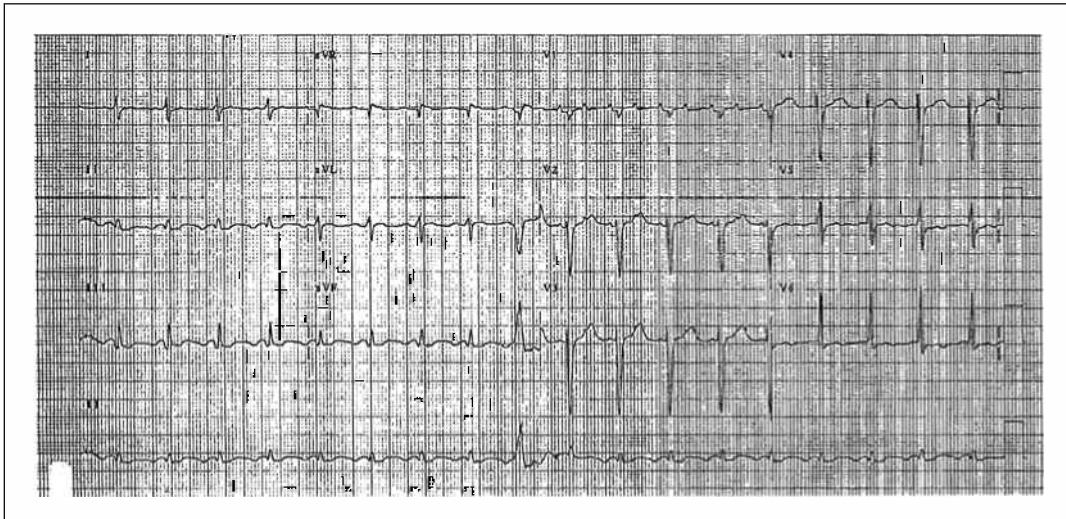
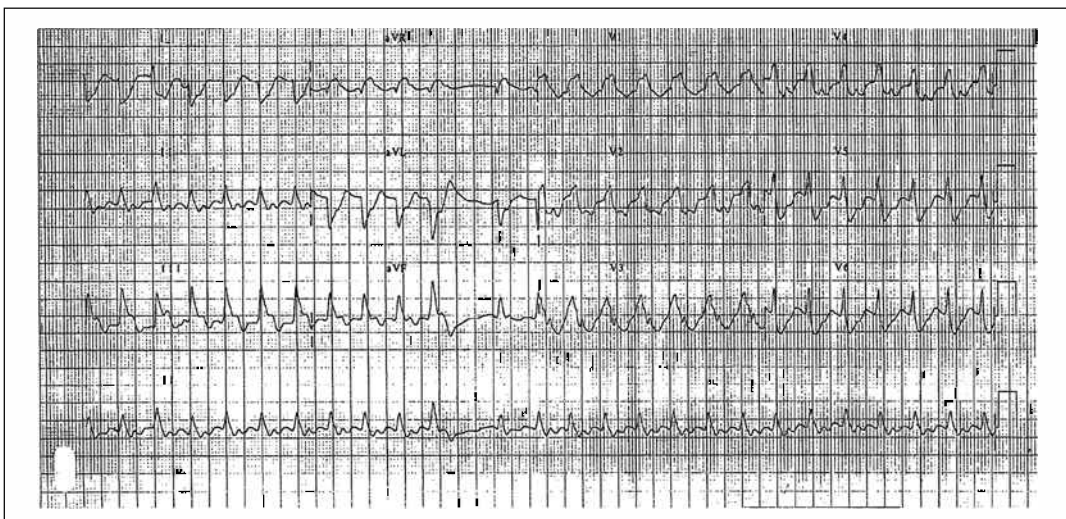


**CME Article****Electrocardiographical case. Elderly man with acute breathlessness**

Sim D, Chia S, Ching C K, Chua T

**Fig. 1** Initial 12-lead electrocardiogram.**Fig. 2** 12-lead electrocardiogram obtained on the sixth day of admission.**CLINICAL PRESENTATION**

A 67-year-old Chinese man presented to the emergency department complaining of abdominal bloating and shortness of breath of several days duration. He had a history of ischaemic cardiomyopathy, chronic renal impairment, chronic obstructive pulmonary disease, hepatic cirrhosis, and type II diabetes mellitus. On examination, he was alert and haemodynamically stable, with a blood pressure of 120/70 mmHg. A 12-lead electrocardiogram (ECG) was performed (Fig. 1). Intravenous frusemide was commenced for treatment

of congestive heart failure and the patient's symptoms gradually improved over the next two days.

