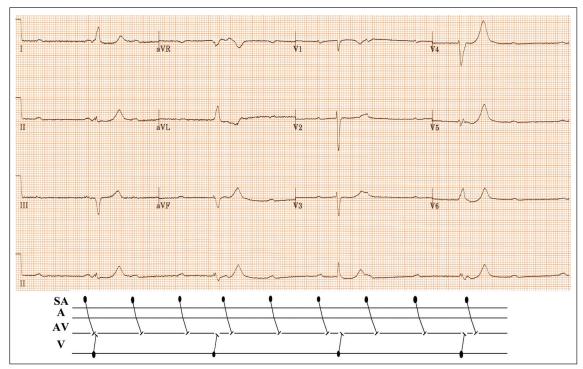
# **CME Article** Variations of atrioventricular block

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**Fig. 1** 12-lead ECG shows broad QRS complex bradycardia with atrioventricular (AV) dissociation. The P waves are independent of the QRS complexes. This is classical of third-degree AV block. The laddergram beneath the ECG shows failure of conduction of all the P waves. The QRS complexes are wide, indicating that the escape rhythm is arising low down from the ventricle (SA: sinoatrial node; A: atrium; AV: atrioventricular node; V: ventricle).

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## CASE I CLINICAL PRESENTATION

An 80-year-old man presented with an episode of syncope preceded by non-vertiginous giddiness. Paramedics attending to him noted a pulse rate of 27 beats per minute (bpm). At the emergency department, intravenous atropine 1.2 mg was administered with minimal improvement in heart rate. Comment on the electrocardiogram (ECG) abnormalities in Fig. 1? What is the ECG diagnosis?

## ECG INTERPRETATION

Fig. 1 shows atrioventricular dissociation. The P waves and QRS complexes have no temporal relationship. The PP interval is regular, with an atrial rate of about 67 bpm, while the RR interval, which is also regular, is noted to be 27 bpm. The QRS complexes are wide at 120–160 milliseconds. This indicates third-degree atrioventricular (AV) block (or complete heart block), with escape rhythm arising from the ventricle (and below the bundle of His).

### **CLINICAL COURSE**

The patient underwent transvenous pacing on admission. A coronary angiogram was performed in view of the mildly elevated cardiac enzymes. This showed moderate narrowing of the proximal left anterior descending artery, which was treated medically. He also underwent a separate procedure during his admission. Post-procedure ECG (Fig. 2) showed a regular ventricular rate of 50 bpm. There were vertical spikes preceding each QRS complex, which had a left bundle branch block morphology. This ECG is typical of right ventricular pacing. Subsequently, the patient had a permanent pacemaker (VVIR) implanted during admission. He has been asymptomatic since then. Chest radiography performed post procedure is shown in Fig. 3.

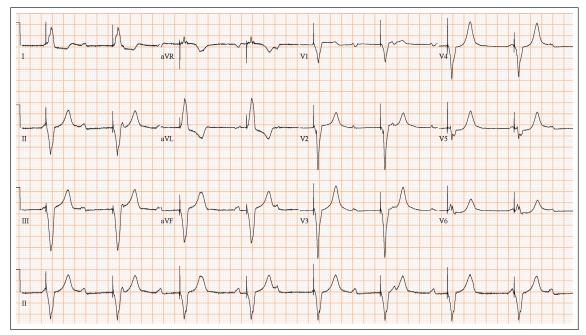


Fig. 2 12-lead ECG shows regular, broad QRS complexes preceded by a pacing spike. The ventricular rate is 50 bpm and atrioventricular dissociation is present, as the P waves are not temporally related to the QRS complexes. This demonstrates right ventricular pacing.

