

Fast-Bleeped: A to E Series – Circulation: Unstable Bradycardia

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A medical educational series comprising practical instructional pieces on how to approach undifferentiated clinical problems in the acute setting

CASE OF MR. D

You are the Foundation Year 1 (FY1) doctor on call overnight covering the medical inpatients in your local district general hospital. You receive a bleep from the nursing team on the acute medical unit (AMU) asking you to review Mr D. He is a 54-year-old Afro-Caribbean man who was admitted following an episode of chest pain. His typical cardiac sounding chest pain, inferior T-wave changes on his electrocardiogram (ECG) and positive troponin led the clerking medical doctor to treat him for an inferior non-ST elevation myocardial infarction (NSTEMI). The chest pain had settled by the time Mr D was seen by the medical team and it was agreed with the local "heart attack service" that he should be managed with medical therapy in the first instance and monitored in the hospital. However, later on the ward, the nursing team are worried because during their observation round, he was found to be "drowsy". The following observations are relayed through the telephone:

- Heart rate (HR): 31 beats per minute (bpm)
- Respiratory rate (RR): 20 respirations per minute
- Oxygen saturations: 95% on room air
- Blood pressure (BP): 90/40mmHg
- Temperature: 35.7°C

You decide to quickly make your way to the AMU and ask the nursing team by telephone to start doing a repeat ECG and perform a venous blood gas (VBG). You ask the team to



stay with him and to put out a medical emergency call if he deteriorates, and that you will be there quickly. On arrival, you find Mr D lying in bed drowsy. You begin your assessment using an ABCDE approach.¹ His airway is patent, but he is breathing rapidly and is only responsive to pain. You palpate his carotid pulse and find it to be slow at approximately 30bpm, and weak. His central capillary refill time (CRT) is six seconds. His peripheries feel cold and clammy. You decide that Mr D is acutely unwell and ask the nursing team to put out a medical emergency call for urgent senior support.²

WHAT SHOULD BE YOUR INITIAL APPROACH WITH MR. D?

Based on your brief clinical examination and observations, you decide that the patient is in shock, with bradycardia and a decreased Glasgow coma score (GCS). These signs are of a medical emergency, and having called for senior support, you begin a more comprehensive A-E assessment.¹

(A)irway: You speak to Mr D and he gives you a mumbled, confused response. His airway appears to be clear. You can hear no additional airway noises, and, although confused, he is able to talk to you. You therefore deem his airway to be patent.¹

(B)reathing: On inspection he has got shallow fast breaths. There is equal bilateral chest movement. The trachea is central. His saturations are adequate at 95% and his RR is on the higher end of normal at 20. You auscultate his chest and discover fine crackles on his bases bilaterally. As he is acutely unwell, you decide to administer additional oxygen via a non-rebreathe mask at 15 litres per minute until senior support arrives.^{1,2} You are satisfied that his breathing is stable for the moment, so you continue your assessment.¹

(C)irculation: You note that his HR is low, at 31 bpm, which immediately worries you. You ask for a repeat BP and for the nurse to put the observation machine in an automated mode to cycle BP readings every 2.5 minutes for up-to-date information.² His central CRT remains prolonged at six seconds. You touch the patient's peripheries and find them cold. You examine his jugular venous pressure which you suspect is high and notice mild pedal oedema. He appears confused, and his face is pale.²

At this point, you decide that he has a bradyarrhythmia with cardiovascular compromise. You attach the defibrillator pads and switch the defibrillator on to monitoring mode so that you have continued 3 lead ECG monitoring.² You review the 12 lead ECG that the nurse performed and decide that it shows sinus bradycardia and there is no evidence of heart block.² You check his drug chart to ensure he is not on any medications that could cause bradycardia; he has been prescribed bisoprolol in the context of his NSTEMI but it has not been administered yet, you cross it off the chart for now.²

The blood gas comes back and shows a compensated

metabolic acidosis with elevated lactate, but with no significant derangement in electrolytes. This provides further evidence that the patient is haemodynamically compromised.³ Therefore, you decide that the bradycardia needs immediate treatment.^{2,4} You ask the nurse to prepare 3 milligrams (mg) of atropine and you ask for someone to establish intravenous (IV) access and send a full set of bloods. You administer 500 micrograms of atropine followed by a saline flush which raises his HR to 49.⁴ You repeat the blood pressure and find that it has improved to 110/62. You find comfort in this for the moment and proceed with your assessment, with the intention to come back to “c” and reassess.

