

EM PIONEERS



Hyperkalemia in the Emergency Department

A study book on the GUARD framework: gauge,
calcium, shift, remove, determine

Edition 2026

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Includes: ECG progression guide · dosing cards · decision tree · quick reference · 8-question quiz



FRONT MATTER

About This Edition

About the author

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About this edition

This book turns the full *Hyperkalemia in the Emergency Department* lecture into a self-study text for emergency physicians, residents, interns and EM nurses, including SCFHS board candidates. It follows the GUARD framework (Gauge severity, Use calcium, Administer shifting agents, Remove potassium, Determine cause and disposition) and ends with a quick-reference sheet and an eight-question quiz. Each chapter closes with Pearls & Pitfalls; use Chapter 9 for revision and Chapter 10 to test yourself.

Learning objectives

- ☐ Recognise the ECG spectrum of hyperkalemia, from peaked T waves to the pre-terminal sine-wave pattern
- ☐ Identify pseudohyperkalemia and avoid over-treating a spurious result
- ☐ Apply GUARD to stabilise, shift and remove potassium in the correct order
- ☐ Select the correct dose and agent for stabilisation, shifting and removal
- ☐ Identify the underlying cause and tailor treatment and disposition
- ☐ Avoid the most common and most dangerous pitfalls

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FRONT MATTER

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CHAPTER 1

Why Hyperkalemia Kills

The case that should not wait

A lab result comes back on a patient who was sent for routine bloods before a minor procedure. Potassium: **6.8**. The patient is sitting comfortably. No palpitations, no weakness, nothing dramatic. It is easy to assume the result is wrong, or that it can wait until the next round.

Twelve minutes later, that patient is in ventricular fibrillation.

The lesson

Hyperkalemia is one of the few numbers in emergency medicine that can kill a patient who looks completely well, in the time it takes to finish a cup of coffee. This book gives you a framework so that the number never gets the chance.

A silent electrical threat

Potassium is the single most important electrolyte for cardiac electrical stability, and hyperkalemia is dangerous for exactly that reason. It disrupts the resting membrane potential of the myocardium, and it can move a heart from a normal rhythm to **ventricular fibrillation or asystole** with very little warning.

What makes hyperkalemia particularly treacherous is the disconnect between symptoms and danger. Patients can have profound hyperkalemia with nothing more than vague weakness, or no symptoms at all, and then arrest without a clear preceding deterioration.

Common causes you will see

Category	Examples
Renal	Acute kidney injury or chronic kidney disease
Medications	ACE inhibitors, angiotensin receptor blockers, potassium-sparing diuretics, NSAIDs
Tissue breakdown	Rhabdomyolysis, tumour lysis syndrome
Metabolic	Diabetic ketoacidosis

Causes are revisited in detail in Chapter 7.

Why the order of treatment matters

Hyperkalemia management is not just about lowering a number. It is about **protecting an electrically unstable heart**, buying time, and then clearing the potassium and treating the cause, in that specific order.

Remember

The heart does not wait for the lab to confirm the result.

KEY POINTS

- Hyperkalemia can arrest a patient who looks well.
- Symptoms are a poor guide to danger.
- Protect the heart first, then clear potassium, then treat the cause.

PEARLS & PITFALLS

PEARL A calm patient with a high potassium is not a safe patient until the ECG and the trajectory say so.

PITFALL Assuming a surprising result is a lab error without looking at the ECG.



CHAPTER 2

The GUARD Framework

One structure for every hyperkalemic patient

GUARD carries you from the moment the result lands in front of you to the moment the potassium is genuinely cleared. Each letter is a step, and the order is deliberate.



One framework. Protect the heart first.

Figure 2.1 — The GUARD framework: five steps, always in this order.

