

CMEARTICLE

Abnormal ECGs secondary to electrolyte abnormalities

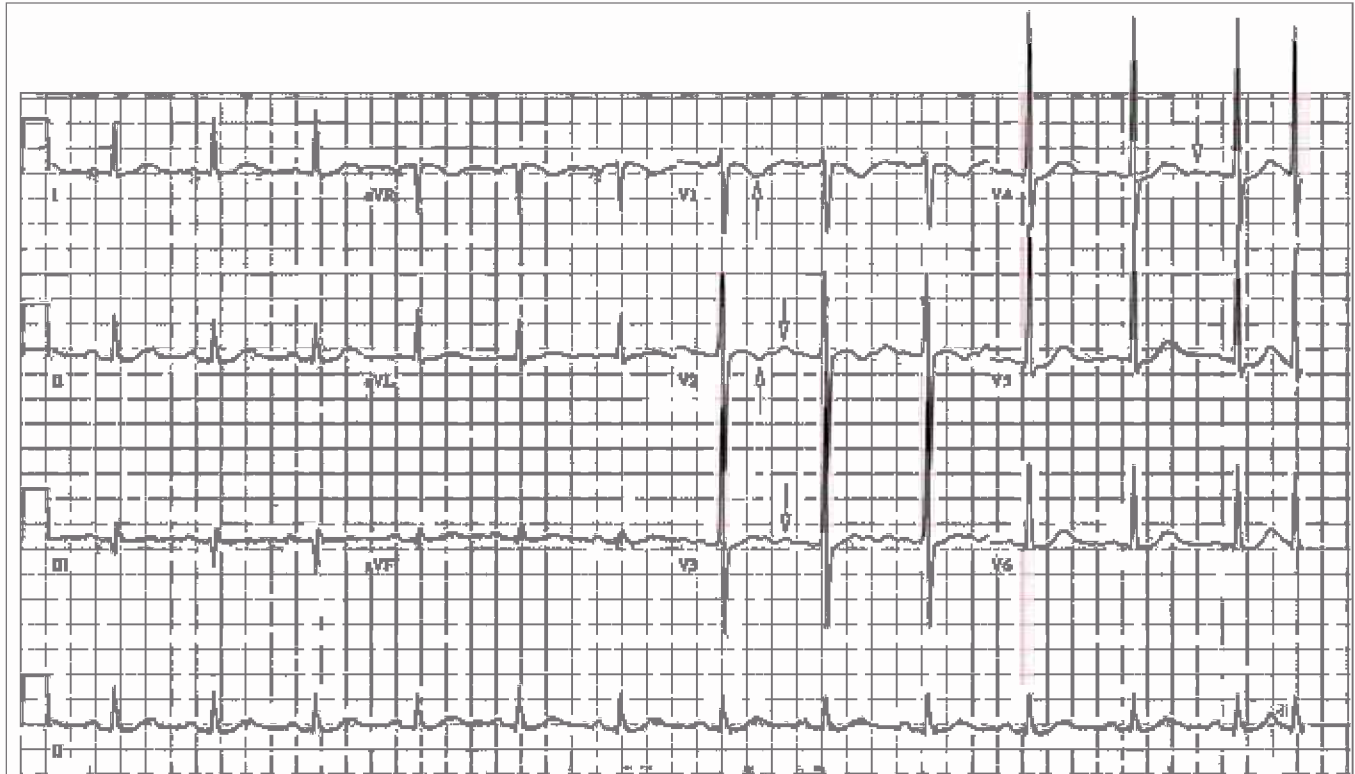
Kuntjoro I¹, MD, Teo SG¹, MBBS, MRCP, Poh KK^{1,2}, FRCP, FACC

Fig. 1 Case 1. ECG shows prominent U wave (downward arrows) in leads V2, V3 and V4 as well as T wave inversion in leads V1 and V2 (upward arrows). The T wave in lead V3 is upright but has the same height ratio as the U wave.