On the sixth day of admission, he developed acute onset of breathlessness while at rest. ECG was performed (Fig. 2) and intravenous amiodarone infusion was initiated. Shortly after, he was witnessed to develop a fit (ECG recording was not obtained during the fit). Potassium level was 7.2 mmol/L. What do the initial (Fig. 1) and subsequent (Fig. 2) ECGs show? What is your diagnosis?

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### ECG INTERPRETATION

Initial ECG (Fig. 1) showed atrial tachycardia (AT) with 2:1 atrioventricular (AV) conduction with atrial rate of 220/minute and ventricular rate of 110/minute (P waves are best seen in lead V1). The regular broad-complex tachycardia with right bundle branch block (RBBB) morphology in the repeat ECG (Fig. 2) is AT with 1:1 AV conduction and hyperkalaemia-induced intraventricular conduction delay. In view of the prior history of ischaemic cardiomyopathy, the differential diagnosis for the repeat ECG (Fig. 2) is ventricular tachycardia. In this case of widened QRS complex with right bundle block morphology, the triphasic RBBB pattern (rSR') in lead V1 and RS pattern in lead V6, with R wave height greater than S wave depth, favour supraventricular tachycardia with aberrant conduction. The absence of AV dissociation, indeterminate axis (QRS axis in the northwest quadrant), concordant pattern, fusion and capture beat also makes ventricular tachycardia less probable.

### DIAGNOSIS

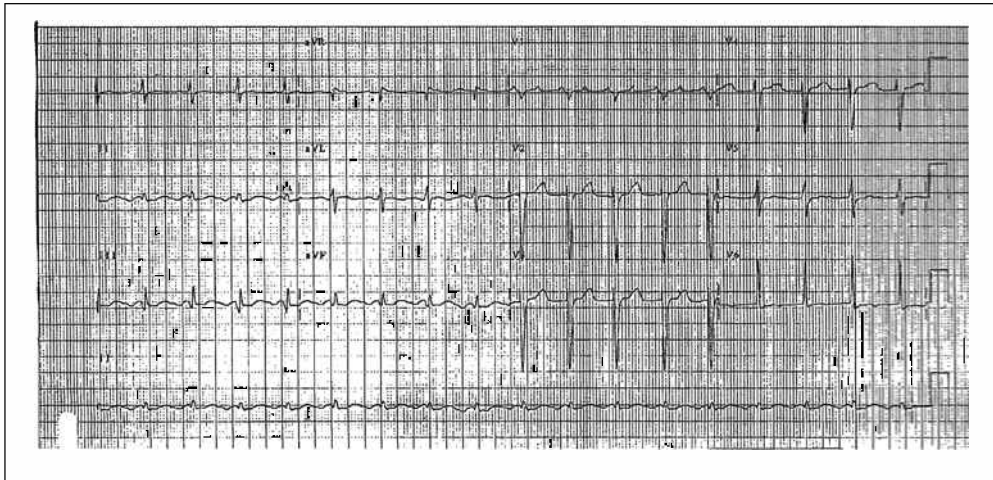
Atrial tachycardia with variable AV conduction block and transient hyperkalaemia-induced aberrant conduction.

