 70
- 4 - 6 years: < 65
- 6 - 8 years: < 59
- 8 - 12 years: < 52
- 12 - 15 years: < 47
- 15 - 18 years: < 43

Using these definitions, we can illustrate an important point. Recall that the normal range of heart rate in adults is 60 to 100 and, by definition, bradycardia in adults is < 60. Depending on the patient's age, bradycardia in pediatrics may be under 60, between 60 and 100, or over 100.

There are also simpler but less precise definitions of pediatric bradycardia. According to the pediatric advanced life support (PALS) guidelines, pediatric bradycardia in the awake patient is defined in bpm as follows:

- Under 1 year: < 100
- 1 - 2 years: < 98
- 3 - 5 years: < 80
- 6 - 11 years: < 75
- 12 and older: < 60

Now, does Adrianne have bradycardia? Recall that she is 16 years old and her heart rate is 30. According to both sets of definitions, Adrianne has bradycardia. For more on pediatric vital signs, check out the PedsCases podcast, '[Pediatric Vital Signs]'. Alright, back to the physical exam.

<u>Case: Physical Exam</u>

Given Adrianne's presentation, you perform a cardiac, respiratory, and neurologic exam. You also examine Adrianne's left shoulder for signs of injury secondary to her fall. Adrianne is alert and appropriate. She has no visible cyanosis and is not dysmorphic. Cardiac examination reveals no radial-femoral delay. The precordium is quiet with a normal apical impulse. S1 is normal, and there is physiologic splitting of S2. There is a vibratory 2/6 systolic ejection murmur that is loudest at the left lower sternal border when supine. Diastole is quiet, and there is no hepatosplenomegaly. Her work of breathing is normal with good air entry bilaterally to the lung bases. There is mild tenderness in the left deltoid. Otherwise, her shoulder and neurologic exams are unremarkable. After finishing the exam, you head off to find your attending.

Case: Presentation

You find Dr. Li at her workstation and present the case:

Dr. Li, I just saw a 16-year-old female who presented after a syncopal episode 3 hours ago. Her syncopal episode came on with exertion and she denies any prodrome. She describes it as 'blacking out' and didn't experience vertigo or a loss of balance. She lost consciousness only briefly and had a spontaneous complete recovery. This is her first episode. Her only other symptoms are of vague fatigue over the last few months and a mild decrease in exercise tolerance. The remainder of her history is unremarkable. Her vitals are within normal limits, except she has bradycardia with a heart rate of 30. Cardiac exam revealed an innocent Still's murmur. She also has some mild tenderness of her left shoulder, but otherwise she has a normal exam.

Cardiogenic syncope is at the top of my differential because of the lack of prodrome, sudden onset during exercise, and her bradycardia. I also considered vagal-mediated syncope, but she denied prodromal symptoms. It doesn't seem like a seizure as she didn't have any rhythmic movements while unconscious, and there was no loss of bowel or bladder function and no postictal period. In addition, hypoglycemia, drugs, and medications are unlikely causes because she has no history of diabetes and denies the use of medications and illicit drugs. The key investigation I would like to order is a 12-lead electrocardiogram (ECG), but I would also like to order a bedside glucose to rule out hypoglycemia.[1,2,3] Also, given how low her rate is, I think we should probably put Adrianne on a monitor while the investigations are underway.

Dr. Li comments on your well-articulated clinical reasoning and agrees with your plan. She then asks how else pediatric bradycardia can present.

Clinical Features of Pediatric Bradycardia

The clinical presentation of bradycardia depends on a number of factors such as the underlying rhythm, the patient's age, and the severity of the bradycardia or how low the heart rate is. For example, sinus bradycardia is most often completely asymptomatic. If the bradycardia is an arrhythmia that is just below normal limits, patients can also be asymptomatic. Even patients with complete heart block with an adequate escape rhythm can be asymptomatic. When bradycardia does cause symptoms, the type of symptoms depends on the patient's age.[5] In addition, the susceptibility to being symptomatic is increased in children having an underlying congenital heart defect.[5] Infants can present with feeding difficulties and lethargy.[5] Children and adolescents can present with fatigue, exercise intolerance, presyncope, and/or syncope.[5] In severe bradycardia, cardiac output may be inadequate, which manifests initially as poor perfusion and may progress to shock and cardiorespiratory arrest.[5,6]