CASE 1

CLINICAL PRESENTATION

A 70-year-old man was admitted from the clinic with abnormal laboratory results. He was known to have 'difficult to control' hypertension over the past few years, and was on Hydralazine, Exforge® (amlodipine and valsartan), Atenolol, Coversyl and Atorvastatin therapy. He had complaints of tiredness with generalised weakness and myalgia. Examination revealed a blood pressure of 155/70 with an essentially normal cardiac and neurological examination. What are the electrocardiography (ECG) abnormalities seen in Fig. 1?

ECG INTERPRETATION

ECG shows normal sinus rhythm with prominent U waves in leads V2, V3 and V4. The T waves in leads V1 and V2 are inverted (Fig. 1). These changes are associated with severe hypokalaemia. As the hypokalaemia worsened, the U wave had become taller than the T wave. The depth of S1 plus the height of R5 measured around 48 mm, and this is indicative of left ventricular hypertrophy.⁽¹⁾

CLINICAL COURSE

The patient's potassium level was 2.1 mmol/L on admission. Intravenous replacement was initiated, followed by oral administration. Further workup revealed normal urine potassium, cortisol level (8 am) 699 (normal range 123–626) nmol/L, normal calcium, phosphate and magnesium levels, as well as normal supine renin and aldosterone levels. A diagnosis of Conn syndrome was considered in the setting of hypertension and hypokalaemia. The patient was started on high-dose potassium replacement. ECG was repeated when his serum potassium level was 3.8 mmol/L (Fig. 2). Repeat renin/aldosterone measurement (off angiotensin-converting-enzyme [ACE] inhibitor) revealed suppressed renin and elevated aldosterone levels. Subsequent computed tomography (CT) of the adrenal gland showed a 1.2 cm × 0.9 cm nodule arising from the lateral limb of the right adrenal gland. However, the patient declined further investigations and continued on long-term potassium replacement therapy.

¹Cardiac Department, National University Heart Centre, ²Yong Loo Lin School of Medicine, National University of Singapore, Singapore

Correspondence: A/Prof Poh Kian Keong, Senior Consultant and Associate Professor, Yong Loo Lin School of Medicine, National University of Singapore, 1E Kent Ridge Road NUHS Tower Block, Level 9, Singapore 119228. kian_keong_poh@nuhs.edu.sg

