PARAMEDIC EDUCATION

Electrolytes in the Field

Sodium · Potassium · Calcium · Chloride · Magnesium

Function, normal ranges, and what the patient looks like when it goes wrong
You will not have a chemistry panel. You will have a history, an exam, and a monitor.

SESSION PLAN

What we are covering

1 Fundamentals

Ions, charge, mEq/L, and why the body cares so much about a tenth of a point.

2 Sodium

Water balance, mental status, and the seizure you did not expect.

3 Potassium

The one that kills fastest and the one your monitor can show you.

4 Calcium

Contraction, coagulation, conduction, and the QT interval.

5 Magnesium

Torsades, refractory arrest, and the obstetric patient.

6 Chloride & acid–base

Anion gap, bicarbonate, and how pH moves potassium.

7 Field patterns

Dialysis, crush, DKA, diuretics, GI losses, heat.

8 What you can actually do

Fluids, protocol options, monitoring, and the destination decision.

9 Case review

Back to the opening scenario, with the physiology behind it.

CASE STUDY

Read now. We solve it at the end.

Dispatch
Generalized weakness, private residence, Sunday afternoon.

On arrival
A 62-year-old man is seated, awake, and says his legs "feel like they are not mine." He is on hemodialysis Monday, Wednesday, and Friday, and missed Friday because of a ride problem. He has a left forearm fistula with a palpable thrill. HR 48 and irregular, BP 148/86, RR 18, SpO_2 96%, glucose 122. He is not short of breath and denies chest pain.

Your monitor shows tall, narrow, symmetrically peaked T waves, a QRS you measure at 0.14 seconds, and P waves you can barely find.

Hold these questions:

1. What single electrolyte explains every one of these findings?
2. Why is the missed dialysis session the most important sentence in the history?
3. Which arm do you use for the IV and the blood pressure, and why?
4. What can you actually do for him before the emergency department?

FUNDAMENTALS

What an electrolyte actually is

1 An ion is a charged particle

Dissolved in water, electrolytes dissociate into ions and the solution conducts electricity. Cations carry a positive charge — sodium, potassium, calcium, magnesium. Anions carry a negative charge — chloride, bicarbonate, phosphate.

2 Measured in mEq/L

Milliequivalents measure combining power, not weight. That is why sodium bicarbonate is ordered as 50 mEq rather than in milligrams. Calcium and magnesium are often reported in mg/dL instead, which is why their numbers look different.

3 Charges must balance

In any compartment, total positive charge equals total negative charge. Move one ion and something else must move with it or in place of it — the reason chloride and bicarbonate trade places, and the basis of the anion gap.

4 Three jobs, that is all

Electrolytes generate electrical gradients across membranes, hold water in the right compartment, and serve as cofactors for chemical reactions. Every sign and symptom in this lecture traces back to one of those three.

You will never have a chemistry panel in the back of the ambulance. What you will have is a history that identifies risk, a physical exam, and a monitor that displays potassium, calcium, and magnesium in real time.

FUNDAMENTALS

Compartments, the pump, and why cells are excitable

WHERE THE WATER IS

- Total body water is about 60% of body mass in an adult
- Two thirds sits inside cells; one third outside
- Of that extracellular third, about three quarters is interstitial and one quarter is plasma
- A blood draw samples plasma only — the smallest compartment of the three

WHERE THE IONS ARE

- Sodium is the dominant cation OUTSIDE the cell
- Potassium is the dominant cation INSIDE the cell
- Magnesium and phosphate are mostly intracellular
- Serum potassium therefore tells you very little about total body potassium

The sodium–potassium pump is why any of this is possible

Three sodium out, two potassium in, powered by ATP. The pump maintains a resting membrane potential of roughly −90 mV in a cardiac cell, and that gradient is what makes the cell excitable. When perfusion fails, ATP fails, the pump slows, sodium and water move into the cell, the cell swells, and potassium leaks out into the serum. Cellular injury and hyperkalemia are the same event described two different ways — which is exactly what happens in crush syndrome and in prolonged cardiac arrest.