Case: Initial Investigations

You return to Adrianne's room to discuss the next steps. On route, Dr. Li quizzes you on the definition of syncope. Having recently listened to the PedsCases podcast, 'Approach to Pediatric Syncope,' you respond, syncope is a 'transient loss of consciousness due to cerebral hypoperfusion.' The key characteristics are 'a rapid onset, short duration, and spontaneous complete recovery.'[7]

You enter Adrianne's room to discuss your working diagnosis and the recommended investigations:

Hello Adrianne, we would like to order a test called an ECG. It is the key first line test in the evaluation of both 'blacking out' and a slow heartbeat, which are called syncope and bradycardia, respectively. It's like an electrical snapshot of your heart's rate and rhythm over 10 seconds. That being said, it's not a perfect test and can miss abnormal rhythms that aren't always present, called intermittent arrhythmias. We would also like to order a bedside glucose measurement because it will help rule out low blood sugar as a cause of your syncopal episode.

Adrianne agrees to the tests. As you leave her room, you kindly ask her nurse to place Adrianne on a heart rate monitor and to update you with any changes in her status.

Back at the workstation, Dr. Li asks you to describe the various waveforms and intervals on an ECG and how to calculate heart rate.

Electrocardiogram

The ECG is a recording of cardiac electrical activity over 10 seconds. Specifically, it measures changes in voltage over time across the entire heart. The normal ECG consists of the following components:

- P wave, corresponding to atrial depolarization
- PR interval, corresponding to the period when the atrial impulse travels through the AV node and bundle of His
- QRS complex, corresponding to ventricular depolarization
- ST segment, corresponding to the period between ventricular depolarization and repolarization
- T wave, corresponding to ventricular repolarization, and
- QT interval, corresponding to the period of ventricular depolarization and repolarization

The heart rate can be determined by dividing 300 by the RR interval or multiplying the number of QRS complexes in the rhythm strip by 6. For more on ECGs, check out the PedsCases podcast, 'Approach to Pediatric ECG'.

Case: Waiting for Results

Dr. Li is then notified about an incoming patient. You check the EMR and see that Adrianne's blood glucose level is in the normal range at 5.1 mmol/L, which is expected as Adrianne has recovered clinically from her syncopal episode. While you wait for Adrianne's ECG, you decide to review the ECG features and mechanisms of common bradyarrhythmias.

ECG Features and Mechanisms of Common Bradyarrhythmias

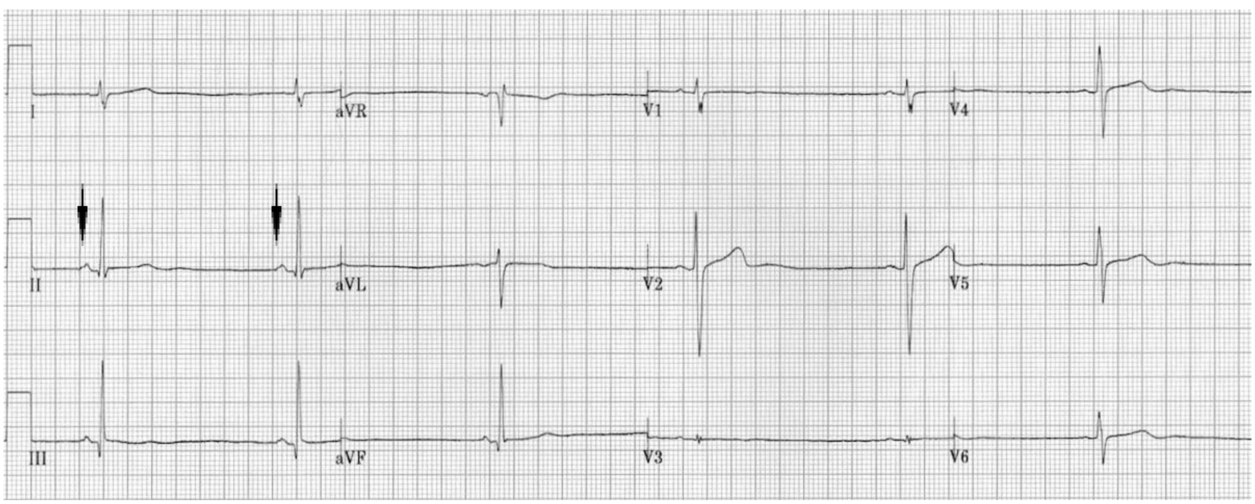
The most common rhythms causing pediatric bradycardia are sinus bradycardia, junctional bradycardia, and third-degree (complete) AV block.[8] Second-degree AV block Mobitz type 2 can also lead to bradycardia. We will discuss each of these in more detail.

