AKI management: hyperkalaemia

In excitable tissues, ↑ K^+→ depolarization of the membrane resting potential → Na^+ channel inactivation →↓ membrane excitability → neuromuscular depression and cardiac dysrhythmias.

What represents a dangerous ↑ K^+?

- Chronically hyperkalaemic patients may tolerate ↑ K^+ of 6.0–7.0mmol/L (► but treat if >6.5mmol/L).
- ⚠ However, an acute ↑ K^+ in AKI is much less likely to be tolerated, particularly if: (i) elderly; (ii) associated cardiac disease (esp. arrhythmias); (iii) oliguria (cannot excrete ↑ K^+).
- Closely monitor (→ cardiac monitor, repeat serum K^+ 2–4-hourly) all patients with ↑ K^+ acutely >6.0mmol/L, and commence treatment to enhance K^+ wasting.
- ►► Treat to urgently lower serum K^+ if ≥6.5mmol/L.

⚠ Although U&E are often repeated to exclude haemolysis or artefact, this should cause delays → put on a cardiac monitor, and start treatment.

The hyperkalaemic ECG

ECG manifestations of ↑ K^+ are manifold (see Fig. 2.13). ⚠ Mild ECG changes can progress to life-threatening disturbances very quickly. All may be exacerbated by coexisting ↓ Ca^{2+} and acidosis. ⚠ A normal ECG does *not* rule out cardiac instability.

- Peaking of T waves ('tenting').
- Flattening and disappearance of P waves.
- Prolonged PR interval (1° heart block).
- Progressive widening of the QRS complex.
  - Deepened S waves and merging of S and T waves.
- Idioventricular rhythm.
- Sine wave pattern.
- VF and asystolic cardiac arrest.

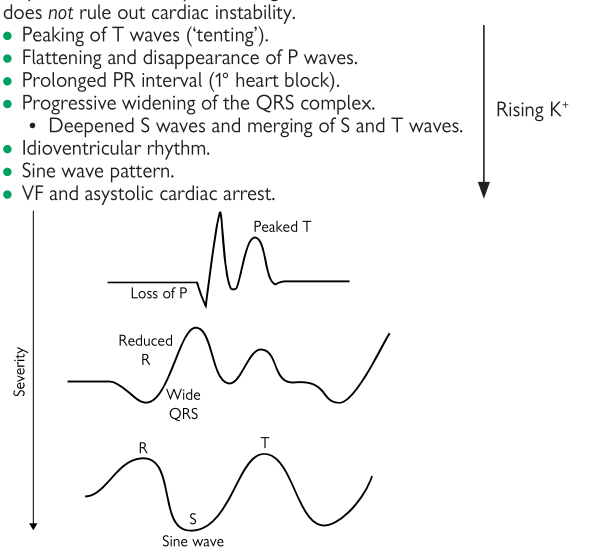

Fig. 2.13 The ECG changes of hyperkalaemia.

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►► Treatment of dangerous hyperkalaemia

The following ↓ *serum* K^+ acutely but DO NOT ↓ overall elevated *total body* K^+. Additional measures, described on 📖 pp. 132–133, are ∴ also required.

Calcium

- If K^+ ≥6.5mmol/L or ECG changes.
- ► Ca^{2+} is cardioprotective—it does not ↓ K^+.
- Antagonizes membrane K^+ effects by poorly understood mechanisms.
  - 10mL 10% calcium gluconate (usually 1 ampoule—calcium gluconate contains 220μmol Ca^{2+}/mL), or
  - 5mL 10% calcium chloride (usually half an ampoule—$CaCl_2$ contains 680μmol Ca^{2+}/mL).
- Give over 2–5min. Repeat if no ECG improvement after 5min (up to 40mL calcium gluconate).
- Acts within minutes, but *protective effect lasts <1h.*
- ⚠ Can induce digitalis toxicity (→ a pragmatic approach: halve the initial dose, and give more slowly if taking digoxin).

Insulin and glucose

- If K^+ ≥6.5mmol/L or ECG changes.
- Insulin binds to its cellular receptor and ↑ Na-K-ATPase activity, moving K^+ into cells. Glucose alone will ↓ K^+ through endogenous insulin release, but insulin/glucose is more effective.
- 10–15IU of soluble insulin (e.g. Actrapid®) in 50mL of 50% glucose IVI over 10min (alternative: 5IU of soluble insulin in 50mL 20% glucose over 15min by syringe pump and repeated).
- 50% glucose is extremely viscous and irritant. Find a large vein, and flush with saline afterwards.
- Effect within 15–30min (peak ~60min), lasts for 2–4h. Expect a ↓ of 0.5–1.5mmol/L. Can be repeated after 4h.
- Check BMs regularly for 6h, and infuse 10% glucose IVI if ↓ glucose.