REFERENCE

Normal ranges — the numbers to actually memorize

Electrolyte	Normal range	Primary location	One-line job
Sodium (Na^+)	135–145 mEq/L	Extracellular	Sets serum osmolality — where the water goes
Potassium (K^+)	3.5–5.0 mEq/L	Intracellular	Sets resting membrane potential — cardiac excitability
Calcium (Ca^{2+})	8.5–10.5 mg/dL total 4.5–5.6 mg/dL ionized	Bone, then extracellular	Muscle contraction, coagulation, conduction
Chloride (Cl^-)	98–106 mEq/L	Extracellular	Follows sodium; balances charge with bicarbonate
Magnesium (Mg^{2+})	1.5–2.5 mEq/L	Intracellular	Cofactor for the Na^+/K^+ pump; stabilizes rhythm
Bicarbonate (HCO_3^-)	22–26 mEq/L	Extracellular	Primary buffer — the acid–base workhorse

Look at the width of each window. Sodium tolerates ten points. Potassium tolerates about one and a half — and the distance from normal to lethal is often less than two. That is why potassium gets the most time today.

SODIUM · NA^+

Water Follows Salt

135–145 mEq/L. The dominant extracellular cation, and the one that decides where the water in your patient ends up.

Osmolality · Mental status · Seizures · ADH

What it does and how the body defends it

Sets serum osmolality

Sodium and its accompanying anions account for most of the osmotic pressure of extracellular fluid. Change sodium and you change where water sits — which is why sodium disorders present as brain problems.

Carries the action potential

Depolarization is sodium rushing into the cell. Sodium channel blockade — tricyclics, cocaine, some antiarrhythmics — widens the QRS for exactly this reason.

Drives absorption and transport

Glucose and amino acid uptake in the gut and kidney are coupled to sodium movement. It is the currency the body spends to move other things.

ADH holds water

Antidiuretic hormone from the posterior pituitary makes the kidney retain free water, which dilutes serum sodium. Too much ADH is the mechanism behind SIADH.

Aldosterone holds sodium

From the adrenal cortex: retain sodium, excrete potassium. One sentence that links the two electrolytes and explains why spironolactone causes hyperkalemia.

Thirst is the last defense

And it fails in the people who cannot act on it — infants, the elderly with dementia, the post-stroke patient, anyone who cannot ask for water.

Sodium disorders are almost always WATER disorders. A low sodium usually means too much water, not too little salt.

Hyponatremia — below 135 mEq/L

WHO GETS IT

- Thiazide and loop diuretics
- Heart failure, cirrhosis, renal failure
- SIADH — cancer, pneumonia, head injury, some medications
- Psychogenic polydipsia
- Endurance athletes replacing sweat with plain water
- MDMA — ADH effect plus heavy water intake
- Adrenal insufficiency (low Na with high K)
- GI losses replaced with free water

HOW THEY PRESENT

- Nausea, vomiting, headache, malaise
- Confusion, lethargy, irritability
- Muscle cramps and weakness
- Ataxia and unsteady gait
- SEIZURE — often with no epilepsy history
- Coma in severe or rapid drops
- Signs are neurologic because the brain is swelling

WHAT YOU DO

- Airway, breathing, seizure precautions
- Check a glucose — the presentation overlaps completely
- Treat seizures per protocol
- Be deliberate with fluids; a large hypotonic bolus moves the number the wrong way
- Take a fluid-intake history and hand it off — how much, of what, over how long
- Correction is a hospital problem and is done slowly on purpose

Speed matters more than the number. A sodium of 120 that fell in an hour seizes; a sodium of 120 that took three months walks into triage.

SODIUM

Hypernatremia — above 145 mEq/L

WHO GETS IT

- Anyone who cannot get their own water
- Elderly with dementia; post-stroke patients
- Infants — gastroenteritis, improperly mixed formula
- Tube-fed patients without enough free water
- Osmotic diuresis from uncontrolled hyperglycemia
- Diabetes insipidus
- Heat illness and heavy sweat losses
- Burns

HOW THEY PRESENT

- Thirst — when they are able to express it
- Dry mucous membranes, poor turgor, sunken eyes
- Tachycardia, orthostasis, low urine output
- Irritability and restlessness FIRST
- Then lethargy, weakness, and confusion
- Seizure and coma when severe
- Fever is common and may be the presenting complaint

WHAT YOU DO

- Isotonic fluid for perfusion, per protocol
- Check a glucose — hyperglycemia is a common driver
- Address the heat exposure if that is the cause
- Assess the living situation and say so in your report
- Expect the hospital to rehydrate slowly — too-fast correction swells the brain

This is rarely too much salt. It is almost always not enough water — which makes it a question about access and function, not just chemistry.