It is important to note that some rhythm abnormalities, such as first-degree AV block and second-degree AV block Mobitz type 1, do not actually cause bradycardia. However, for completeness, they are included here and will be discussed later on.

Now, let's go through each of these rhythms to help distinguish them on an ECG. If you would like to follow along visually, pictures of each rhythm can be found in the script of this podcast.

Sinus Bradycardia

Sinus bradycardia occurs when there is decreased automaticity at the SA node. All of the ECG features (e.g., rhythm, P wave morphology, PR interval, QRS width, etc.) are normal, except for a low heart rate. Basically, it is sinus rhythm with a heart rate below normal limits. Importantly, sinus bradycardia is most often asymptomatic, as mentioned earlier, and of no clinical concern.



Sinus bradycardia. Black arrows indicate P waves.
Source: https://litfl.com/sinus-bradycardia-ecg-library/

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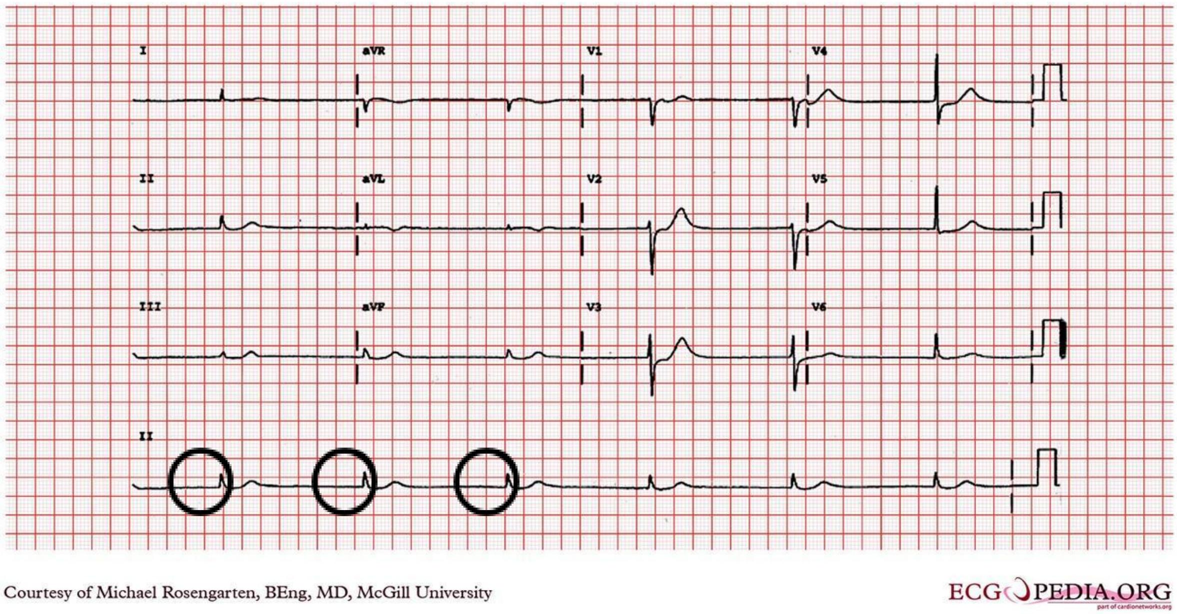
Junctional Bradycardia