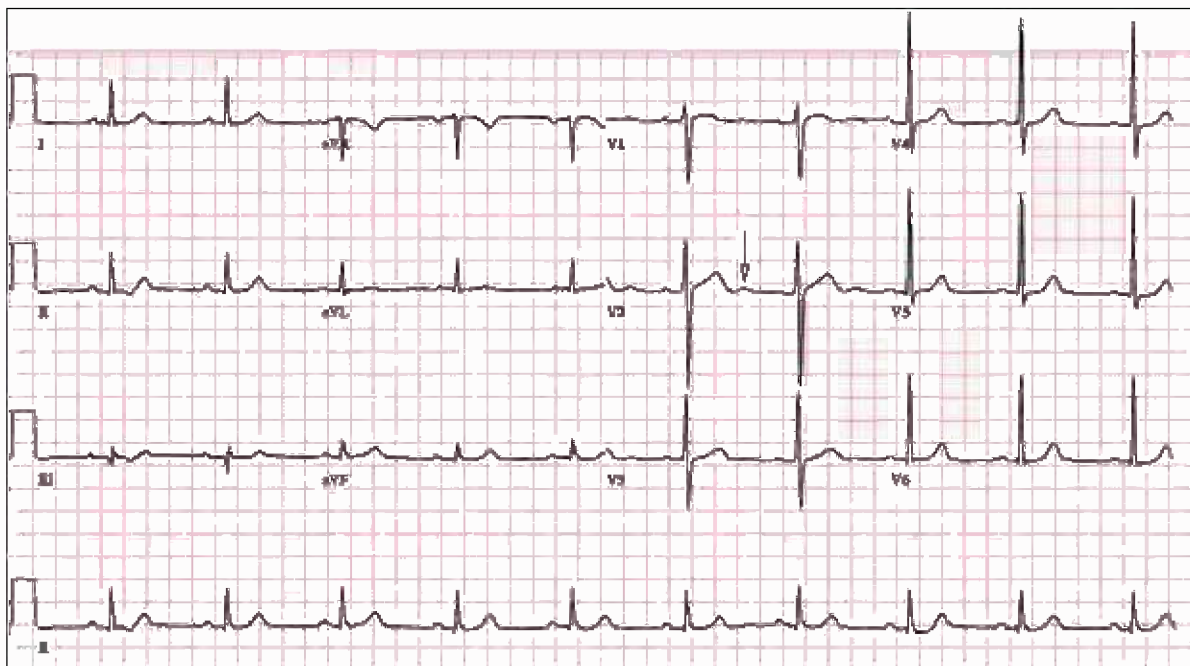


Fig. 2 Case 1. ECG shows that the U wave (arrow) is not as prominent as that in the first ECG. T wave inversion, previously seen in leads V1 and V2, has resolved.

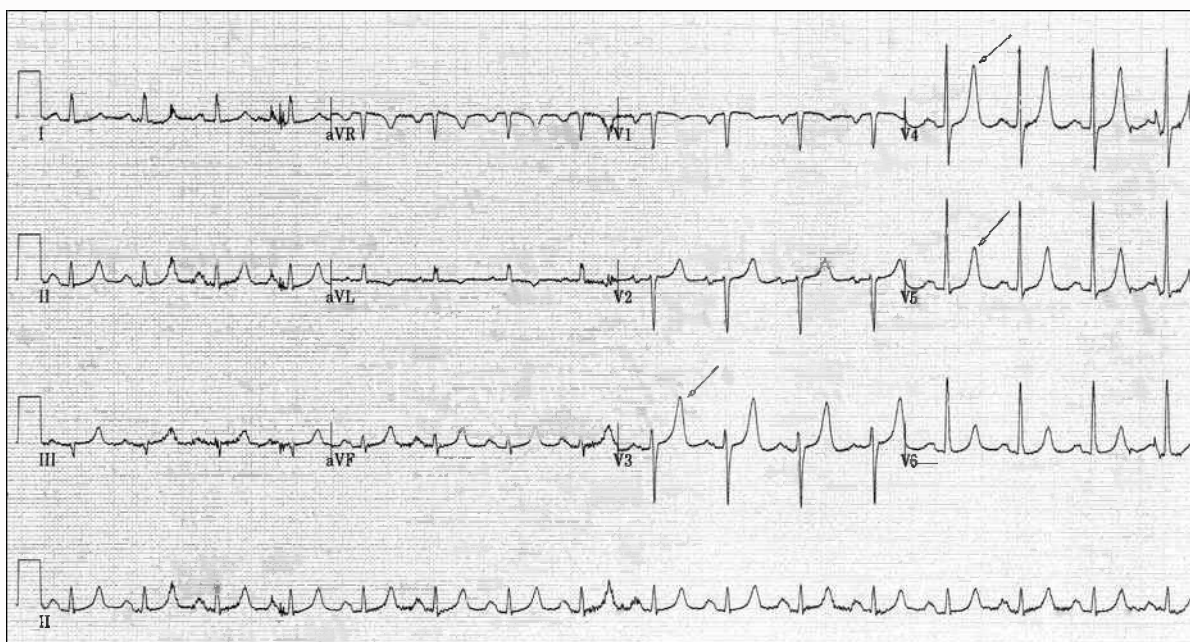


Fig. 3 Case 2. ECG shows normal sinus rhythm with tall, peaked, narrow-based and symmetrical T waves (arrows). Note the ‘P-mitrale’ in lead II and widened negative deflection of P wave in lead V1.