POTASSIUM · K^+

The One That Kills Fastest

3.5–5.0 mEq/L. A window about a point and a half wide, guarding the electrical stability of every cardiac cell.

Membrane potential · ECG · Dialysis · Crush

Function, and the three things that move it fast

WHAT IT DOES

- 98% of body potassium is INSIDE cells
- The gradient sets resting membrane potential (~−90 mV)
- Governs repolarization — the T wave is potassium leaving the cell
- Required for nerve conduction and muscle contraction, skeletal and cardiac

HOW IT LEAVES THE BODY

- The kidney does essentially all of it, under aldosterone control
- Small amounts in stool and sweat
- When the kidney fails, that route closes — which is why renal patients dominate this topic
- Dialysis replaces the missing route on a schedule, not continuously

THREE THINGS THAT MOVE POTASSIUM IN MINUTES — WITHOUT CHANGING TOTAL BODY POTASSIUM AT ALL

1 pH

Acidosis pushes K^+ OUT of cells as H^+ moves in — serum potassium rises. Alkalosis does the reverse. This is why DKA, sepsis, and prolonged arrest all raise serum potassium.

2 Insulin

Drives K^+ INTO cells. It is why insulin with glucose is a hospital hyperkalemia treatment, and why treating DKA drops potassium fast enough to be dangerous.

3 Beta$_2$ stimulation

Drives K^+ INTO cells. This is the entire reason nebulized albuterol appears in your crush injury protocol — it is not being given for wheezing.

Shifting is not removing. Albuterol, bicarbonate, and insulin buy time. Only dialysis, a working kidney, or a binder actually takes potassium out of the body.

POTASSIUM

Hyperkalemia — above 5.0 mEq/L

WHO GETS IT

- **Renal failure — the dominant cause**
  - Missed or shortened dialysis session is the classic field history
- **Crush injury and rhabdomyolysis**
  - Damaged muscle dumps intracellular potassium into the blood
- **Acidosis of any cause**
  - DKA, sepsis, prolonged cardiac arrest
- **Medications**
  - ACE inhibitors, ARBs, spironolactone, potassium supplements, NSAIDs, TMP-SMX
  - Salt substitutes — they are potassium chloride
- **Massive tissue injury**
  - Burns, extensive trauma, tumor lysis, GI bleed
- **Adrenal insufficiency (low Na with high K)**

HOW THEY PRESENT

- Muscle weakness, classically ASCENDING — legs first, described as heavy or "not mine"
- Fatigue and malaise out of proportion to anything else
- Paresthesias, numbness, tingling
- Nausea, vomiting, diarrhea, abdominal cramping
- Palpitations or an irregular pulse
- Bradycardia — and it will not respond well to atropine
- And then, without much warning, cardiac arrest

The presentation is bland right up until it is fatal. Ascending weakness plus a renal history means the monitor goes on before you finish the history.

Ask about salt substitutes by name. Patients on a renal or heart-failure diet use them daily and do not consider them medication.

The ECG progression — your field potassium level

Approx. K^+	What appears on the monitor	What it means
5.5–6.5	Tall, narrow, symmetric, pointed ("tented") T waves. Shortened QT.	Repolarization is faster and more complete. Often the only clue, and easy to dismiss.
6.5–7.5	P wave flattens then disappears. PR interval lengthens.	Atrial tissue is losing the ability to depolarize. This is a warning, not a subtlety.
7.0–8.0	QRS widens. Bundle branch patterns that fit no anatomic block.	Ventricular conduction is slowing. A wide complex here is not ventricular tachycardia.
> 8.0	Sine wave — QRS merges into the T. Then VF, idioventricular rhythm, or asystole.	Pre-arrest. Treat immediately; do not wait for a confirmatory number.

The numbers are approximate — the SEQUENCE is reliable

How fast the potassium rose matters as much as the value. A chronic dialysis patient may tolerate a number that would arrest someone else. Teach the trend, not the threshold.

The sentence to memorize

A wide complex in a dialysis patient is hyperkalemia until proven otherwise. Do not treat it as ventricular tachycardia and reach for an antiarrhythmic.

