A Hyperkalaemia: a dangerous electrolyte disturbance

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INTRODUCTION
The most important electrolytes are sodium, potassium, calcium and magnesium. The flow of these electrolytes into and out of cardiac cells, as currents, creates the energy needed for depolarisation and repolarisation and allows contractile mechanisms to function. The levels of electrolytes in the fluid bathing the cardiac cells has an effect on these currents and the appearance of the complexes seen on the electrocardiograph (ECG).

Of all the electrolyte changes that can occur, hyperkalaemia is the most dangerous. Hyperkalaemia can not only kill, but it can kill in minutes, or even seconds, and it prevents response to the drugs used in resuscitation (Garcia & Holtz, 2001). Hyperkalaemia causes changes in the appearance of the ECG, symbolising the changes in cell function, and can cause any and all arrhythmias. Immediate recognition and action to stabilise the myocardial membrane and reverse the pathological processes are the keys to combating effectively the complications of hyperkalaemia (Garcia & Holtz, 2001). The nurse within the critical care area is in the optimum position to diagnose and direct such management.

PATHOPHYSIOLOGY
Hyperkalaemia refers to an increased serum potassium concentration and is said to exist when the potassium (K⁺) level is above 5 mmol/l. The normal range for potassium is 3.8–5 mmol/l (Tortora & Anagnostakos, 2000). For the cardiac patient, a serum level of at least 4.0 mmol/l should be aimed for (Connaughton, 2001). There is a clear relationship between hypokalaemia and the frequency of ventricular fibrillation (VF) in acute myocardial infarction (Nordrenhaug & von der Lippe, 1983).

Hyperkalaemia is caused by increased potassium intake; a shift in potassium ions from the cells to the extracellular fluid; decreased potassium excretion; or any combination of these factors (Fiever, 1991). Table 1 lists some examples of specific aetiological factors in each of these categories.

Hyperkalaemia occurs across a spectrum of presentation, both in actual blood levels and in the ECG representation of the pathology. The characteristic ECG changes of hyperkalaemia are:

- T wave abnormalities, especially tall and peaked (tented) with a narrow base and symmetrical shape – in any combination
- Intraventricular conduction delays, leading to widening of the QRS complex [Author: Correct as edited?]
- ST segment changes simulating an injury pattern (typically depression, although elevation may occur)
- P waves decreased amplitude and prolongation (maybe missing at higher K⁺ levels)

ECG IDENTIFICATION
Although ECG changes reportedly take place along a spectrum, these changes are more apparent with severe hyperkalaemia (>8 mmol/l). However, these changes are not reliably correlated with the pathology involved [Author: Correct as edited?] (Szerlip et al., 1986). The characteristic ECG changes of hyperkalaemia are:
PR interval prolongation
Cardiac arrhythmias, any and all varieties! A sine-wave pattern appears in severe, often terminal, hyperkalaemia. The patient is typically haemodynamically stable and the sequence of ECG changes can be extremely rapid with catastrophic consequences (Weizenberg et al., 1985; Rastergar & Soleimani, 2001; Wagner, 1994; Metcalfe & Seidelin, 1994; Jenkins & Gerrard, 1999).

MANAGEMENT
When hyperkalaemia of more than 6 mmol/l is present, life-threatening cardiac arrhythmias may occur. An intravenous infusion of calcium chloride or calcium gluconate is used as a short-term measure to restore the membrane potential to a more stable state. Calcium promotes the entry of potassium ions from the intravascular to the intracellular compartment. In hyperkalaemia, this helps to reduce the serum potassium to safer levels (Prosser et al., 2000).

Insulin facilitates the transport of potassium as well as glucose into the cells, and so intravenous insulin, within an infusion of glucose to prevent hypoglycaemia, may be given; the serum potassium and glucose levels must be closely monitored (Williams et al., 1988).

As a temporary measure, the production of alkalosis by the administration of sodium bicarbonate also produces a shift of potassium into the cells; however, this is only a short-term measure as sodium overload will exacerbate water retention. In addition, rapid alteration of the blood pH can cause disequilibration with the pH of the central nervous system tissue fluid.

There is evidence that pacemakers will not initiate 'capture' on the hyperkalaemic patient (Garcia & Holtz, 2001; Humphreys, 2001).

### Table 1. Examples of the different causes of hyperkalaemia

<table>
<thead>
<tr>
<th>Types of aetiology</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Increased potassium intake</td>
<td>Excessive intravenous administration of K+</td>
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<tr>
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<td>Massive transfusion (&gt;10 units) of blood stored longer than 3 days (K+ ions leave the red blood cells)</td>
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<td>Large oral intake and reduced renal excretion</td>
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<td>Movement of potassium out of cells</td>
<td>Acidosis (e.g. diabetic ketoadidosis, lactic acidosis)</td>
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<td>Insulin deficiency</td>
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<td>Massive cell death (crushing injuries, burns, cytotoxic drugs)</td>
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<td></td>
<td>Large overdose of digitals</td>
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<td>Decreased potassium excretion</td>
<td>Oliguria</td>
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<td>Extra cellular volume depletion</td>
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<td></td>
<td>Renal failure</td>
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<td>Decreased aldosterone</td>
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<td>Addison disease</td>
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<td>Chronic heparin administration</td>
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<td>Lead poisoning</td>
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<td>Angiotensin-converting enzyme inhibitors (ACE)</td>
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<td></td>
<td>Other</td>
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<td></td>
<td>Potassium-sparring diuretics (spironolactone, amiloride)</td>
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</tbody>
</table>

### BOX 1

Undertake a systematic analysis of the following 12-lead ECG.

**RHYTHM STRIP:** 25mm/sec: 1cm/mV

**COMMENTARY**

The 12-lead ECG shows the following features:

- Heart rate: 85 bpm
- Rhythm: regular pattern, sinus rhythm
- P waves: normal
- PR interval: normal, 0.16 seconds
- QRS complex: rS in V1; QR in V6 – normal; rapid and early R wave progression, early transition zone V2-V3; voltage less in V5 than in V5 – normal variant; R wave voltage measured in V5 = 30 mm (just exceeding normal parameter of 27 mm)
- QRS duration: 0.08 seconds
- QT interval: 0.36 seconds
- T wave: tall, peaked and narrow in V2-V4, III and aVF greater than two-thirds of the height of the R wave (abnormal).

The diagnosis is sinus rhythm with T wave changes that are suggestive of hyperkalaemia. In this particular patient, the serum potassium was 6.4 mmol/l. T wave changes do not need to be apparent in all leads – as evidenced here. The selective lead changes apparent on this ECG may be accounted for by their closer proximity to the heart.
CONCLUSION

Hyperkalaemia is readily recognised from a 12-lead ECG recording, with the earliest features being T wave changes. T wave changes may be altered as a result of the intraventricular conduction delay that develops with severe hyperkalaemia and can be confused with haemodynamically stable ventricular tachycardia. With hyperkalaemia, there are only seconds to minutes to intervene before possible life-threatening arrhythmias may develop (Garcia and Holtz, 2001). Thus, early recognition and intervention are vital for patient welfare. This article has reviewed the pathophysiological characteristics and the essential management in an attempt to promote insightful, individualistic care within the critical care area.

REFERENCES


