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 combatting 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 has important and potentially serious effects on cardiac function. When the serum potassium concentration increases, there is an increase in the ratio of extracellular to intracellular potassium concentrations. This influences depolarisation by lowering the resting membrane potential, so that cardiac cells become partially depolarised. Initially, this increases their excitability because the resting potential is close to the threshold potential. However, as the extracellular potassium concentration increases, the cardiac cells depolarise to the extent that they cannot repolarise. Cells in this state are non-excitable, meaning that no further contractile activity occurs.

As the serum potassium level increases further **[Author: Correct as edited?]**, the rate of rise and amplitude of the action potential decreases and slows the cell-to-cell conduction velocity. Hyperkalaemia reduces conduction velocity at all levels of the conduction system, including atrial, atrioventricular nodal, and intraventricular levels. In severe hyperkalaemia, intraventricular conduction may be completely inhibited. Bundle-branch block or, less frequently, complete heart block may occur (Smith and Kampine, 1990; Woods *et al.*, 1995).

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 (Flever, 1991). [AUTHOR: Reference missing from final list.] 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. [Author: Correct as edited?] There may be a poor correlation between serum potassium and the typical ECG changes (Wagner, 1994). However, T wave changes are constantly reported as the earliest sign of hyperkalaemia and are typically noted in V2 to V4 in the first instance (Garcia & Holtz, 2001). ECG changes can be apparent when the K+ exceeds 5.5 mmol/l.

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:

_ 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)

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_ PR interval prolongation
_ Cardiac arrhythmias, any and all varieties! A sine-wave pattern appears in severe, often terminal, hyperkalaemia.

Table 1. Examples of the different causes of hyperkalaemia	
Types of aetiology	Examples
Increased potassium intake	Excessive intravenous administration of K^+ Massive transfusion (>10 units) of blood stored longer than 3 days (K^+ ions leave the red blood cells) Large oral intake and reduced renal excretion
Movement of potassium out of cells	Acidosis (e.g. diabetic ketoacidosis, lactic acidosis) Insulin deficiency Massive cell death (crushing injuries, burns, cytotoxic drugs) Large overdose of digitalis

Decreased potassium excretion	Oliguria
	  Extra cellular volume depletion
	  Renal failure
	Decreased aldosterone
	  Addison disease
	  Chronic heparin administration
	  Lead poisoning
	  Angiotensin-converting enzyme inhibitors (ACE)
	Other
	  Potassium-sparing diuretics (spironolactone, amiloride)

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).

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:
Σ Heart rate: 85 bpm
Σ Rhythm: regular pattern, sinus rhythm
Σ P waves: normal
Σ PR interval: normal, 0.16 seconds
Σ QRS complex: rS in V1; qR in V6 – normal; rapid and early R wave progression, early transition zone V2–V3; voltage less in V6 than in V5 – normal variant; R wave voltage measured in V5 = 30 mm (just exceeding normal parameter of 27 mm)
Σ QRS duration: 0.08 seconds
Σ QT interval: 0.36 seconds
Σ 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.

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BOX 2
[Consider the following single-lead ECG recording

[SINGLE LEAD ECG HERE]

Lead II
ECG broad complex rhythm – sine wave pattern

COMMENTARY
_ Pulse rate: 100 bpm
_ Puulse rhythm: regular
_ QRS complex: broad and bizarre; 0.28 seconds. This would suggest ventricular tachycardia (VT) or intraventricular conduction delay (IVCD). Some individual complexes can be determined. However, they may merge into one another as time continues.
_ P waves: some notches apparent before QRS complexes (which would favour diagnosis of IVCD rather than VT)
_ T waves: unable to discern.

If this patient has progressed into this rhythm, treat immediately for hyperkalaemia. The next most likely rhythm will be asystole! An arterial blood gas with potassium level will give a rapid preliminary answer. For the unknown patient who presents in this pattern, it is appropriate to treat as VT and hyperkalaemia simultaneously (Resuscitation Council, 2000).
This particular recording was taken from a patient with chronic renal failure. The serum potassium was 9.6 mmol/l and an injection of calcium gluconate led to a return of a P wave, narrowing of the QRS complex, return of an ST segment and a T wave more typical of hyperkalaemia.

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.

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