(D)isability: Mr D has a fluctuating conscious level and appears confused. You remember the importance of assessing glucose levels, especially in patients with decreased GCS, and you find it to be normal (5.5) on the VBG. You calculate his GCS as 10/15. His eyes are opening to pain (2), his verbal response is inappropriate (3) and is moving towards localised pain (5). His temperature is 35.7°C.

(E)verything else: He has no signs of haemorrhage. His abdomen is soft on palpation.

Just as you begin to reassess the patient, the medical emergency team (MET) arrive. You promptly deliver a situation, background, assessment, and recommendation (SBAR):

This is Mr. D, he is a 54-year-old with cardiovascular compromise secondary to an unstable bradycardia, he was admitted earlier in the evening with a suspected inferior NSTEMI. I have assessed him, and he is peripherally shut down with signs of shock. He was bradycardic and hypotensive, with a heart rate of around 30bpm. I have administered 500 micrograms of atropine IV, which improved his HR to around 50, however, I think that he needs further assessment and definitive treatment.

What are the important concepts in cardiovascular electrophysiology?

The natural pacemaker of the heart is the sinoatrial (SA) node.⁵ It generates impulses by automatic spontaneous depolarisation. The most significant factor in inciting depolarisation is a small influx of sodium into the cell. The rate of depolarisation is controlled by autonomic tone via sympathetic and parasympathetic nervous systems.⁵

The depolarisation is propagated across the atria down into the atrioventricular (AV) node. The AV node delays further conduction for a short time before the electrical signal is propagated along the Bundle of His. This structure conducts the depolarisation down the septum of the heart

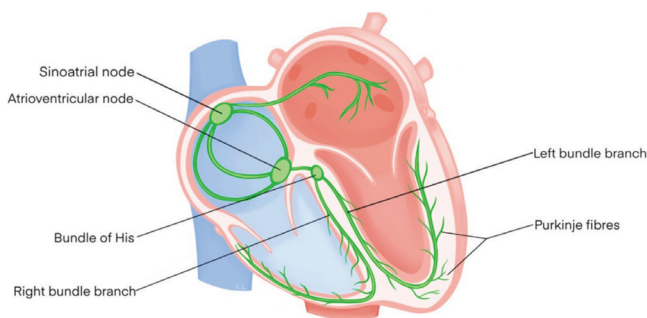


Figure 2: Structures within cardiac electrophysiology⁵

before splitting into the right and left bundle branch. Through depolarisation, contraction of the myocardial cells occurs, leading to the coordinated contraction of the heart muscle, which expels blood out of the ventricular cavities.⁵ Figure 2 depicts the electrophysiological structures of the heart.

What are the causes of bradycardia?

Bradycardia is caused by a variety of different conditions. Table 1 shows the various causes of bradycardia.^{5,6}

Physiological	Athletes
Cardiovascular	<ul style="list-style-type: none"> • Myocardial ischaemia/infarction • Myocarditis • Congenital cardiac disorders • Cardiomyopathies • His-Purkinje fibre degeneration e.g., AV block (1st/2nd/3rd degree) • Sinus node disease (e.g. sick sinus syndrome)
Metabolic/ Electrolyte Disturbance	<ul style="list-style-type: none"> • Hyperkalaemia • Hypoglycaemia • Hypercalcaemia • Hypothyroidism • Hypothermic
Neurological	<ul style="list-style-type: none"> • Raised ICP (intracranial pressure) • Cushing’s response • Vagal responses • Autonomic neuropathy including diabetic neuropathy
Infective	<ul style="list-style-type: none"> • Lyme’s disease • Diphtheria • Typhoid fever
Autoimmune/ Infiltrative	<ul style="list-style-type: none"> • Sarcoidosis • Amyloidosis • Systemic lupus erythematosus
Medication/ Poisons	<ul style="list-style-type: none"> • Beta-blockers • Digoxin • Calcium channel blockers • Amiodarone • Clonidine • Anaesthetic drugs • Organophosphate

Table 1: Causes of bradycardia^{5,6}

What is the management of bradycardia?