Managing it: stabilize, shift, eliminate

1 STABILIZE the myocardium

Raises the threshold potential so the cell is harder to depolarize chaotically. Works in minutes. Does NOT lower the potassium level at all — it buys you rhythm stability while something else works. Calcium is the hospital first line; confirm what your system carries and under whose order.

2 SHIFT potassium into cells

Nebulized albuterol via beta$_2$ stimulation. Sodium bicarbonate in the acidotic patient. Insulin with glucose in hospital. Onset in minutes to tens of minutes, and temporary — the potassium comes back out as the effect wears off.

3 ELIMINATE it from the body

Dialysis, diuresis if the kidney still works, or a binding resin. This is the only category that actually fixes the problem, and none of it happens on your ambulance. It is the reason destination matters.

What you can do on this ambulance

Monitor and twelve-lead early, and repeat it — you are trending a potassium level. · Vascular access, avoiding the fistula arm. · Treat the rhythm consequences per protocol, recognizing that atropine rarely fixes hyperkalemic bradycardia. · Contact OLMC early with a specific report: the renal history, the missed session, the ECG findings you can name, and what you are asking for. · Choose a destination that can dialyze.

REGION SOP Crush injury protocol: NORMAL SALINE before release of the crushed part, ALBUTEROL 2.5 mg NEB (repeat at 5 min, max 5 mg), and SODIUM BICARBONATE 50 mEq IV/IO as an OLMC order for crush syndrome. In cardiac arrest, SODIUM BICARBONATE 50 mEq is specified for dialysis patients. Verify your current formulary for calcium.

POTASSIUM

Hypokalemia — below 3.5 mEq/L

WHO GETS IT

- Loop and thiazide diuretics — by far the most common
- Vomiting, diarrhea, NG suction
- Alkalosis of any cause
- Insulin therapy and DKA treatment
- Repeated beta$_2$ agonist treatments
- Chronic alcohol use and poor intake
- Diuretic or laxative misuse
- Hypomagnesemia — the two travel together

HOW THEY PRESENT

- Generalized weakness and fatigue
- Muscle cramps; in severe cases, flaccid paralysis
- Decreased bowel sounds, ileus, constipation
- Palpitations, PVCs, and other ectopy
- Polyuria and thirst
- Torsades de pointes when it is severe
- Worsening digoxin toxicity if they take it

ON THE MONITOR

- Flattened or inverted T waves
- ST segment depression
- U waves — a small deflection AFTER the T, best seen in the precordial leads
- As it worsens, U merges with T and the QT looks very long
- PVCs, then polymorphic VT (torsades)

Potassium is very hard to correct while magnesium is low. That is why magnesium, not potassium, is the first drug in torsades.

CALCIUM · CA^{2+}

Contraction, Coagulation, Conduction

8.5–10.5 mg/dL total. Ninety-nine percent of it is in bone; the one percent in circulation runs muscle, clotting, and the QT interval.

Ionized vs total · QT interval · Tetany · Parathyroid

What it does, and why there are two numbers

1 Contraction

Calcium entry triggers actin–myosin cross-bridging in skeletal, cardiac, and smooth muscle. No calcium, no contraction — anywhere.

2 Conduction

The calcium current drives phase 2, the plateau of the cardiac action potential. That is why calcium levels move the QT interval in a predictable direction.

3 Coagulation

Calcium is clotting factor IV and is required at several points in the cascade. Citrate in banked blood binds it, which is why massive transfusion can cause acute hypocalcemia.

4 Total vs ionized

About half of serum calcium is bound to albumin and inactive. Ionized calcium is the free, working fraction. A malnourished patient can have a low total with a normal ionized and no symptoms.

5 Parathyroid hormone

Raises serum calcium: pulls it from bone, increases gut absorption through activated vitamin D, and increases renal reabsorption. Calcitonin does the reverse.

6 Vitamin D

Required for absorption from the gut. Deficiency, malabsorption, and chronic kidney disease all reduce it — which is why renal patients run low on calcium.

99% of body calcium is in bone. The 1% in circulation runs muscle contraction, clotting, and the plateau phase of every cardiac action potential.