Sodium bicarbonate

- If ↑ K^+ in the presence of acidosis (HCO_3^- <16) and volume depletion.
- ↑ Na^+/H^+ exchange →↑ intracellular Na^+→↑ Na-K-ATPase activity (i.e. K^+ in for Na^+ out). Additional pH-independent mechanisms operate.
- 1.26% or 1.4% solutions as 200–500mL over 15–60min IVI.
- In cardiac arrest: 50mL of 8.4% (1 ampoule) IVI.
- ⚠ CAUTION: do not infuse bicarbonate solutions into the same cannula as calcium gluconate/carbonate unless thoroughly flushed.
- Action within hours, not minutes.
- Involves an appreciable Na^+ load (150mmol Na^+). ⚠ Volume overload.
- Rapid correction of acidosis in a patient with ↓ Ca^{2+} may induce tetany and seizures, as ionized calcium drops rapidly as pH ↑.

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β2-agonists (salbutamol, etc.)

- 10–20mg (i.e. a large dose) of nebulized salbutamol will ↓ K^+ by up to 1mmol/L but has limited additive benefit beyond insulin/glucose (it acts via the same Na-K-ATPase and has a slower onset of action).
- ⚠ It may also precipitate angina or arrhythmias in those with underlying cardiac disease and can cause an increase in lactate acid (→ worsening acidosis).

Once (if) the immediate arrhythmic danger is past, the aim should be to reduce total body potassium to prevent further hyperkalaemic episodes.

Urinary K^+ wasting: diuretics

- Only useful in patients expected to pass urine and ∴ urine into which K^+ can be excreted. Particularly useful if coexisting volume overload.
- Act on the renal tubule—K^+ loss as one of several effects.
- Furosemide 40–120mg IVI as a slow bolus or 10–40mg/h to a maximum of 1000mg/day. Bumetanide offers a better absorbed oral alternative.
- Effect depends on onset of diuresis. Can lose substantial amounts of K^+ over 24h, with a UO >2L/day.
- *Much* less effective, as GFR deteriorates.

Gut K^+ wasting: cation exchange resins

Overused, particularly orally.

- Exchange Na^+/Ca^{2+} for K^+ in the gut so actually removes K^+, rather than just redistributing it.
- Calcium polystyrene sulfonate (CPS) (Calcium Resonium®) or sodium polystyrene sulphonate (SPS) (Resonium A® or Kayexalate®). Can give 15g orally (supplied as a powder to be suspended in water) up to qds or 15–30g suspended in 2% methylcellulose and 100mL water rectally up to qds, retained for at least 2 (preferably >4) hours. May require saline irrigation through a catheter to remove the resin from the colon.
- Rectal route is more effective, as there is more K^+ available for exchange: colonic $[K^+]$ = 60–90mmol/L, whereas upper GI tract = 5–10mmol/L.
- The constipating effect of these agents given orally may paradoxically prevent K^+ losses in the stool—equally, the laxatives (e.g. lactulose 10–20mL tds) given with these agents may be more efficacious than the agent itself!
- Modest effect seen within 24–48h.
- May cause colonic ulceration and necrosis (recognized with SPS when given with sorbitol as a hyperosmotic laxative—previously a common practice in the USA. Post-op patients with an ileus are at highest risk).

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Extracorporeal K^+ wasting: dialysis

- Consider if K^+ >6.0mmol/L, or rapidly rising, and renal function cannot be restored quickly.
- Lowers K^+ within minutes.
- Haemodialysis (HD) can process 20–60L of blood against a dialysate K^+ of 1–2mmol/L and is ∴ a potent means of removing K^+.
- Haemofiltration (📖 p. 174), with returned infusate free of K^+, can achieve much the same thing although much slower.
- Peritoneal dialysis is effective but rarely indicated acutely (📖 p. 188).
- Requires dialysis access and transfer to a dialysing facility (which potentially introduces delays).
- ⚠ Never transfer a dangerously hyperkalaemic patient—if they are not responding to emergency measures, speak to your ITU (📖 p. 123).

Further management

The aim is to prevent further dangerous rises.

- Restrict oral K^+ intake to <2g per day. ▶ Speak to your dietetic staff (📖 p. 258). ⚠ K^+ content of enteral and parenteral feeds may need modification.
- ⚠ No K^+ in IV fluids.
- Avoid K^+-sparing diuretics, ACE-I, ARB, spironolactone, and NSAIDs.
- ▶ Refractory ↑ K^+ is an indication for dialysis.
- If ↑ K^+ persists, despite dialysis, then:
  - Review dietary intake and compliance.
  - Triple-check the drug chart.
  - Check for GI or occult bleeding (reabsorbed red cells are rich in K^+).
  - Exclude concealed tissue or muscle damage (e.g. compartment syndrome).
  - Review (and consider changing) dialysis access and dialysis adequacy (📖 p. 181). Check dialysate K^+ concentration (📖 p. 179).

Blood transfusion

⚠ Caution is needed when administering a blood transfusion to a patient with AKI, particularly if oligo-anuric. The volume and K^+ content of red cell transfusions can precipitate pulmonary oedema and hyperkalaemia, respectively. If the patient requires renal support, then transfusions are safest given during dialysis treatment (▶ seek expert advice).

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