The acute management of bradycardia is based on the cause of the bradycardia, as well as whether the patient has “adverse features” which would indicate instability and risk of cardiac arrest.⁴ Advanced life support (ALS) defines patients with “adverse features”, as having signs of myocardial ischaemia, syncope, heart failure or shock.⁴ Table 2 highlights signs to look for to establish this. Remember, there may be an overlap in the above conditions. The pathophysiology of bradycardia leading to “adverse features” is due to low cardiac output (CO) leading to poor blood flow to the coronary arteries (leading to myocardial ischaemia), the brain (leading to

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Condition	Signs and symptoms
Myocardial ischaemia	Chest pain, shortness of breath, sweatiness, pallor, clamminess.
Syncope/pre-syncope	Loss of consciousness or unresponsiveness. Drop in GCS or dizziness may indicate pre-syncope.
Heart failure and shock	Bibasal crackles, shortness of breath due to pulmonary oedema, chest pain, prolonged peripheral and central capillary refill, unresponsive, hypotension.

Table 2: Signs of myocardial ischaemia, syncope, heart failure and shock^{2,4}

syncope) and the rest of the body (leading to heart failure and shock).³

Remember to always think about the cause of the bradycardia so that you can commence any specific treatment for any reversible causes identified. If there are any adverse features, ALS recommends administering atropine 500 micrograms and re-evaluating.⁴ This can be done to a maximum accumulated dosage of 3 milligrams.^{2,4} Alongside adverse features, there are other stand-alone criteria for administering atropine, which are: recent asystole, Mobitz type 2 AV block, complete heart block with broad QRS and ventricular pauses of more than 3 seconds.⁴ Isoprenaline, adrenaline and transcutaneous pacing are also recommended