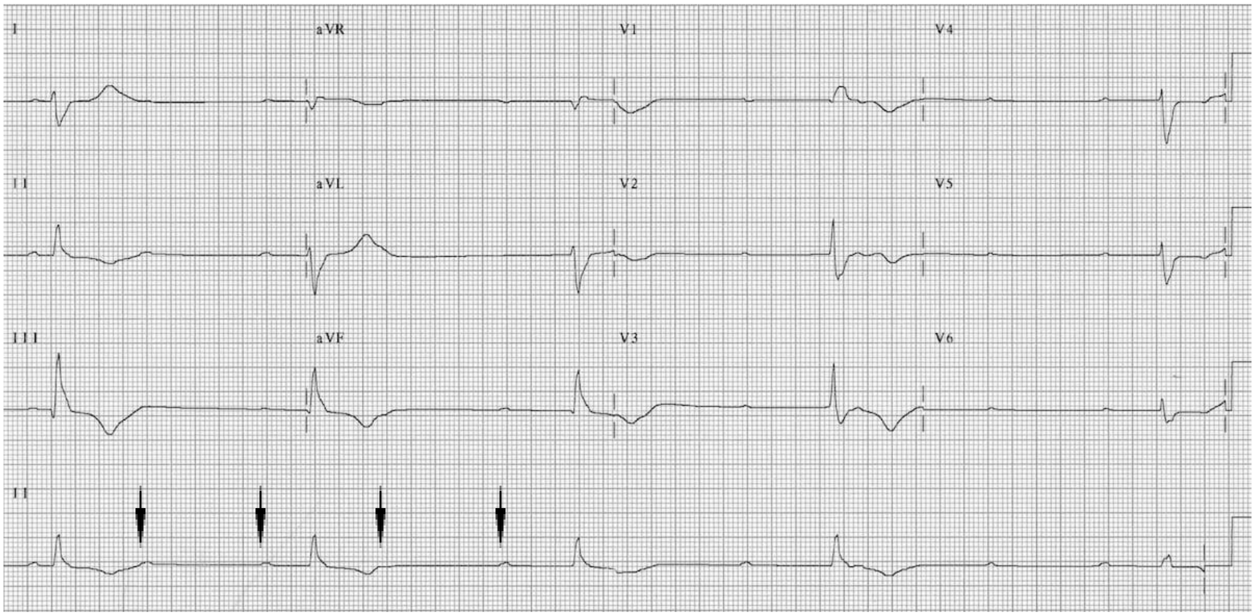
In order to understand junctional bradycardia, it helps to recall that there are 'backup' pacemakers along the cardiac conduction pathway; when the pace of an upstream site is too slow, the next site takes over. In junctional bradycardia, the automaticity of the SA node drops below that of the AV node resulting in the AV node setting the pace. The ECG features of junctional bradycardia are a low heart rate and absent or abnormal P waves with a narrow QRS complex.[9] These P waves are abnormal because they are inverted and occur after the QRS complex.[9] This P wave inversion occurs because the wave of depolarization is travelling from the AV junction retrograde through the atria (i.e., the opposite direction of normal). These P waves are appropriately called retrograde P waves. They occur after the QRS complex because atrial depolarization is happening after ventricular depolarization. The narrow QRS complex is characteristic of a junctional rhythm, as the impulse initiates at the AV node and travels along the usual ventricular conduction pathways.



Junctional bradycardia. Black circles indicate the absence of a P wave before the QRS complex.
Source: https://www.wikidoc.org/index.php/Junctional_bradycardia_EKG_examples

Third-Degree (Complete) AV Block

Third-degree (complete) AV block occurs when the conduction between the atria and ventricles is absent (i.e., completely blocked), resulting in atrial-ventricular dissociation. The ECG features of complete AV block are a low ventricular rate, an atrial rate greater than the ventricular rate, no association between the P waves and QRS complexes, regular but unrelated atrial and ventricular rhythms, possible buried P waves, and a narrow or wide QRS complex depending on whether it is junctional (narrow QRS) or ventricular (wide QRS) in origin.