Step	Action	Purpose
G	Gauge severity	Decide how dangerous this is. Treat the ECG, not just the number.
U	Use calcium	Protect the heart first. Stabilise the myocardial cell membrane.
A	Administer shifting agents	Move potassium out of the blood into cells, temporarily.
R	Remove potassium	Take it out of the body permanently.
D	Determine cause & disposition	Only then turn fully to the underlying cause and where the patient goes.

Why the order is not random

You **gauge** how dangerous this is, you **protect the heart** first, then you **shift** potassium out of the blood temporarily, then you **remove** it from the body permanently, and only then do you turn fully to the **underlying cause**. Get this order right, and you will rarely go wrong in this condition.

PEARLS & PITFALLS

PEARL Say the letters out loud: Gauge, Use calcium, Administer shifting agents, Remove, Determine.

PITFALL Skipping to the cause before the myocardium is stabilised.



The ECG matters more than the number

The single most important lesson in this book: **treat the ECG, not just the lab value.**

Treat the ECG, not just the number

A well-looking patient with a high potassium and ECG changes needs treatment now. A high result with a normal ECG and a doubtful sample needs a careful repeat.

The ECG spectrum

ECG changes typically progress in a recognisable sequence, though not every patient follows it neatly, and some deteriorate straight to a lethal rhythm without warning.

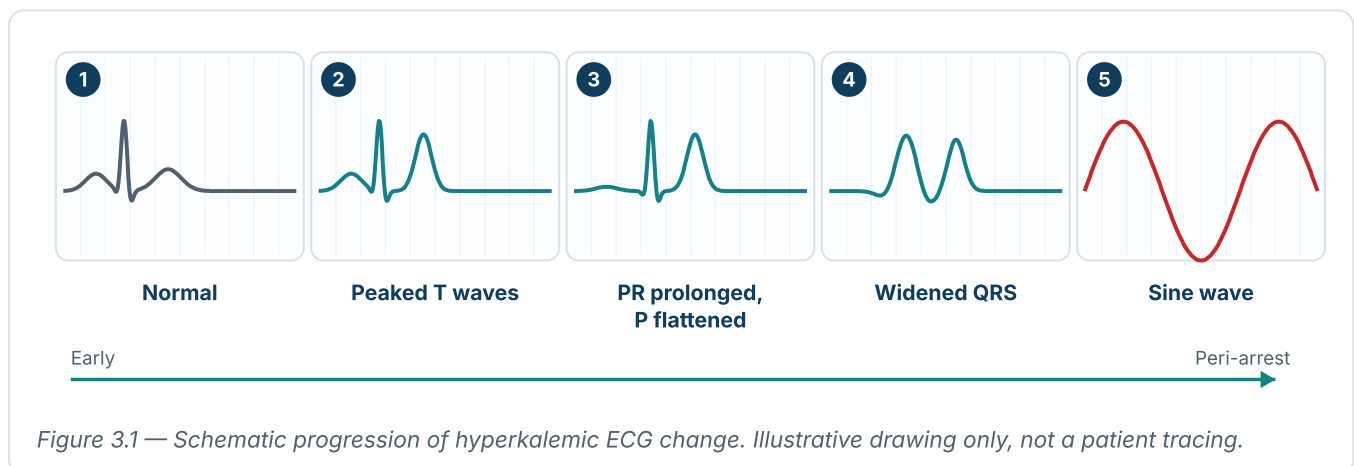


Figure 3.1 — Schematic progression of hyperkalemic ECG change. Illustrative drawing only, not a patient tracing.

Stage	ECG finding	What it tells you
1	Tall, peaked, tented T waves	Usually the first sign, often visible before potassium is markedly elevated.
2	PR prolongation; flattening or loss of the P wave	Atrial conduction is being suppressed.
3	Widened QRS complex	Direct ventricular conduction delay. Predicts imminent risk of a malignant rhythm.
4	QRS merges with T wave: sine-wave pattern	Pre-terminal, peri-arrest ECG.

TIME-CRITICAL **Sine-wave pattern:** do not wait for a formal potassium result. Treat immediately, in parallel with everything else.