Hypocalcemia — the irritable patient

WHO GETS IT

- Chronic kidney disease — low calcium with high phosphate
- Massive transfusion — citrate binds ionized calcium
- Pancreatitis
- After thyroid or parathyroid surgery
- Vitamin D deficiency, malabsorption
- Low magnesium — PTH cannot work without it
- Rhabdomyolysis
- Acute hyperventilation (alkalosis increases binding)

HOW THEY PRESENT

- Perioral and fingertip paresthesias FIRST
- Muscle cramps, then carpopedal spasm
- Generalized tetany
- LARYNGOSPASM — the airway emergency here
- Seizure
- Anxiety, irritability, confusion
- Hypotension and poor contractility when severe

AT THE BEDSIDE

- **Chvostek sign**
  - Tap over the facial nerve just in front of the ear; watch for facial twitching
- **Trousseau sign**
  - BP cuff above systolic up to 3 minutes; carpal spasm appears. More specific of the two
- **On the monitor**
  - Prolonged QT, and the torsades risk that comes with it

The tingling and carpal spasm in an acutely hyperventilating patient is real physiology — alkalosis binds ionized calcium. Treat the breathing, not the drama.

Hypercalcemia — the sluggish patient

STONES, BONES, GROANS, THRONES, AND PSYCHIATRIC OVERTONES

- **Stones**
  - Kidney stones and flank pain
- **Bones**
  - Bone pain, pathologic fracture
- **Groans**
  - Nausea, vomiting, constipation, abdominal pain, pancreatitis
- **Thrones**
  - Polyuria, then thirst and profound dehydration
- **Psychiatric overtones**
  - Depression, confusion, lethargy, psychosis, coma
- **Plus generalized weakness and decreased deep tendon reflexes**

WHO GETS IT

- Malignancy — one of the most common metabolic emergencies in cancer patients
- Primary hyperparathyroidism
- Prolonged immobilization
- Excess vitamin D or calcium supplementation
- Thiazide diuretics; lithium

WHAT YOU DO

- Isotonic fluid — these patients are genuinely, severely dry, and volume is the first-line treatment
- Monitor: expect a SHORTENED QT, the mirror image of hypocalcemia
- Watch for bradycardia and block when severe
- Ask about cancer history and about supplements

A cancer patient with new confusion, constipation, and dehydration is hypercalcemic until someone proves otherwise.

MAGNESIUM · Mg^{2+}

The Forgotten Electrolyte

1.5–2.5 mEq/L. A cofactor in hundreds of enzyme reactions, including the sodium–potassium pump itself.

Torsades · Alcohol · Obstetrics · Reflexes

Function and hypomagnesemia

WHAT MAGNESIUM DOES

- Cofactor for 300+ enzyme systems, including every ATP reaction
- REQUIRED for the sodium–potassium pump to work
- Stabilizes cardiac cell membranes
- Acts as a physiologic calcium antagonist
- Relaxes smooth muscle — the basis of its use in asthma and eclampsia

WHO RUNS LOW

- Chronic alcohol use — poor intake, GI losses, and renal wasting together
- Chronic diuretic therapy
- Prolonged diarrhea, vomiting, malabsorption
- Malnutrition and critical illness
- DKA during treatment

Neuromuscular

Tremor, muscle twitching, hyperreflexia, tetany, seizures. It looks like hypocalcemia because both produce irritable tissue.

Neurologic

Confusion, agitation, hallucinations. Easy to attribute to intoxication or withdrawal in the patient most likely to have it.

On the monitor

Prolonged QT and PR, widened QRS, ectopy — and torsades de pointes, polymorphic VT twisting around the baseline.

The partnership

Low magnesium travels with low potassium and low calcium, and neither of the others will correct until the magnesium does.

REGION SOP Torsades: contact OLMC to consider MAGNESIUM SULFATE 2 g in 100 mL D5W IVPB over 5 minutes. Note the printed exclusion — NOT to be given to renal failure or dialysis patients, who cannot excrete it.

Hypermagnesemia — where you will actually see it

WHO GETS IT — ESSENTIALLY TWO GROUPS

- Renal failure — the kidney is the only real route out
- Obstetric patients on a magnesium infusion for pre-eclampsia or eclampsia
- Heavy magnesium-containing antacid or laxative use with impaired kidneys
- Rarely, over-aggressive magnesium therapy

THE PROGRESSION, IN ORDER

- Loss of deep tendon reflexes — FIRST
- Flushing, warmth, nausea, somnolence, weakness
- Hypotension and bradycardia
- Respiratory depression
- Cardiac arrest

Why you check reflexes on a magnesium drip

Reflex loss appears BEFORE respiratory depression. The patellar reflex is therefore an early-warning monitor you can perform with your hands, in a moving ambulance, with no equipment. Check it at intervals and document each check with a time.