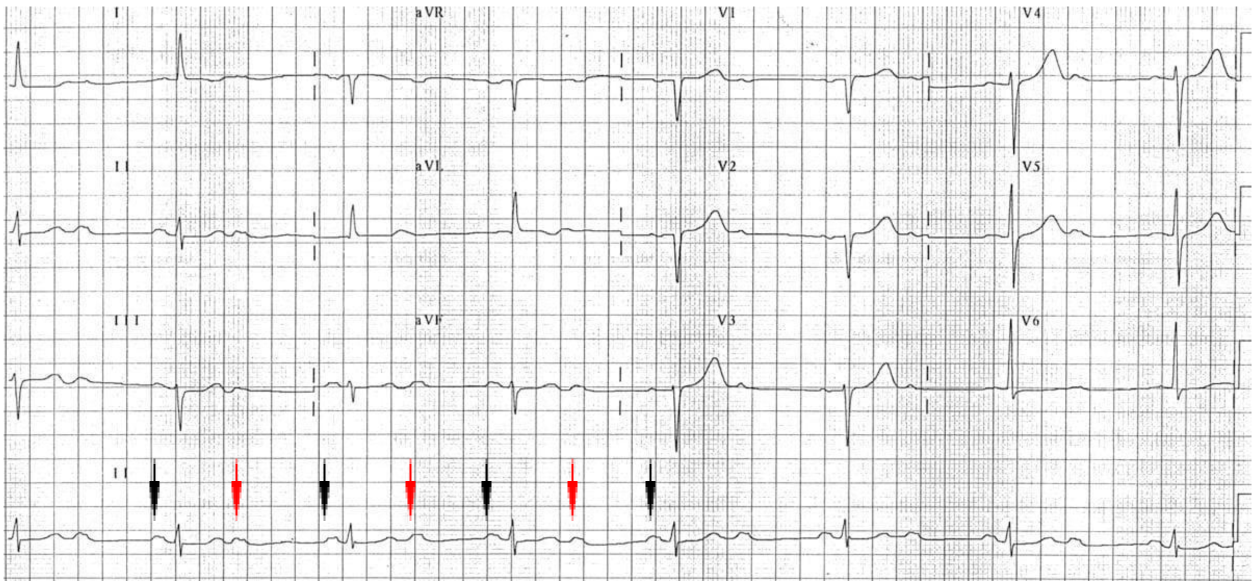
Complete heart block. Black arrows indicate P waves that have no association with the QRS complexes. Note that the third arrow indicates a P wave that is obscured by a T wave.
Source: https://litfl.com/av-block-3rd-degree-complete-heart-block/

Second-Degree AV Block – Mobitz II

In second-degree AV block, there is an increased ratio of P waves to QRS complexes (i.e., some, but not all of the P waves are conducted to the ventricles resulting in missed ventricular beats).

Mobitz type 2 occurs because of faulty conduction at the level of the His bundle, bundle branches, or the fascicles.[13] In Mobitz type 2, the PR interval is constant and the missed

QRS complex occurs randomly, unlike the predictable pattern in Mobitz type 1. In addition, the length of the PR interval may be normal or prolonged. This causes a narrow QRS complex, a regular atrial rhythm, and an irregular ventricular rhythm.[12] However, in some cases of Mobitz type 2, every other QRS complex is dropped, which produces 2:1 AV block.[12] In these cases, both the atrial and ventricular rhythms are regular. For example, if there is 2:1 AV block with an atrial rate of 60, the ventricular rate—and, therefore, the patient's pulse—will be 30. Importantly, Mobitz type 2 can cause bradycardia and can also progress to complete AV block, making Mobitz type 2 more concerning than type 1.[12]