### CLINICAL COURSE

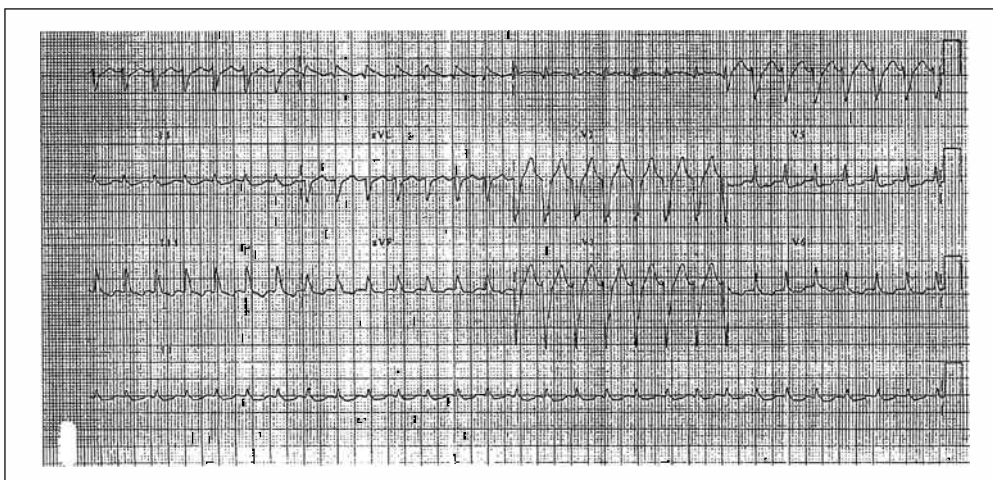
He was treated with intravenous calcium gluconate, insulin and 50% dextrose solution. Ten minutes later, the wide-complex tachycardia reverted to baseline narrow-complex AT with 2:1 AV conduction (Fig. 3). On the following day, the patient developed recurrent wide-complex tachycardia (Fig. 4) which was again treated with intravenous amiodarone infusion and oral resonium. The rhythm reverted to baseline AT, and bisoprolol was added to the treatment regimen. Unfortunately, the patient subsequently developed acute renal failure, acute ischaemic hepatitis and overwhelming sepsis. He died one week later.

### DISCUSSION

AT is characterised by regular atrial activation from atrial areas with centrifugal spread.<sup>(1)</sup> The atrial rate is generally between 150 and 250 beats/minute, and rarely exceeds 300 beats/minute. The prevalence of AT has been estimated to be 0.3% in asymptomatic patients and 0.5% in symptomatic patients.<sup>(2)</sup> Sustained forms of AT may lead to tachycardia-induced cardiomyopathy.<sup>(3)</sup> Although AT can manifest in patients without structural heart disease,



**Fig. 3** 12-lead electrocardiogram obtained after correction of hyperkalaemia.



**Fig. 4** 12-lead electrocardiogram shows recurrence of wide complex tachycardia.

it is more commonly associated with underlying cardiac abnormalities.<sup>(4)</sup> AT associated with AV dissociation may also be caused by digitalis toxicity, and often exacerbated by hypokalaemia.

In AT, the P waves are usually located in the second half of the tachycardia cycle (long R-P/short P-R tachycardia), and frequently obscured by the T waves of the preceding QRS complex. At high atrial rates, Wenckebach phenomenon may occur, a feature that can help to distinguish AT from AV re-entrant tachycardia or AV nodal reentrant tachycardia. Although the absence of regular flutter waves is useful to differentiate atrial flutter from AT, this can be difficult at rapid atrial rates with P wave morphology mimicking atrial flutter.<sup>(1)</sup>

The P wave morphology in AT and sinus tachycardia are different, and analysis of the P wave configuration may be useful in localising the origin of AT. Positive P waves in lead V1 and negative P waves in lead I or aVL predict a left atrial focus, while negative P wave in lead V1 and negative P waves in lead I predict a right atrial focus. In addition, positive and negative P waves in the inferior leads favours cranial and caudal foci, respectively. In the left atrium, the AT foci cluster over the pulmonary vein, atrial septum, and mitral annulus.<sup>(5)</sup> The majority of right-sided AT originates along the crista terminalis within the right appendage and coronary coronary sinus ostium.<sup>(6)</sup> Three types of AT have been distinguished: enhanced automaticity, triggered activity, and micro-reentrant. In most cases, the mechanism of focal discharge cannot be ascertained clinically. However, the presence of progressive increase of the atrial rate at onset of tachycardia ("warming up") and progressive decrease before termination of tachycardia ("cooling down") are suggestive of an automatic mechanism.