Teaching point: recognising this sequence at a glance is the single highest-yield skill in hyperkalemia.

Number, tracing and trajectory

The correlation between the absolute potassium level and the ECG is not perfectly linear. Some patients with a potassium of 6.5 show dramatic ECG changes; others with a similar level show almost none, particularly if the rise has been slow and chronic, such as in stable dialysis patients. Chronic kidney disease patients tolerate higher potassium levels better than patients with an acute rise.

Always correlate the number with the tracing, and both with the **trajectory**. Acute rises are far more dangerous than chronic elevations at the same absolute number.

Pseudohyperkalemia

Before committing to aggressive treatment, consider a falsely elevated result. Causes include:

- ☐ Haemolysed sample
- ☐ Prolonged tourniquet time
- ☐ Fist clenching during venepuncture
- ☐ Marked thrombocytosis
- ☐ Marked leukocytosis

If the result does not match the clinical picture and the ECG is normal, send a repeat sample, ideally a free-flowing, non-haemolysed draw, before committing a well patient to unnecessary treatment.

But if the ECG shows changes

Treat first and query the sample second.

KEY POINTS

- ECG progression: peaked T, then PR prolongation and flat P, then wide QRS, then sine wave.
- Sine wave means treat now, without waiting for labs.
- Chronic rises are better tolerated than acute rises at the same number.
- Normal ECG plus mismatched picture means repeat a clean sample.

PEARLS & PITFALLS

PEARL Recognise the sequence at a glance: it is the highest-yield skill here.

PITFALL Treating a haemolysed result in a well patient with a normal ECG.

PITFALL Dismissing a high result because the patient looks fine and the sample 'might be haemolysed' while the ECG is abnormal.



CHAPTER 4

U — Use Calcium

Protecting the myocardium first

Calcium does **nothing to lower the potassium level**. Its entire purpose is to stabilise the cardiac cell membrane and buy you time. When the ECG shows changes, it should be your very next move.

Dosing card **TIME-CRITICAL**

PERIPHERAL LINE

10 mL of calcium gluconate 10% over 2–3 minutes

MAXIMUM

Up to 30 mL over 10 minutes

CENTRAL LINE

10 mL of calcium chloride 10% over 10 minutes

ONSET · DURATION

Within minutes · about 30–60 minutes

Repeat if ECG changes persist or recur.

Which calcium?

	Calcium gluconate 10%	Calcium chloride
Dose and route	10 mL of 10% over 2–3 minutes through a peripheral line	10 mL of 10% over 10 minutes through a central line
Role	Standard first-line choice in most emergency departments	May be preferred in a peri-arrest patient with central access already in place
Potency	Less potent per mL	More potent per mL
Extravasation	Safer in peripheral lines; lower risk of tissue injury	Significantly more irritating to peripheral veins and tissue

Know which preparation your department stocks and follow your local protocol.

A bridge you may need to rebuild

Onset is fast, but the effect is temporary. If ECG changes persist or recur after the 30–60 minute window, **repeat the dose**. This is not a one-and-done intervention in a patient with ongoing severe hyperkalemia; it is a bridge you may need to rebuild more than once while shifting and removal agents take effect.

DON'T MISS Digoxin toxicity

In a patient on digoxin with suspected digoxin toxicity, calcium has historically been associated with precipitating severe, treatment-resistant arrhythmia, sometimes called '**stone heart**'. Give calcium more cautiously, **as a slow infusion rather than a rapid push**, and treat the digoxin toxicity in parallel.

KEY POINTS

- Calcium protects the heart; it does not lower potassium.
- Peripheral line: calcium gluconate 10%, 10 mL over 2–3 minutes. Central line: calcium chloride 10%, 10 mL over 10 minutes. Up to 30 mL over 10 minutes.
- Repeat if ECG changes persist or recur.
- Suspected digoxin toxicity: slow infusion, treat in parallel.