2:1 AV block. Black arrows indicate P waves that are conducted to the ventricles. Red arrows indicate P waves that are not conducted to the ventricles.
Source: https://litfl.com/av-block-2nd-degree-fixed-ratio-blocks/
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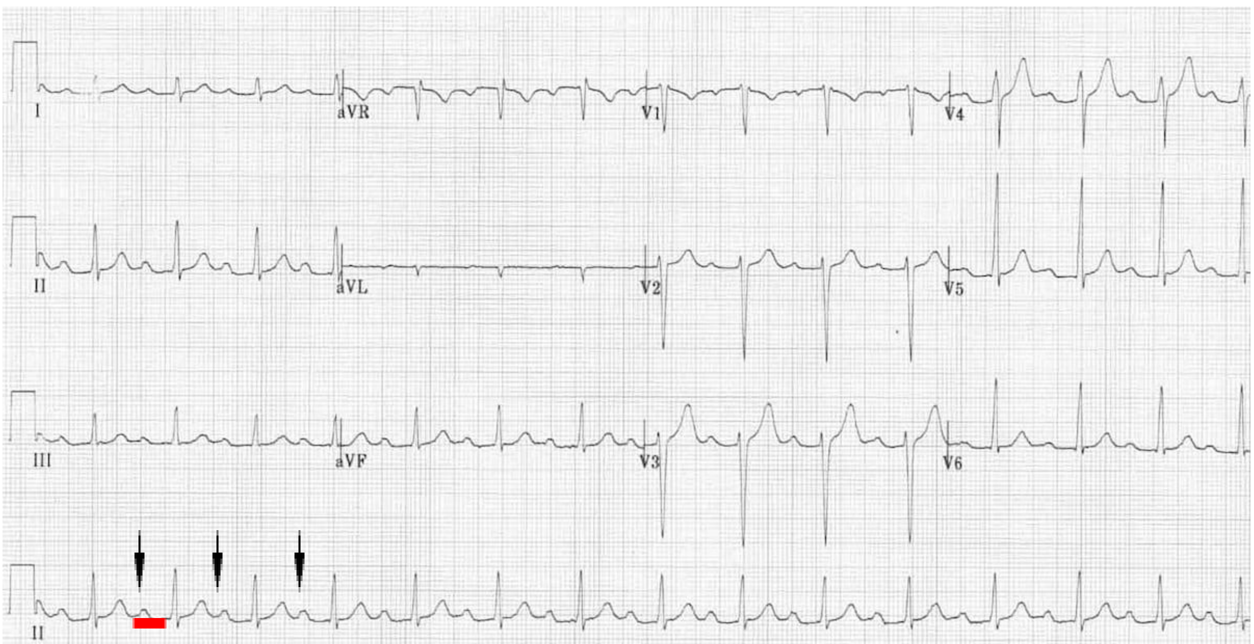
Atrioventricular (AV) Blocks that do not cause Bradycardia

For completeness, we will also review first-degree AV block and second-degree AV block Mobitz type I, noting again that they do not typically result in bradycardia.

First-Degree AV Block

First-degree AV block occurs when there is decreased conduction velocity at the AV node. It is most often due to increased vagal tone, which is very common in pediatrics. All of the ECG features are normal except for a prolonged PR interval, which like heart rate, depends on age.[10] The normal PR interval in infants and young children is < 160 ms or 4 small squares.[10] The normal PR interval in older children and adolescents is < 200 ms or 5 small squares.[11] Therefore, a PR interval of >= 160 ms in a 5-year-old and >= 200 ms in a 16-year-old is considered to be prolonged. Importantly, first-degree AV block does not cause bradycardia and does not predispose to a higher degree of heart block.