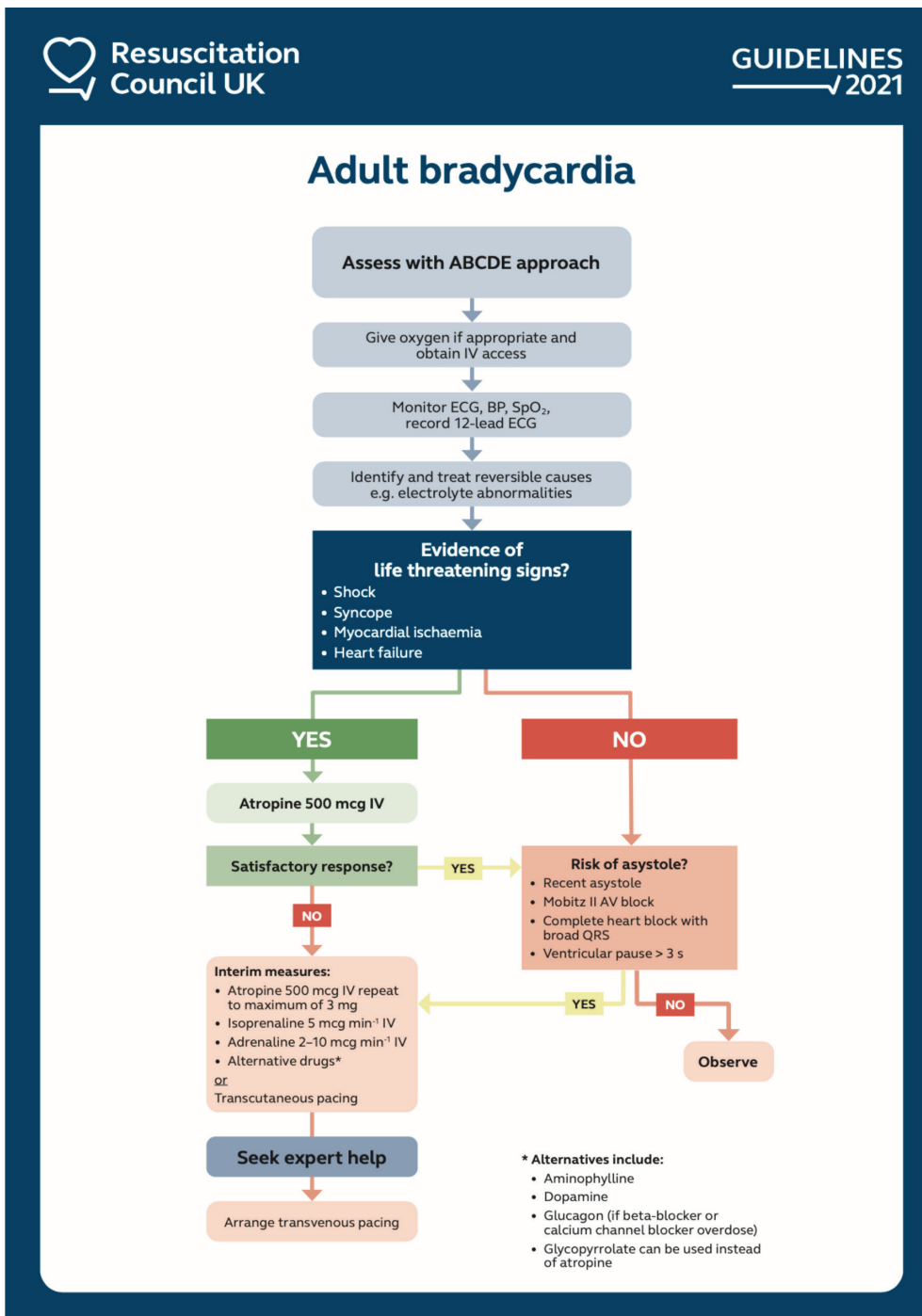


Figure 2: ALS algorithm for the management of bradycardia⁴

with specialist input.^{3,4} A summary of the ALS bradycardia management is shown in Figure 2.

How do the drugs used for treating bradycardia work?

Atropine works by antagonising muscarinic receptors, consequently blocking parasympathetic responses, and leading to unopposed sympathetic responses which increases the HR.⁷ Atropine is the first line as it can be bolused in an emergency.

Isoprenaline hydrochloride is a non-selective beta-adrenergic agonist which acts primarily on the beta-1 receptors of the heart and the beta-2 receptors of the bronchi. The effects of isoprenaline increase the heart rate and cardiac output. Isoprenaline does not affect blood pressure as adrenaline does.⁷ IV isoprenaline has a short half-life of 2.5-5 minutes, so an infusion is required for the effects to be continued. This takes time to set up in an emergency or ward environment.^{7,8}

Epinephrine, also known as adrenaline, is a naturally occurring hormone secreted by the medulla of the adrenal glands. It is considered a sympathomimetic drug, as it activates the adrenergic (sympathetic) response on effector cells across the body.⁷ It works on both alpha and beta-adrenergic receptors. This means it also causes an increase in BP alongside the HR. It may be useful in someone with bradycardia and hypotension.⁷ Isoprenaline and adrenaline should always be initiated by a senior, experienced member of the team.² Initiating these drugs would necessitate the patient being moved to an appropriate environment such as a coronary care unit, high dependency unit or an intensive care unit.

Transcutaneous pacing takes over the SA node in the patient's heart and mechanically paces the electrical activity. It is done by connecting the patient to pads from a defibrillator in a "pacing setting".⁹ The defibrillator will then produce a current that causes the heart to contract. Transcutaneous pacing is indicated when unstable bradycardias are resistant to medications such as atropine and isoprenaline/adrenaline.⁹ Senior input is always needed, as the patient needs sedation. This is a short-term treatment, and the patient will likely go on to require a more permanent solution such as a temporary pacing wire or permanent pacemaker inserted in the cath lab, under cardiology.^{2,9}

Always consider rate-limiting medication as a potential cause of bradycardia, for example in deliberate overdose or because of a change in the patient's physiology such as renal failure.^{5,6} If the offending medication is a beta-blocker or calcium channel blocker, glucagon may be indicated.^{4,7} If digoxin is considered a possible cause of bradycardia, then digoxin specific antibodies may be considered.¹⁰

Outcome of Case

When the MET team arrived, the patient deteriorated with continued bradycardia and hypotension. Further doses of atropine were required to keep the patient stable. The team contacted the on-call cardiology registrar at the local electrophysiology and intervention centre, and he was accepted for transfer. The patient required emergency percutaneous intervention and fortunately made a good recovery. In the debrief after the patient was transferred,

the medical registrar spoke to the juniors about how the most common pathological cause of sinus bradycardia was acute myocardial infarction. She said that this happens particularly with inferior infarcts affecting the right coronary artery, as this usually supplies the SA and AV nodes.¹¹

DISCLAIMER

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