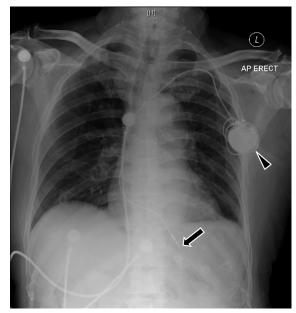
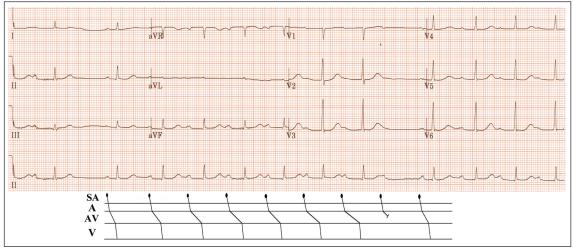


Fig. 3 Chest radiograph shows the presence of a permanent pacemaker in the left pectoralis region. The pulse generator (arrowhead) is seen close to the left axilla, and is a singlechamber pacemaker, as there is only one lead extending from the subclavian vein to the right ventricle of the heart, which is the chamber that is being paced. The tip of the pacemaker lead (arrow) rests on the inter-ventricular septum, below the right ventricular outflow tract. The ideal position can be reviewed with a concurrent lateral chest radiograph following insertion.



**Fig. 4** 12-lead ECG shows progressive prolongation of the PR interval leading to a dropped beat in a 8:7 ratio. These features are consistent with Mobitz Type I atrioventricular (AV) block. The accompanying laddergram depicts conduction from the sinoatrial node through the atria, AV node and finally the ventricles. The progressive prolongation of the PR interval is clearly seen at the level of the AV node. This culminates in a dropped beat where the impulse fails to conduct down the AV node toward the ventricles (SA: sinoatrial node; A: atrium; AV: atrioventricular node; V: ventricle).

# CASE 2

#### **CLINICAL PRESENTATION**

An 82-year-old woman with multiple comorbidities, including diabetes mellitus, previous stroke, hypertension and hyperlipidaemia, was admitted for drowsiness. Paramedics had found her blood sugar to be 1.6 mmol/L. Her hypoglycaemia and symptoms were resolved with intravenous glucose administration. A referral to the cardiology service was made following assessment of her routine ECG done during admission (Fig. 4). What is the ECG diagnosis?

#### ECG INTERPRETATION

There is sinus rhythm with progressive prolongation of the PR interval until failure of conduction of a P wave, as well as a progressive decrease in the RR interval. The RR interval containing the blocked P wave (1.30 sec) is shorter than the sum of two PP intervals (1.36 sec) preceding the blocked P wave. This is a second-degree Mobitz Type I AV block or the Wenckebach phenomenon.

#### **CLINICAL COURSE**

The patient remained asymptomatic during admission following the correction of her hypoglycaemia. Despite the ECG findings, she did not complain of any giddiness or lethargy. She was discharged well, with no further cardiac intervention.

#### DISCUSSION

AV conduction delay or block refers to the delay or interruption of electrical impulses from the atria to the ventricles due to an abnormality in the conduction system. There are typically three forms of AV block: first-, second- and third-degree. In first-degree AV block, the PR is prolonged, but there are no blocked P waves. Second-degree AV block can be divided into (a) Mobitz Type I AV block or the Wenckebach phenomenon; (b) Mobitz Type II AV block; and (c) high-grade AV block (e.g. 2:1, 3:1).

The patient in Case 2 demonstrated Mobitz Type I AV block. Here, the PR interval becomes gradually more prolonged and is associated with a decreasing RR interval, finally ending with a failure of the P wave to conduct. The absolute increase in PR interval decreases with every successive beat until the AV block occurs. This mechanism also leads to a decreasing RR interval and hence, a shorter RR interval containing the dropped beat compared to the sum of two PP intervals preceding the dropped beat.