PEARLS & PITFALLS

PEARL Think of calcium as buying time, not fixing the problem.

PITFALL Treating the potassium number and forgetting to repeat calcium when the ECG changes return.

PITFALL Rapid calcium push in suspected digoxin toxicity.



CHAPTER 5

A — Administer Shifting Agents

Moving potassium out of the blood, temporarily

Shifting agents drive potassium from the bloodstream **into cells**. They do not remove it from the body, which is why removal (Chapter 6) is never optional.

Insulin and glucose: the workhorse

Dosing card

INSULIN

10 units regular insulin IV

DEXTROSE

25 g (typically 50 mL of 50% dextrose, or equivalent volume of a more dilute solution per local protocol)

Insulin drives potassium into cells via the sodium-potassium ATPase pump. The glucose is given to prevent hypoglycaemia, not to treat the potassium.

Check the patient's glucose before you give this. If the patient is already significantly hyperglycaemic, you may withhold the co-administered dextrose, but you must still monitor glucose closely afterwards.

DON'T MISS Delayed hypoglycaemia

Hypoglycaemia can be delayed by several hours after insulin. It is one of the most common preventable complications of hyperkalemia treatment. Check capillary glucose at **30 minutes, 1 hour, then hourly for at least 4–6 hours**.

Nebulised beta-2 agonists

Salbutamol (albuterol) is a useful adjunct. The dose for potassium shifting is substantially higher than a standard asthma dose, typically **10–20 mg nebulised**. It works by the same intracellular shift mechanism through a different receptor pathway. It is **additive to** insulin and glucose, **not a substitute**, and is particularly useful when IV access or insulin administration is delayed.

Sodium bicarbonate

More controversial. Current evidence does not support its routine, isolated use purely to shift potassium in a patient with normal acid-base status; the effect is inconsistent and slow in that context. It clearly has a role in the hyperkalemic patient with a **concurrent significant metabolic acidosis**, where correcting the acidosis contributes to potassium shifting as a secondary benefit. Do not rely on bicarbonate alone as your shifting strategy.

Shifting agents at a glance

Agent	Dose / use	Key point
Insulin + dextrose	10 units regular insulin IV with 25 g dextrose	Workhorse. Check glucose before; monitor for hours after.
Nebulised salbutamol	10–20 mg nebulised	Additive, not a substitute. Useful when IV access or insulin is delayed.
Sodium bicarbonate	Per local protocol	Reserve for concurrent significant metabolic acidosis.

KEY POINTS

- Shifting is temporary; potassium is still in the body.
- Check glucose before insulin and for 4–6 hours afterwards.
- Salbutamol is an adjunct, not a substitute.
- Bicarbonate is for concurrent metabolic acidosis, not routine use.

PEARLS & PITFALLS

- PEARL** Dextrose is there for glucose safety, not for potassium.
- PITFALL** Stopping glucose checks after the first hour.
- PITFALL** Using bicarbonate alone as the shifting strategy in a patient with normal acid-base status.



CHAPTER 6

R — Remove Potassium

The step that is never optional

Calcium protects the heart. Insulin, glucose and beta-agonists buy time by shifting potassium into cells. **None of that potassium has left the body**, and as those temporary effects wear off it will shift back into the bloodstream unless a removal strategy is running in parallel.

Shifting is temporary. Removal is what actually clears the potassium.

Removal options

Option	Onset and role	Cautions
Loop diuretics (e.g. furosemide)	Promote renal potassium excretion. Reasonable when renal function is adequate and the patient is not volume-depleted.	Not suitable in renal failure or volume depletion.
Sodium polystyrene sulfonate (Kayexalate)	Slow onset, several hours.	Rare but serious risk of intestinal necrosis, particularly postoperatively or with bowel dysmotility.
Patiromer and sodium zirconium cyclosilicate	More favourable safety profile; SZC has a somewhat faster onset, still measured in hours, not minutes.	Not appropriate as sole emergent treatment.
Dialysis	Definitive and fastest true removal option.	Needs early nephrology involvement.