**CASE 2
CLINICAL PRESENTATION**

A 54-year-old woman with end-stage renal failure due to glomerulonephritis status, post-failed cadaveric kidney transplant, was admitted with acute onset of shortness of breath. She was on regular haemodialysis. Her clinical examination and radiographic findings were consistent with acute pulmonary oedema. What are the ECG abnormalities seen in Fig. 3?

ECG INTERPRETATION

ECG (Fig. 3) shows normal sinus rhythm with tall T waves best seen in leads V3, V4 and V5. The tall T wave is symmetrical, narrow, scooped inwards and ‘tentled’. This classic appearance

of the T wave is usually associated with hyperkalaemia. The PR interval, QRS duration and QTc interval are all within normal range. The P wave is wide (> 0.12 sec in duration) and bifid in shape – so called ‘P-mitrale’ in lead II. There is a negative component in lead V1, which is widened and slurred, consistent with left atrial enlargement.

CLINICAL COURSE

The patient’s potassium level was elevated on admission, and in the presence of acute pulmonary oedema, she was sent for urgent dialysis. Her potassium post haemodialysis was normalised. This was also associated with normalisation of the ECG T wave morphology.

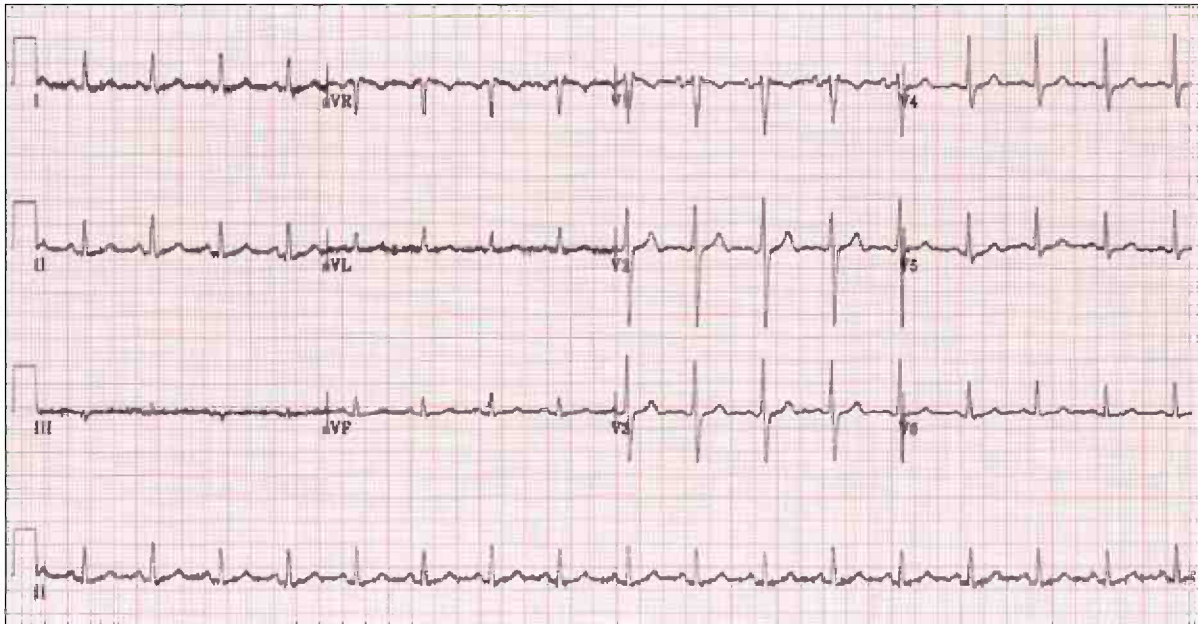


Fig. 4 Case 3. ECG shows sinus tachycardia with normal QRS duration and normal QTc interval. In lead V1, the QRS complex shows an incomplete right bundle branch pattern.