The obstetric interfacility transfer

- Know the infusion rate and what is hanging before you leave
- Check reflexes and respiratory rate at set intervals, not just on arrival
- Be ready to stop the infusion and support ventilation
- Calcium is the antidote — confirm whether your system carries it and under whose order

CHLORIDE · Cl^-

And the Acid–Base Connection

98–106 mEq/L. The quiet one — it follows sodium, balances bicarbonate, and shows up in the anion gap.

Anion gap · Bicarbonate · pH and potassium

CHLORIDE

The follower, the buffer partner, and the gap

1 It follows sodium

The principal extracellular anion, moving passively with sodium to maintain electroneutrality and osmotic balance. Where sodium goes, chloride generally goes.

2 It makes stomach acid

Chloride is half of hydrochloric acid. Persistent vomiting or NG suction loses acid and chloride together — producing hypochloremia with a metabolic alkalosis.

3 It trades with bicarbonate

The chloride shift in red cells lets blood carry carbon dioxide. Chloride and bicarbonate move in opposite directions to keep the charge balanced.

4 It defines the anion gap

$Na^+ - (Cl^- + HCO_3^-)$, normally about 8–12. A WIDE gap means unmeasured acid has accumulated — lactate, ketones, or a toxic alcohol.

Wide-gap acidosis — the short list

Methanol, uremia, DKA, propylene glycol, isoniazid, lactic acidosis, ethylene glycol, salicylates. You will not calculate a gap in the field — but a Kussmaul-breathing patient with an unexplained acidosis has one of a short list of causes, several of them toxicologic.

The normal saline footnote

Normal saline carries 154 mEq/L of chloride, well above the physiologic range. Large volumes push toward a hyperchloremic metabolic acidosis — one reason balanced solutions are preferred in some resuscitation settings.

ACID–BASE

How pH moves potassium — and why DKA lies to you

THE EXCHANGE

- **ACIDOSIS → serum potassium RISES**
  - H^+ moves into cells, K^+ moves out to balance the charge. Total body potassium has not changed at all
- **ALKALOSIS → serum potassium FALLS**
  - The same trade, running the other direction
- **Direction is reliable; magnitude is not**
  - Do not try to predict a number from a pH

WHY PROLONGED ARREST IS HYPERKALEMIC

No perfusion means no ATP. No ATP means the sodium–potassium pump stops. Potassium leaks out of every cell in the body while lactic acid accumulates and drives more of it out. That is why hyperkalemia sits on the reversible-cause list, and why the dialysis patient in arrest gets specific treatment in your protocol.

The DKA paradox — the hardest idea in this session

A patient in diabetic ketoacidosis usually arrives with a NORMAL OR HIGH serum potassium while being massively depleted of total body potassium. Two mechanisms did it: acidosis pushed potassium out of cells into the serum, and osmotic diuresis has been flushing that potassium into the toilet for hours or days. When insulin is started, potassium rushes back into cells and the true deficit is unmasked — fast enough to cause a lethal rhythm. That is why potassium is checked before insulin is given, and why fluids come first.

REFERENCE

One monitor, four electrolytes

Finding on the monitor	Think first	Also consider
Tall, narrow, peaked T waves	HYPERkalemia	Early ischemia; normal variant in young patients
Flat T waves with U waves	HYPOkalemia	Often with hypomagnesemia in the same patient
Widened QRS with no anatomic block pattern	HYPERkalemia	Sodium channel blockade — TCAs, cocaine; paced rhythm
Absent P waves with a wide complex	HYPERkalemia	A dialysis patient here is hyperkalemic until proven otherwise
Prolonged QT	HYPOcalcemia	HYPOmagnesemia, HYPOkalemia, and many medications
Shortened QT	HYPERcalcemia	Digoxin effect
Torsades de pointes	HYPOmagnesemia	Low potassium, low calcium, QT-prolonging drugs

The ECG rules these abnormalities IN. It never rules them out. A slowly rising potassium can produce a dangerous level with an unremarkable twelve-lead — do not let a clean strip reassure you against a strong history.