Binders: part of the strategy, not the rescue

None of the binders is appropriate as the sole emergent treatment in a patient with dangerous ECG changes. They are part of the removal strategy, running alongside more immediate measures, and are useful for **preventing rebound** over the following hours.

Dialysis

Dialysis is the treatment of choice in severe hyperkalemia with renal failure, or in any patient with life-threatening hyperkalemia refractory to medical therapy.

TIME-CRITICAL Call nephrology early

If your patient has ECG changes, a dangerously high potassium, and limited or no renal function, involve nephrology early. Do not wait until medical therapy has already failed to make that call.

KEY POINTS

- Removal must run in parallel with shifting.
- Binders take hours; they prevent rebound but do not rescue.
- Dialysis is the fastest definitive removal option.
- Loop diuretics only when renal function and volume status allow.

PEARLS & PITFALLS

PEARL Start the removal plan at the same time as the shifting agents.

PITFALL Relying on Kayexalate for a peri-arrest patient.

PITFALL Waiting for medical therapy to fail before calling nephrology.



CHAPTER 7

D — Determine Cause & Disposition

Closing the loop

Treating the potassium without addressing why it happened sets your patient up to be back in your department with the same emergency.

Ask systematically

Domain	What to look for
Renal function	Acute kidney injury or chronic kidney disease
Medications	ACE inhibitors, ARBs, potassium-sparing diuretics (e.g. spironolactone), NSAIDs, trimethoprim
Tissue breakdown	Rhabdomyolysis, tumour lysis syndrome, large haematoma resorption
Endocrine	Adrenal insufficiency
Acid-base	Diabetic ketoacidosis

DON'T MISS DKA

DKA is a classic cause of a falsely reassuring-looking potassium that will drop sharply once insulin therapy begins. Anticipate that fall and monitor closely.