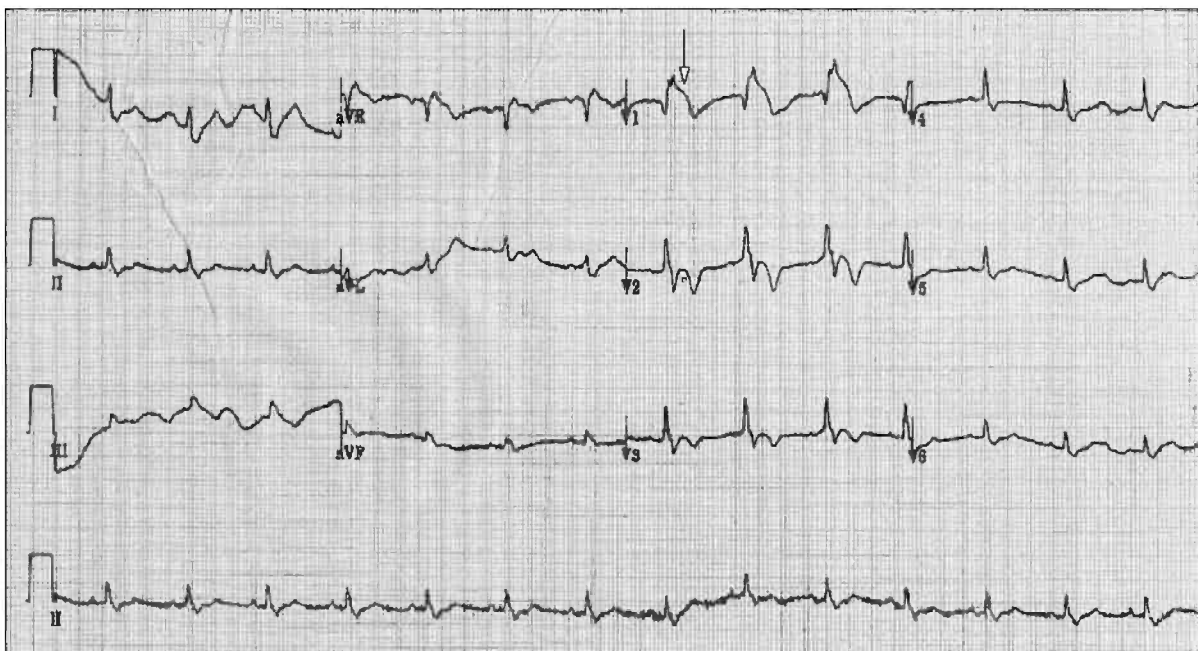


Fig. 5 Case 3. ECG shows sinus rhythm with widening of the QRS complex. The morphology of the QRS complex shows right bundle branch block with coved ST segment elevation, best seen in lead V1. The P wave is still present but not prominent.

CASE 3 CLINICAL PRESENTATION

An 84-year-old man with chronic obstructive pulmonary disease (COPD), who was recently diagnosed with metastatic lung cancer, was admitted with shortness of breath. He was treated for COPD exacerbation. Initially, he responded to the treatment but soon began to deteriorate. He was found to be in respiratory distress with mild haemoptysis. Fig. 4 shows the ECG done on admission and Fig. 5, subsequently. What are the ECG findings?

ECG INTERPRETATION

Fig. 4 shows sinus tachycardia with a normal QRS duration of

94 ms. The sinus tachycardia could be explained by the frequent nebulizer treatment that he had been getting. Subsequently, the ECG in Fig. 5 shows significant widening of the QRS complex to 158 ms with right bundle branch block morphology associated with coved ST segment elevation in lead V1, resembling 'Brugada ECG pattern', which can be seen in severe hyperkalaemia.