Mobitz Type I AV block most commonly occurs at the level of the AV node (75% of the cases), with the remaining 25% being situated in the bundle of His, distal to the AV node (infranodal).<sup>(1)</sup> The common causes of Mobitz Type I AV block include an increased vagal tone in normal individuals (usually athletes), inferior ST elevation myocardial infarction, degenerative changes of the conduction system in the elderly, underlying heart disease affecting the conduction system (mainly the AV node), and medications that block the AV node (eg. beta blockers). Finally, electrolyte and endocrine abnormalities (e.g. hypokalaemia) not uncommonly contribute to AV block as well.<sup>(2)</sup>

In normal individuals, especially well-trained endurance athletes, there is increased parasympathetic activity contributed by prolonged training. Symptoms are rare and there is minimal impact on daily activities. The abnormal ECG findings are known to resolve with de-training.<sup>(3)</sup> Any cardiac condition that affects the AV node can result in AV block. These include intrinsic AV nodal disease, myocarditis, myocardial ischaemia (especially involving the right coronary artery, as it supplies the AV node), as well as instrumentation from cardiac surgery.

AV-nodal, beta-adrenergic, non-dihydropyridine calcium channel blocking agents and cardiac glycosides (such as digoxin) are commonly prescribed for a variety of cardiac conditions, and may contribute to significant AV block, especially in the elderly. Routine scrutiny of the patient's medication list is important, as AV block in such instances is potentially completely reversible. The same can be said for patients with hypokalaemia, as treatment of this condition also results in the resolution of AV block.

In general, Mobitz Type I AV block does not result in symptoms unless there is significant bradycardia. It is considered a benign condition that does not require specific treatment apart from assessing and managing possible underlying causes such as revascularisation in myocardial ischaemia or discontinuing common culprit medications.

Third-degree AV block or complete heart block is caused by a conduction block at either the level of the AV node, the bundle of His or the bundle-branch Purkinje system.<sup>(4)</sup> When this occurs, an escape rhythm is triggered distal to the block. The duration of the escape rhythm (QRS complex) depends on the site of the block and the site of the escape rhythm pacemaker. The subject, as described in Case 1, had a ventricular escape rhythm manifesting as broad QRS complexes at a significantly slower rate. This is typical of a conduction block occurring below the bundle of His, usually at the level of the bundle-branch/Purkinje system (also known as infra-Hisian or infra-nodal block).<sup>(1)</sup> Patients with this type of block are commonly haemodynamically unstable, and their heart rates do not respond to exercise or atropine.<sup>(5,6)</sup> In such situations, correction of the bradycardia by cardiac pacing should to be done urgently so as to minimise any potential consequences of hypoperfusion (see below).

In general, the more distal the conduction block, the slower the ventricular escape rate. Heart rates < 40 bpm are often hazardous, and patients commonly present with symptoms such as dizziness, pre-syncope or syncope (Stokes-Adams attack).<sup>(7)</sup> Additionally, profound bradycardia can also worsen pre-existing heart failure or angina. This is in contrast to the narrow QRS complex seen in third-degree AV block, where the ventricular escape mechanism arises from above the bundle of His, likely at the level of the AV node. Consequently, a junctional pacemaker with a rate of 45–60 bpm may be present. Patients with complete heart block at this level are frequently haemodynamically stable and their heart rate may respond to exercise and atropine. In the absence of symptoms, urgent correction of bradycardia may not be necessary.

The causes of complete heart block may be divided into congenital or acquired. Congenital complete heart block is rare and is associated with maternal antibodies to SS-A (Ro) and SS-B (La). Acquired causes include those leading to Mobitz Type I AV block discussed earlier. Medications, ischaemic heart disease and idiopathic progressive cardiac conduction disease are three very common causes, especially in the elderly. In any patient with sudden onset of complete heart block, acute ischaemia as a precipitating factor has to be excluded. Fibrosis and sclerosis of the conduction system accounts for about half of the cases of AV block, and involvement of the mitral ring or central fibrous body may be the most common cause of complete heart block with a narrow QRS complex in the elderly.

Treatment of patients with complete heart block involves correcting all reversible causes (if any) as per management of Mobitz Type I AV block mentioned earlier. In addition, implantation of a permanent pacemaker should to be considered in selected patients based on recommendations of the 2008 American College of Cardiology/American Heart Association/Heart Rhythm Society device guidelines.<sup>(8)</sup> Class I indications include individuals with symptomatic bradycardia, ventricular pauses  $\geq$  3 sec, a resting heart rate < 40 bpm while awake, or those who require anti-arrhythmic medication resulting in symptomatic bradycardia.