FIELD RECOGNITION

Who is at risk — the history does the diagnosing

Missed or shortened dialysis

Potassium UP. The strongest single history in this entire session. Ask what day their last session was and whether they completed it.

Crush injury, prolonged entrapment

Potassium UP, and most dangerously at the moment the pressure is released. Fluids before release, not after.

Loop or thiazide diuretics

Potassium DOWN, magnesium DOWN. The most common outpatient cause of hypokalemia by a wide margin.

Persistent vomiting or NG suction

Potassium DOWN, chloride DOWN, metabolic alkalosis from losing hydrochloric acid.

Prolonged diarrhea

Potassium DOWN and bicarbonate lost — a normal anion gap acidosis. Common and easily underestimated in the elderly.

Chronic alcohol use, poor intake

Magnesium, potassium, and calcium all DOWN together. Expect the potassium not to correct until the magnesium does.

DKA and hyperglycemia

Serum potassium normal or HIGH; total body potassium profoundly LOW. Sodium falls with osmotic shift. The most deceptive panel in medicine.

Heat illness, endurance exertion

Sodium DOWN when losses are replaced with plain water. Consider it in an unexplained seizure after prolonged exertion.

Cancer, immobility

Calcium UP. New confusion, constipation, and dehydration in an oncology patient is hypercalcemia until proven otherwise.

Two questions find most of it: what has gone IN, and what has come OUT, over the last few days.

APPLIED

Crush syndrome — the potassium bolus you release yourself

1 Compression stops perfusion

No blood flow means no ATP. The sodium–potassium pump fails, cells swell and die, and the compartment fills with potassium, myoglobin, phosphate, and lactic acid.

2 The patient looks fine

While the weight is on, all of that is trapped locally. Your protocol warns explicitly that a crush patient may initially present with very few signs and symptoms.

3 Release delivers the bolus

Lift the weight and the trapped load enters the central circulation at once. Sudden hyperkalemia, acidosis, hypotension, and dysrhythmia — sometimes arrest on the backboard.

4 So you treat before you lift

Fluids first, expanding the volume that bolus is diluted into. Coordinate extrication with treatment rather than sequencing them.

Watch the monitor at three specific moments

Before release, immediately after release, and throughout transport. You are looking for peaked T waves, a widening QRS, a lengthening QT, and loss of the P wave — the same progression from the potassium block, now on a trauma patient.

Why a bronchodilator is in a trauma protocol

Albuterol is not being given for wheezing. Beta$_2$ stimulation drives potassium into cells — the same shifting mechanism used in hospital hyperkalemia care, delivered by a route you already carry and can give while the patient is still trapped.

REGION SOP Crush injury protocol: NORMAL SALINE before release of the crushed part; ALBUTEROL 2.5 mg NEB, repeat at 5 min, max 5 mg; SODIUM BICARBONATE 50 mEq IV/IO for widened QRS, and as an OLMC order for crush syndrome. Coordinate extrication with treatment.

APPLIED

The dialysis patient

ASK THESE THREE

- **When was your last treatment?**
  - Name the day. Sunday is the long gap for a Mon/Wed/Fri schedule
- **Did you finish the whole session?**
  - A shortened run counts as a missed one — ask why it ended early
- **What is your dry weight?**
  - Compare it to today. The difference is fluid they are carrying

WHAT THEY CARRY BESIDES POTASSIUM

- Fluid overload and pulmonary edema — a missed session often presents as shortness of breath, not weakness
- Metabolic acidosis
- Low calcium with high phosphate
- Hypertension
- Anemia
- Access-site infection and bleeding

HANDLING RULES

- No BP and no IV in the arm with a graft or fistula
- Bleeding fistula: direct pressure and transport
- In arrest, a graft or fistula may be used for access — contact medical control first
- Peritoneal dialysis: do not disconnect the bags, do not infuse into the catheter, keep the drainage bag below waist height

REGION SOP Cardiac arrest protocols specify SODIUM BICARBONATE 50 mEq IV/IO for dialysis patients, and list hyper/hypokalemia among reversible causes. The torsades magnesium order carries a printed exclusion for renal failure and dialysis patients.