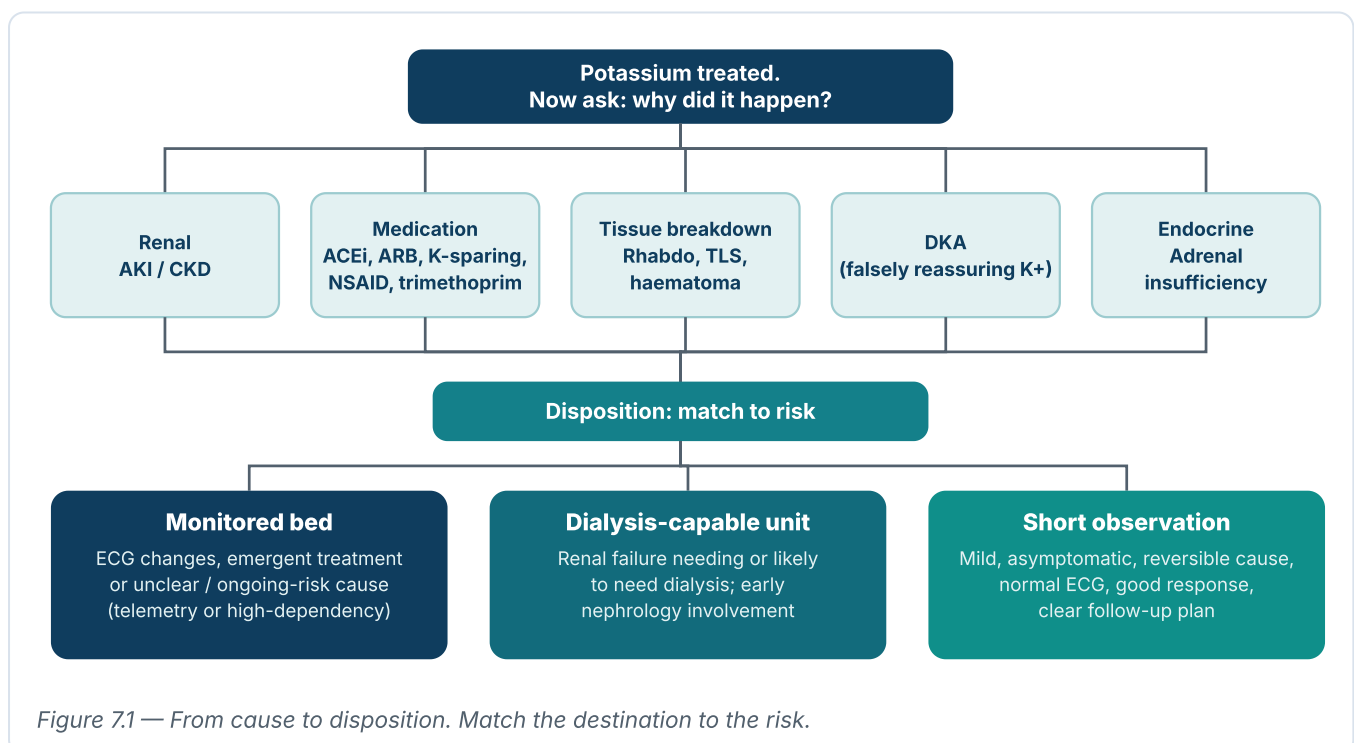


Figure 7.1 — From cause to disposition. Match the destination to the risk.

Disposition

Patient	Disposition
ECG changes, potassium requiring emergent treatment, or an unclear or ongoing-risk cause	Monitored bed at minimum (telemetry or high-dependency) with continuous cardiac monitoring and serial potassium levels
Renal failure who required or may require dialysis	Early nephrology involvement; admission to a unit able to deliver dialysis without delay
Mild, asymptomatic elevation; clearly identified and reversible cause; normal ECG; good response to initial treatment	May suit a shorter period of observation with a clear outpatient follow-up plan, decided deliberately, not by default

The disposition rule

Do not discharge a patient whose potassium trajectory you have not confirmed is truly falling and staying down, on a repeat, reliable sample.

KEY POINTS

- Always ask why the potassium rose.
- DKA potassium will fall after insulin starts.
- Any ECG change or emergent treatment means a monitored bed.
- Discharge only after a reliable repeat shows a falling, sustained trend.

PEARLS & PITFALLS

PEARL Short observation is a deliberate decision, never the default.

PITFALL Discharging on a single improved value without a reliable repeat.



CHAPTER 8

Tips & Tricks

What catches even experienced clinicians

- 1 Do not wait for the lab if the ECG is sine-wave.** In a critically unwell patient, treat immediately with calcium, shifting agents and urgent removal planning while confirmatory bloods are still processing.
- 2 Keep rechecking glucose after insulin and dextrose,** repeatedly, for hours. Delayed hypoglycaemia is one of the most common iatrogenic complications and is entirely preventable with a monitoring plan.
- 3 Anticipate the rebound.** Calcium wears off after 30–60 minutes and shifting agents after a few hours. Recheck potassium within 1–2 hours of treatment, not just at the end of the shift.
- 4 In DKA, total body potassium is often depleted** even when the serum level looks high or normal. Once insulin starts, potassium can fall sharply. Monitor closely and be ready to replace, not just to lower.
- 5 Do not be falsely reassured by a normal ECG** with a very high reported potassium, especially if the sample could be haemolysed or delayed. If the context does not fit, repeat the sample properly rather than over-treating or dismissing it.
- 6 Management is sequential, not simultaneous chaos.** Gauge severity, protect the heart, shift, remove, and only then chase the cause. Clinicians who treat the cause before stabilising the myocardium lose patients they did not need to lose.