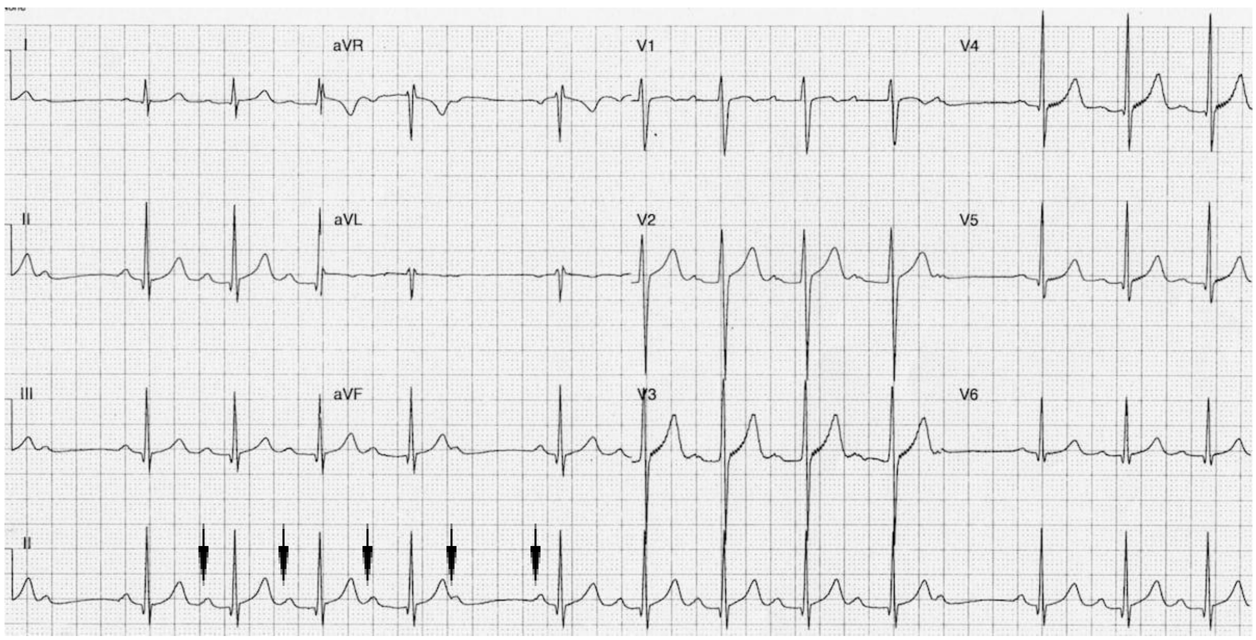


First-degree AV block. Black arrows indicate P waves. Red line indicates the prolonged PR interval.
Source: https://litfl.com/first-degree-heart-block-ecg-library/

Second-Degree AV Block – Mobitz I (Wenckebach)

As noted above, second-degree AV block occurs when there is an increased ratio of P waves to QRS complexes (i.e., some, but not all of the P waves are conducted to the ventricles resulting in missed ventricular beats).

Mobitz type 1 (also called Wenckebach) most often occurs because of decreased conduction at the AV node due to increased vagal tone, which, as mentioned earlier, is very common in pediatrics. In Mobitz type 1, the PR interval progressively lengthens until an atrial depolarization fails to be conducted to the ventricles. After the missed QRS complex, the cycle repeats itself. This causes a narrow QRS complex and a regular atrial rhythm, but an irregular ventricular rhythm.[12] Importantly, Mobitz type 1 does not cause bradycardia and is typically benign with no progression to a higher degree of heart block.[12]



Wenckebach. Black arrows indicate P waves. Note that the PR interval becomes progressively longer until the impulse is not conducted to the ventricles resulting in a missed QRS complex.
Source: https://litfl.com/av-block-2nd-degree-mobitz-i-wenckebach-phenomenon/

Case: Results of Initial Investigations

After finishing your reading, you check the EMR and see that Adrianne's ECG is back, which demonstrates a complete AV block. Given this bradyarrhythmia with no prior history of complete AV block, you and Dr. Li decide to consult pediatric cardiology and admit her to their service.

Before returning to Adrianne's room to discuss the test results and next steps, you and Dr. Li discuss the etiologies of pediatric bradycardia.

Etiologies of Pediatric Bradycardia

There are multiple ways to organize the etiologies of bradycardia. These include the following categories:

- Extrinsic vs intrinsic causes
- Congenital vs acquired causes, and
- Anatomic or system based

The specific framework chosen is less important than being able to work through the diagnostic possibilities in an organized manner. For this discussion, we will use the categorization of extrinsic vs intrinsic. The etiologies of bradycardia affect the cardiac conduction system in a variety of ways and reviewing each mechanism and the resulting rhythm is beyond the scope of this podcast. However, we will discuss a few of them below.

Extrinsic causes are factors disrupting native conduction; these include [12]:

- Hypervagatonia (i.e., increased vagal tone)
- Drugs
- Hypoxia
- Hypothermia
- Hypothyroidism, and
- Electrolyte abnormalities

The most common extrinsic causes of bradycardia in children are hypervagatonia and drugs.[8,14] Hypervagatonia is actually the most common cause overall. Hypervagatonia decreases heart rate by decreasing automaticity at the SA node and slowing conduction at the AV node. Hypervagatonia can be secondary to, for example, endurance training, anorexia nervosa, obstructive sleep apnea, and increased intracranial pressure.[5,8] However, as mentioned earlier, hypervagatonia can also be normal in children. Drugs causing bradycardia include opioids (e.g., fentanyl), alpha-2-agonists (e.g., clonidine), beta-blockers (e.g., metoprolol), calcium channel blockers (e.g., diltiazem and verapamil), digoxin, lithium, and amiodarone.[5,8] Electrolyte abnormalities include hyper and hypokalemia, hyper and hypocalcemia, and hypomagnesemia.[8]