CLINICAL COURSE

The patient's potassium level was normal on admission. He then developed acute kidney injury, liver failure, septic shock, severe metabolic acidosis and persistent hypotension. The ECG in Fig. 5 was performed when his condition deteriorated and his potassium level was found to be 6.4 mmol/L. Intravenous calcium gluconate

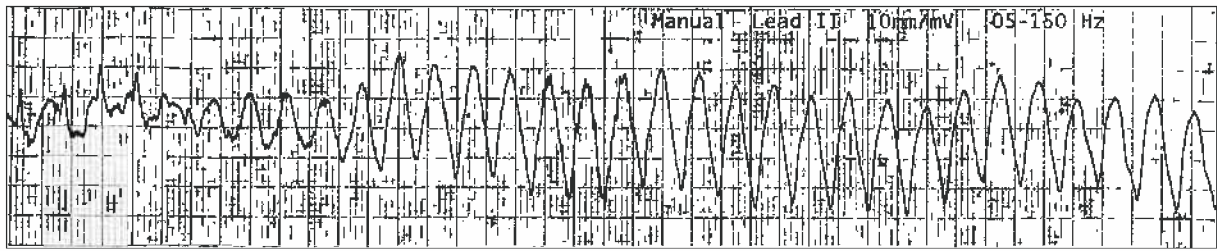


Fig. 6 Case 3. Telemetry strip shows progression to ventricular tachyarrhythmia.

and insulin with dextrose was immediately administered. His potassium level continued to rise to 6.8 mmol/L despite medical intervention. The patient collapsed and was subsequently resuscitated. What is the interpretation of the telemetry strip (Fig. 6)?

ECG INTERPRETATION

Telemetry clearly shows ventricular fibrillation in the beginning of the ECG strip and ventricular tachycardia in the rest of the tracing (Fig. 6).

CLINICAL COURSE

Defibrillation was performed immediately when the patient collapsed. Although he was responding to the treatment, he continued to have recurrent ventricular arrhythmia events despite medical intervention to correct the hyperkalaemia. Intravenous amiodarone was started. Due to his overall condition and terminal cancer diagnosis, the family opted for a 'Do-Not-Resuscitate' status. The patient succumbed following another episode of ventricular fibrillation.

DISCUSSION

In the first case, the ECG findings were associated with severe hypokalaemia. If present, ECG changes can be a very useful, quick, inexpensive and widely available tool to help in the identification of electrolyte abnormalities. As the serum potassium falls, the U wave becomes more prominent and the T wave becomes flatter. The characteristic reversal in the relative amplitude of the T and U waves is the most distinctive change in waveform morphology in hypokalaemia.⁽²⁾ In severe hypokalaemia, T wave inversion and ST segment depression can also be seen.⁽³⁾ The U wave in hypokalaemia is caused by prolongation of the recovery phase of the cardiac potential.⁽²⁾ A delay in ventricular repolarisation will prolong the duration of the relative refractory period, and thus, predisposes to reentrant tachyarrhythmias. Hypokalaemia will also potentiate tachyarrhythmia produced by digitalis toxicity.

Case 2 highlights the classic and initial ECG changes seen with hyperkalaemia, which consists of tall tented T waves. The ratio of intracellular to extracellular potassium is important for determining the cellular membrane potential. Small changes in the extracellular potassium level can have profound effects on the function of the electrical conduction system of the heart. In the setting of hyperkalaemia, the excess extracellular potassium will increase the activity of the potassium channel, resulting in faster repolarisation and thus creating the narrow base and tall T

wave. On the other hand, this will inactivate the sodium channel, preventing proper conduction of electrical activity and resulting in the flattened P wave and widened QRS complex.