The objective of initial therapy for AT, which includes intravenous or oral  $\beta$ -receptor antagonists and calcium channel antagonists, is to achieve rate control or more rarely, termination of the tachycardia. Alternatively, Class IA, IC or III anti-arrhythmic drugs may be used to suppress the tachycardia focus directly. As AT is often associated with structural heart disease, amiodarone may be preferred over class I drugs unless coronary artery disease or left ventricular impairment have been excluded. In pharmacotherapy-resistant AT, a trial of electrical DC cardioversion should be considered in the haemodynamically-unstable patient. In patients with digitalis toxicity, administration of digitalis antibodies and potassium replacement should be considered, although discontinuation of digoxin is often the only necessary measure. For pharmacotherapy-resistant AT, radiofrequency catheter ablation is usually effective in eliminating the tachycardia focus. However, recurrence of AT may occur from a different focus following

initial successful ablation. Data from 105 patients who underwent catheter ablation for AT showed a 77% success rate, with the highest incidence of recurrence found in the anterior right atrial foci.<sup>(7)</sup> AT remains an uncommon arrhythmia that is difficult to manage. However, recent advances in delineating the mechanism and anatomic locations of focal AT, and the advent of radiofrequency ablation have changed our approach to the management of these difficult arrhythmias.

## ABSTRACT

**A 67-year-old Chinese man presented to the emergency department with a history of abdominal bloating and shortness of breath. Initial electrocardiogram (ECG) showed atrial tachycardia (AT) with 2:1 atrioventricular (AV) conduction block. Six days after admission, he developed acute dyspnoea and confusion. Repeat ECG demonstrated a regular wide-complex tachycardia. Serum analysis revealed hyperkalaemia secondary to acute on chronic renal failure. Emergency treatment with intravenous calcium gluconate, 50% dextrose solution and short-acting insulin was instituted. The ECG promptly reverted to a narrow-complex AT with 2:1 AV conduction block. The diagnosis and treatment of AT are discussed.**

**Keywords: atrial tachycardia, atrioventricular conduction block, broad complex tachycardia, tachycardia**

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**SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME**  
**Multiple Choice Questions (Code SMJ 200705B)**

	<b>True</b>	<b>False</b>
<b>Question 1.</b> ECG features favouring VT over SVT with aberrancy include:		
(a) AV dissociation.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Capture and fusion beats.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Concordant pattern.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Indeterminate QRS axis.	<input type="checkbox"/>	<input type="checkbox"/>
 <b>Question 2.</b> With regard to AT:		
(a) The atrial rate usually exceeds 300 beats per minute.	<input type="checkbox"/>	<input type="checkbox"/>
(b) A sustained form may lead to cardiomyopathy.	<input type="checkbox"/>	<input type="checkbox"/>
(c) It is commonly associated with underlying cardiac abnormalities.	<input type="checkbox"/>	<input type="checkbox"/>
(d) It is associated with digitalis toxicity.	<input type="checkbox"/>	<input type="checkbox"/>
 <b>Question 3.</b> The following are ECG features of AT:		
(a) Short R-P interval.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Wenckebach phenomenon at high atrial rate.	<input type="checkbox"/>	<input type="checkbox"/>
(c) It mimics atrial flutter at rapid atrial rate.	<input type="checkbox"/>	<input type="checkbox"/>
(d) P wave morphology in AT and sinus tachycardia are similar.	<input type="checkbox"/>	<input type="checkbox"/>
 <b>Question 4.</b> The mechanism of AT include:		
(a) Triggered activity.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Enhanced automaticity.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Micro-reentrant.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Accessory pathway.	<input type="checkbox"/>	<input type="checkbox"/>
 <b>Question 5.</b> Pharmacotherapy for AT includes:		
(a) Beta blockers.	<input type="checkbox"/>	<input type="checkbox"/>
(b) Calcium channel blockers.	<input type="checkbox"/>	<input type="checkbox"/>
(c) Digoxin.	<input type="checkbox"/>	<input type="checkbox"/>
(d) Amiodarone.	<input type="checkbox"/>	<input type="checkbox"/>

**Doctor's particulars:**

Name in full: \_\_\_\_\_

MCR number: \_\_\_\_\_ Specialty: \_\_\_\_\_

Email address: \_\_\_\_\_

**SUBMISSION INSTRUCTIONS:**

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

**RESULTS:**

(1) Answers will be published in the SMJ July 2007 issue. (2) The MCR numbers of successful candidates will be posted online at [www.sma.org.sg/cme/smj](http://www.sma.org.sg/cme/smj) by 15 July 2007. (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 2007 SMJ 3B CME programme): 12 noon, 25 June 2007**