Intrinsic causes of bradycardia are injury to or intrinsic dysfunction of the cardiac conduction system. These include [5,12]:

- Complications from cardiac surgery performed near the conduction tissue
- Congenital heart defects
- Endocarditis
- Myocarditis
- Cardiomyopathy, and
- Idiopathic fibrosis

The most common intrinsic causes are complications from cardiac surgery performed near the conduction tissue.[8,14] That being said, it is important to note that cardiac surgery resulting in bradycardia is still quite rare.[15] Nevertheless, these surgeries risk injuring the conduction tissue and disrupting native conduction. The AV node may be affected in a VSD repair, Tetralogy of Fallot repair, subaortic resection, and tricuspid or mitral valve replacement. The resulting bradyarrhythmia is typically an AV block. The SA node is less commonly injured, but it can be affected in an ASD repair and Fontan procedure. Another intrinsic cause of pediatric bradycardia is neonatal lupus, which causes the vast majority of congenital complete AV block.[8] The pathophysiology of this disease is not completely clear. However, the proposed mechanism involves maternal antibodies crossing the placenta and disrupting the development of the fetal conduction system leading to dysfunction of the AV node.[5,8] It is also important to remember that sometimes the underlying cause of complete AV block cannot be identified, and it is then characterized as idiopathic. This is the case in idiopathic complete heart block.

In summary, like much of medicine, it is important to have an organized approach to the etiologies of bradycardia. One logical approach is extrinsic vs intrinsic causes. The most common extrinsic causes of bradycardia in children are hypervagatonia and drugs, while the most common intrinsic causes are complications from cardiac surgery performed near the conduction tissue and congenital heart defects.

Alright, let's apply what we've learned to our case. In terms of the extrinsic causes, Adrianne doesn't take any medications or consume illicit drugs, she has a normal temperature, and doesn't have any clinical features of hypothyroidism. This makes drugs, hypothermia, and hypothyroidism unlikely culprits. In terms of the intrinsic causes, Adrianne has no history of cardiac surgery and no known congenital heart defects or heart disease, which argues against these etiologies. Nevertheless, we won't know for sure until a full work-up is performed by pediatric cardiology.

Case: Next Steps

You and Dr. Li head back to Adrianne's room to discuss the results and next steps. However, on your way, you get called overhead to help out in the trauma bay. A

14-year-old male has taken a toxic dose of his medication and will likely need resuscitation on arrival. EMS says that he is bradycardic at 20 and is showing signs of shock. He has hypotension, respiratory depression, altered mental status, and weak pulses.

Conclusion

Alright team, that wraps up part 1 of this PedsCases podcast. Stay tuned for part 2 in which we will focus on management of both the stable and unstable patient with bradycardia and resolve our two cases. Before we finish up, let's review the learning objectives.

1. **Differentiate cardiogenic syncope from seizure and other causes of syncope.** Cardiogenic syncope can be differentiated from seizure and other causes of syncope by onset during exercise and no prodrome, urinary or fecal incontinence, rhythmic movements while unconscious, and postictal period.
2. **Define bradycardia in the pediatric population.** The definition of bradycardia in the pediatric population depends on age and is defined differently between different sources.
3. **List at least five clinical features of pediatric bradycardia.** The clinical features of pediatric bradycardia include:
   - No signs and symptoms, as is commonly the case in sinus bradycardia
   - Feeding difficulties and lethargy in infants
   - Fatigue, exercise intolerance, presyncope, and/or syncope in children and adolescents, and
   - Poor perfusion, shock, and cardiorespiratory arrest in severe bradycardia at all ages
4. **List four rhythms that cause bradycardia.** Four rhythms that cause bradycardia are sinus bradycardia, junctional bradycardia, second-degree AV block, and complete AV block.
5. **List the three most common causes of pediatric bradycardia.** The three most common causes of pediatric bradycardia are hypervagatonia, drugs, and complications from cardiac surgery performed near the conduction tissue.

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