Closing: your commitment

Return to the patient from Chapter 1: a potassium of 6.8, no symptoms, twelve minutes from an arrest that a systematic approach could have prevented. The clinicians who catch that risk are not the ones with the fastest hands. They treat the ECG as seriously as the lab value, protect the myocardium before chasing the number down, and never assume the job is finished once the potassium starts to fall.

Gauge severity. Use calcium. Administer shifting agents. Remove potassium. Determine the cause and disposition. GUARD, every single time.



CHAPTER 9

GUARD Quick Reference

One-page revision sheet

Step	Key actions	Watch for
G Gauge severity	Treat the ECG, not just the number. Sequence: peaked T, PR/P change, wide QRS, sine wave.	Pseudohyperkalemia; acute vs chronic; sine wave means treat now.
U Use calcium	Peripheral: calcium gluconate 10%, 10 mL over 2–3 min. Central: calcium chloride 10%, 10 mL over 10 min. Up to 30 mL over 10 min. Lasts about 30–60 min; repeat if ECG changes persist.	Digoxin toxicity: slow infusion. Calcium does not lower potassium.
A Administer shifting agents	10 units regular insulin IV + 25 g dextrose. Salbutamol 10–20 mg nebulised. Bicarbonate only with metabolic acidosis.	Check glucose before; monitor at 30 min, 1 h, then hourly for 4–6 h.
R Remove potassium	Loop diuretic if renal function and volume allow. Binders (SPS, patiomer, SZC): hours. Dialysis: fastest definitive.	Kayexalate bowel necrosis risk. Call nephrology early.
D Determine cause & disposition	Renal, drugs, tissue breakdown, endocrine, DKA. Monitored bed if ECG changes or emergent treatment.	DKA potassium falls with insulin. Do not discharge without a reliable falling repeat.

Bedside checklist

- ☐ ECG reviewed: peaked T, PR/P change, wide QRS, sine wave?
- ☐ Sample reliable? Haemolysis, tourniquet, fist clenching, high platelets or white cells?
- ☐ Calcium given (slow infusion if digoxin toxicity suspected)?
- ☐ Insulin and dextrose given; glucose checked before and scheduled afterwards?
- ☐ Removal plan started; nephrology involved if renal failure or refractory?
- ☐ Potassium rechecked within 1–2 hours?
- ☐ Cause identified; DKA potassium fall anticipated?
- ☐ Disposition decided deliberately; reliable repeat confirms falling potassium?



CHAPTER 10

Self-Assessment Quiz

Eight questions to test yourself. Cover the answers, choose one option, then check the model answer. A score of **6 out of 8** is a pass; if you score lower, revisit Chapters 3, 4 and 5.

1

Which ECG finding represents a pre-terminal, peri-arrest pattern in severe hyperkalemia?

- A. Isolated peaked T waves
- B. PR interval prolongation only
- C. A sine-wave pattern (widened QRS merging with the T wave)
- D. Sinus tachycardia

MODEL ANSWER C — A sine-wave pattern (widened QRS merging with the T wave)

The sine-wave pattern reflects severe conduction delay and is a peri-arrest finding. It should prompt immediate treatment — calcium, shifting agents and urgent removal planning — without waiting for confirmatory labs.

2

What is the primary purpose of administering calcium gluconate in hyperkalemia?

- A. To lower the serum potassium level
- B. To stabilise the cardiac cell membrane and reduce arrhythmia risk
- C. To shift potassium into cells permanently
- D. To treat the underlying cause of hyperkalemia

MODEL ANSWER B — To stabilise the cardiac cell membrane and reduce arrhythmia risk

Calcium does not lower potassium at all. Its role is purely to stabilise the myocardial cell membrane and reduce the risk of a malignant arrhythmia, buying time for shifting and removal therapies to work.

3

A patient receives IV insulin and dextrose for hyperkalemia. What is the most important monitoring step afterward?