APPLIED

What is in the bag, and what moves the numbers

WHAT IS ACTUALLY IN THE BAG

- **Normal saline (0.9% NaCl)**
  - 154 mEq/L sodium and 154 chloride. Isotonic, no potassium, no buffer. Large volumes push toward a hyperchloremic acidosis
- **Lactated Ringer's**
  - Closer to plasma; small amounts of potassium and calcium, plus lactate metabolized to bicarbonate. Follow your protocol on renal patients
- **D5W**
  - Free water once the dextrose is used. Not a volume resuscitation fluid

ASK FOR THESE BY NAME

- Loop and thiazide diuretics → potassium and magnesium DOWN
- ACE inhibitors, ARBs, spironolactone → potassium UP
- Potassium supplements and salt substitutes → potassium UP
- $Beta_2$ agonists and insulin → potassium DOWN (shifted)
- Lithium, thiazides → calcium UP
- Digoxin → no direct effect, but its toxicity depends on all of them

Digoxin is where the electrolytes all meet

Digoxin toxicity is potentiated by LOW potassium, LOW magnesium, and HIGH calcium. The classic setup is a patient on digoxin plus a loop diuretic: nausea and vomiting, visual disturbance often described as yellow-green or halos, confusion, and a new dysrhythmia. Ask about both drugs together, and remember that the diuretic is what makes the digoxin dangerous.

CASE REVIEW

Back to your 62-year-old dialysis patient

The electrolyte

Potassium, high. Ascending weakness, bradycardia unresponsive to vagal logic, peaked T waves, vanishing P waves, and a 0.14 QRS are one diagnosis, not four findings.

Why the missed session matters

Friday to Sunday on a Mon/Wed/Fri schedule is his longest interval even when he attends. Skipping Friday means roughly three days of potassium with no route out of the body.

Which arm

The right, for both the blood pressure and the IV. The left forearm has the fistula with a palpable thrill — no cuff, no venipuncture, no exceptions short of arrest and an order.

What you can actually do

Serial twelve-leads as a potassium trend. Early, specific OLMC contact. The shifting options your protocol supports. And a destination that can dialyze — the only thing that removes potassium.

The mistake waiting to be made

Do not read that wide complex as ventricular tachycardia. He is awake, talking, and perfusing, with a history that explains everything on the screen. Antiarrhythmics will not help him and some will make him worse. In a dialysis patient, a wide complex is hyperkalemia until someone proves otherwise.

SUMMARY

What to carry out of this session

1 You predict; you do not measure

No panel on the ambulance. The history identifies who is at risk, and the monitor confirms it for potassium, calcium, and magnesium.

2 Potassium is the emergency

Narrow window, fast onset, and visible on your screen. Peaked T → flat P → wide QRS → sine wave is a countdown, not a list.

3 Sodium problems are water problems

And they present as brain problems. An unexplained seizure after heavy water intake or prolonged exertion deserves the question.

4 Calcium owns the QT

Low calcium lengthens it and makes tissue irritable; high calcium shortens it and makes everything sluggish. Two bedside signs cost you nothing.

5 Magnesium unlocks potassium

The pump needs it. Potassium will not correct while magnesium is low, which is why magnesium is the drug in torsades.

6 Shifting is not removing

Everything you can do buys transport time. Only dialysis or a working kidney removes potassium — which makes destination part of the treatment.

Electrolytes are not a chapter. They are the mechanism underneath a dozen protocols you already run.

REVIEW

Check your understanding

1. Both sodium and potassium are cations with defined normal ranges. Explain why a 0.5 mEq/L change in potassium is far more dangerous than the same change in sodium.

2. List the hyperkalemia ECG progression in order and state what each stage tells you about the time you have left.

3. A DKA patient arrives with a serum potassium of 5.4. Explain why they are nonetheless profoundly potassium-depleted, and what happens when insulin is started.

4. Your crush injury protocol includes nebulized albuterol. Explain the mechanism, and why it is there.

5. Why are fluids given BEFORE the crushed part is released rather than after?

6. Explain why potassium is difficult to correct while magnesium remains low, and what that means for torsades management.

7. Name three electrolyte abnormalities that prolong the QT interval and the rhythm they converge on.

8. A hemodialysis patient with a left forearm fistula needs an IV and a blood pressure. Describe exactly what you do and what you avoid.

Answer these before the session and again after — that gap is the measure of what you learned today.