#### ABSTRACT

Atrioventricular (AV) block comprises a spectrum of cardiac conduction delays with varying clinical presentations. It is commonly encountered in both hospital as well as ambulatory settings, and recognition of the type of AV conduction delay is essential for appropriate subsequent management. The electrocardiogram is a key tool for identification of patients with AV conduction delays. Contrasting management strategies should be employed for differing levels of conduction block.

Keywords: complete heart block, Mobitz Type I atrioventricular block, second-degree heart block, third-degree heart block

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#### REFERENCES

- Peuch P, Grolleau R, Guimond C. Incidence of different types of A-V block and their localization by His bundle recordings. In: Wellens HJJ, Lie KI, Janse MJ, eds. The Conduction System of the Heart. Stenfert Kroese: Leiden, 1976: 467.
- 2. Major causes of atrioventricular block. UpToDate 2011 ver 19.1.
- Zeppilli P, Fenici R, Sassara M, Pirrami MM, Caselli G. Wenckebach second-degree A-V block in top-ranking athletes: an old problem revisited. Am Heart J 1980; 100:281-94.
- 4. Narula OS, Javier RP, Samet P, Maramba LC. Significance of His and left bundle recordings from the left heart in man. Circulation 1970; 42:385-96.
- Schweitzer P, Mark H. The effect of atropine on cardiac arrhythmias and conduction. Part 1. Am Heart J 1980; 100:119-27.

- Schweitzer P, Mark H. The effect of atropine on cardiac arrhythmias and conduction. Part 2. Am Heart J 1980; 100:255-61.
- Ezri M, Lerman BB, Marchlinski FE, Buxton AE, Josephson ME. Electrophysiologic evaluation of syncope in patients with bifascicular block. Am Heart J 1983; 106 (4 Pt 1):693-7.
- 8. Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices): developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. Circulation 2008; 117:e350-408.

## SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME Multiple Choice Questions (Code SMJ 201105A)

Oresting 1 Three fratures are seen as the ECC is consolet, heart block	True	False
<ul><li>Question 1. These features are seen on the ECG in complete heart block:</li><li>(a) AV dissociation.</li></ul>		
(b) Regular PP interval.		
(c) Regular RR interval.		
(d) ST segment elevation.		
Question 2. The following symptoms may be present in complete heart block:		
(a) Dizziness and giddiness.		
(b) Syncope and pre-syncope.		
(c) Heart failure.		
(d) Angina.		
Question 3. Common causes of complete heart block include:	_	_
(a) Hyperthyroidism.		
(b) Ischaemic heart disease.		Ц
(c) Age-related degeneration of the cardiac conduction system.		Ц
(d) Congenital.		
Question 4. These medications commonly cause complete heart block:	_	_
(a) Beta blockers.		Ц
(b) Calcium channel blockers (non-dihydropyridine).		
(c) Cardiac glycosides (e.g. digoxin).		
(d) Diuretics (e.g. frusemide).		
Question 5. The following are Class I indications for permanent pacemaker implantation:	_	_
(a) Symptomatic third-degree AV block.		Ц
(b) Ventricular pause $> 3$ sec.		
(c) Resting awake heart rate $< 40$ bpm.		
(d) Patients requiring anti-arrhythmics causing symptomatic bradycardia.		

Doctor's particulars:	
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MCR number:	Specialty:
Email address:	
SUBMISSION INSTRUCTIONS: (1) Log on at the SMJ website: http://www.sma.org.sg/cme/smj and s address and MCR number. Click on "Submit answers" to submit.	select the appropriate set of questions. (2) Select your answers and provide your name, email

#### **RESULTS:**

(1) Answers will be published in the SMJ July 2011 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 01 July 2011. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (May 2011 SMJ 3B CME programme): 12 noon, 24 June 2011.