Tall T waves can be seen in several other conditions; however, classic T wave in hyperkalaemia is not only tall but also symmetrical, peaked, narrow, 'tented' and scooped inwards⁽⁶⁾ (Fig. 3). The most serious manifestations of hyperkalaemia include cardiac conduction abnormalities and cardiac arrhythmia.⁽⁵⁾ As the hyperkalaemia worsens, the PR interval and QRS duration will get progressively lengthened, followed by disappearance of the P wave and finally, further widening of the QRS duration. These may be followed by fatal arrhythmia such as ventricular fibrillation, ventricular tachycardia and asystole (as highlighted in Case 3). Hyperkalaemia can induce a Brugada-like pattern in the ECG.⁽⁴⁾ This usually occurs in critically ill patient with significant hyperkalaemia (serum potassium > 7.0 mmol/L) and is associated with pseudo right bundle branch block and persistent coved ST segment elevation in at least two precordial leads. Prompt recognition of this ECG entity will enable clinicians to identify severe hyperkalaemia, which may result in high mortality.⁽⁴⁾

ABSTRACT Electrocardiography (ECG) is a useful, quick and widely available tool to confirm the suspicion of, as well as diagnose hypokalaemia and hyperkalaemia. Different ECG changes can be seen with different levels and acuity of changes.

Keywords: ECG, hyperkalaemia, hypokalaemia
Singapore Med J 2012; 53(3): 152-156

ACKNOWLEDGEMENT

We acknowledge Professor Chia Boon Lock for his advice and help in the writing of this article.

REFERENCES

- Poh KK, Teo SG, Tay EL, Yip JW. Electrocardiography Series. ECGs of structural heart disease: Part 1. Singapore Med J 2011; 52:855-9.
- Wagner GS. Marriott's Practical Electrocardiography. 10th ed. Philadelphia: Lippincott Williams & Wilkins, 2001: 222-5.
- Mount DB. Clinical manifestations and treatment of hypokalaemia. In: Uptodate 2011 ver 19.3 [online]. Available at: www.uptodate.com. Accessed December 28, 2011.
- Littmann L, Monroe MH, Taylor L 3rd, Brearley WD Jr. The hyperkalemic Brugada sign. J Electrocardiol 2007; 40:53-9.
- Mount DB. Clinical manifestations and treatment of hypokalaemia. In: Uptodate 2011 ver 19.3 [online]. Available at: www.uptodate.com. Accessed December 28, 2011.
- Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalaemia. Am J Emerg Med 2000; 18:721-9.
- Dubin D. Rapid Interpretation of EKG's. 6th ed. Tampa: COVER Publishing Company, 2000:309-15.

SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME

(Code SMJ 201203A)

Question 1. The following ECG changes are associated with hypokalaemia:

- (a) Severe ST segment depression.
- (b) Increased amplitude and width of the P wave.
- (c) Prolongation of the PR interval.
- (d) Increased height ratio of the U wave compared to the T wave.

Question 2. These statements are true for hypokalaemia:

- (a) The ECG can resemble changes seen in ischaemia.
- (b) Hypokalaemia can cause fatal arrhythmia.
- (c) U wave seen on ECG is caused by prolonged ventricular depolarisation.
- (d) Patients with hypokalaemia will always have ECG changes consistent with hypokalaemia.

Question 3. These conditions can cause the tall T waves seen in ECG:

- (a) Hyperkalaemia.
- (b) Ischaemia.
- (c) Early repolarisation.
- (d) Normal variant.

Question 4. The following ECG changes are associated with hyperkalaemia:

- (a) Pseudo right bundle branch block with ST elevation.
- (b) P wave becomes more prominent in the anterior lead.
- (c) Ventricular tachycardia.
- (d) Sinus tachycardia.

Question 5. ECG changes in hyperkalaemia are associated with:

- (a) Slower repolarisation that will create tall T waves with a narrow base.
- (b) Inactivation of sodium channels.
- (c) Hyperactivity of potassium channels.
- (d) Faster conduction of electrical activity.

True False

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<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: <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 May 2012 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 20 April 2012. (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. (6) One CME point is awarded for successful candidates.

Deadline for submission: (March 2012 SMJ 3B CME programme): 12 noon, 13 April 2012.