- A. Repeat ECG only, no further glucose checks needed
- B. Monitor capillary glucose repeatedly for several hours, as hypoglycaemia can be delayed
- C. No monitoring needed once the dose is given
- D. Check potassium immediately and discharge if normal

MODEL ANSWER B — Monitor capillary glucose repeatedly for several hours, as hypoglycaemia can be delayed

Delayed hypoglycaemia is one of the most common preventable complications of insulin/dextrose therapy. Check glucose at 30 minutes, 1 hour, then hourly for at least 4–6 hours.

4 What is the current evidence-based role of sodium bicarbonate as a potassium-shifting agent?

- A. First-line therapy in all hyperkalemic patients
- B. Reserved for patients with concurrent significant metabolic acidosis, not for routine isolated use
- C. A substitute for insulin and glucose
- D. Contraindicated in all hyperkalemic patients

MODEL ANSWER B — Reserved for patients with concurrent significant metabolic acidosis, not for routine isolated use

Evidence does not support routine bicarbonate use purely to shift potassium in patients with normal acid-base status. Its clearest role is in hyperkalemia with concurrent significant metabolic acidosis.

5 Which of the following is a true statement about potassium-removal strategies?

- A. Insulin and glucose permanently remove potassium from the body
- B. Sodium polystyrene sulfonate (Kayexalate) has a rapid onset suitable for peri-arrest use
- C. Dialysis is the fastest definitive removal option, especially in renal failure
- D. Nebulised salbutamol removes potassium from the body

MODEL ANSWER C — Dialysis is the fastest definitive removal option, especially in renal failure

Dialysis is the fastest true removal option and the treatment of choice in severe hyperkalemia with renal failure or refractory cases. Insulin/glucose and salbutamol only shift potassium temporarily; Kayexalate has a slow onset of several hours.

6 A patient with known digoxin toxicity develops hyperkalemia with ECG changes. What is the key caution regarding calcium administration?

- A. Calcium is absolutely contraindicated and must never be given
- B. Calcium should be given as a rapid IV push at double the normal dose
- C. Calcium should be given more cautiously, as a slow infusion, due to risk of precipitating a severe treatment-resistant arrhythmia
- D. Calcium has no special considerations in digoxin toxicity

MODEL ANSWER C — Calcium should be given more cautiously, as a slow infusion, due to risk of precipitating a severe treatment-resistant arrhythmia

In suspected digoxin toxicity, rapid calcium has historically been associated with precipitating severe, treatment-resistant arrhythmia ('stone heart'). Give it more cautiously as a slow infusion, and treat the digoxin toxicity in parallel.

7

A diabetic ketoacidosis patient has a serum potassium of 5.2 on arrival. What should the clinician anticipate once insulin therapy begins?

- A. Potassium will remain unchanged throughout treatment
- B. Potassium will likely rise further and require urgent removal therapy
- C. Potassium will likely fall, sometimes sharply, because total body potassium is often depleted despite the presenting level
- D. No potassium monitoring is needed once DKA treatment starts

MODEL ANSWER C — Potassium will likely fall, sometimes sharply, because total body potassium is often depleted despite the presenting level

In DKA, acidosis and insulin deficiency shift potassium out of cells, so the presenting level often overestimates total body potassium. Once insulin starts, potassium typically falls and may need replacement rather than further lowering.

8

What is the correct sequence emphasised by the GUARD framework?

- A. Determine the cause first, then treat the potassium level
- B. Remove potassium first, then stabilise the myocardium
- C. Gauge severity, stabilise the myocardium with calcium, shift potassium, remove potassium, then determine cause and disposition
- D. Administer shifting agents before assessing the ECG

MODEL ANSWER C — Gauge severity, stabilise the myocardium with calcium, shift potassium, remove potassium, then determine cause and disposition

The GUARD sequence is deliberate: gauge severity and treat the ECG, protect the heart with calcium, shift temporarily, remove definitively, and only then focus on the underlying cause and disposition. Skipping ahead to cause-treatment before stabilising the myocardium is a common and dangerous error.

E M P I O N E E R S



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