ELECTROLYTE RELATIONSHIP TABLE

CORRECTED - QC COPY

How the major electrolytes interact and what those relationships mean clinically

↑ increases ↓ decreases ↔ closely related • Opposite = often inverse • Relationships are tendencies; the cause and the patient determine the meaning

RELATIONSHIP	WHAT HAPPENS?	WHY / PHYSIOLOGY	WHAT YOU MAY SEE	NURSING / EXAM CONNECTION
Ca ↔ phosphate OPPOSITE - often	**↓ Ca often ↔ ↑ phosphate** **↑ Ca often ↔ ↓ phosphate**	Phosphate binds Ca. PTH raises serum Ca and increases renal phosphate excretion.	High phosphate can lower Ca → tingling, spasms, **hyperreflexia**, tetany.	**Trousseau:** BP cuff → hand spasm. **Chvostek:** face tap → twitch. Severe: **SEIZURE / LARYNGOSPASM** risk. Assess neuro + **ECG**.
Mg ↔ Ca NEUROMUSCULAR / REFLEXES	**↓ Mg may cause ↓ Ca → excitability ↑** **↑ Mg → CNS / muscle depression**	Both affect nerve/muscle stability. Severe Mg deficiency can impair PTH release or action, lowering Ca.	Low: tremors, cramps, hyperreflexia, tetany. High: ↓ DTRs, lethargy, weakness, low BP/HR, **RESPIRATORY DEPRESSION**.	**Check DTRs**, breathing + **ECG**. LOW = too excitable. HIGH = too relaxed/depressed.
K ↔ acid-base MAINLY METABOLIC	**Metabolic acidosis → K tends OUT → serum K ↑** **Metabolic alkalosis → K tends IN → serum K ↓**	pH and HCO3 changes influence K shifts. Respiratory effects are usually smaller.	Either K extreme → weakness + **DYSRHYTHMIA** risk. Alkalosis may ↓ ionized Ca → cramps/tetany.	**WATCH K + ECG.** This is a tendency. Cause matters: GI loss can lower K; in DKA, insulin lack is a major reason serum K shifts out.
K ↔ insulin / glucose DIABETES	**INSULIN → GLUCOSE + K INTO CELLS** → serum K can fall	DKA causes osmotic diuresis → total-body K loss. Insulin then moves K into cells.	Serum K may start normal/high, then fall during treatment → weakness + ECG/rhythm changes.	Trend K before/during insulin; monitor rhythm as indicated. **SERUM K ≠ TOTAL-BODY K.**
Cl ↔ bicarbonate ACID-BASE	**Vomiting / NG:** lose H+ + Cl → hypochloremic alkalosis; K often ↓. **Diarrhea:** lose HCO3 → metabolic acidosis; Cl may ↑ and K often ↓.	Gastric loss removes acid + Cl. Intestinal loss removes HCO3. Interpret Cl and HCO3 together.	Vomiting/NG: weakness, cramps, alkalosis. Diarrhea: dehydration, acidosis; deep/fast breathing may occur.	**READ TOGETHER: Cl + HCO3 + K + FLUID STATUS.**
Na ↔ water WATER ↔ BRAIN	**Water excess vs Na → dilution → Na ↓** **Water deficit vs Na → concentration → Na ↑**	Serum Na reflects water relative to Na, not total-body Na. Water moves toward higher osmolality.	Brain cells swell or shrink. Significant changes → confusion, LOC change, **SEIZURES**.	**SODIUM = NEURO + FLUID.** Check neuro, I/O, weight. **USE CONTROLLED CORRECTION** → rapid shifts can cause neurologic injury.
Na ↔ hormones ADH / ALDOSTERONE	**↑ ADH:** retain water → dilute Na ↓ **↓ / ineffective ADH (DI):** water loss → Na may ↑ **Aldosterone:** retain Na; water follows; excrete K	ADH controls water retention. Aldosterone promotes Na retention and K excretion.	SIADH → dilutional hyponatremia. DI → high urine output, water loss, hypernatremia risk. Excess aldosterone → K may fall.	Compare **Na + K + I/O + urine output + BP + daily weight.**
Phosphate ↔ energy ATP / MUSCLE / RESPIRATORY	**↓ phosphate → ↓ cellular energy / ATP** → muscle function ↓	Phosphate is needed for cellular energy production and muscle function.	General weakness, weak respiratory muscles, poor activity tolerance, ↓ cardiac performance, fall risk.	**LOW PHOSPHATE = THINK WEAK.** Check breathing strength, mobility, fall/cardiac risk + **RENAL FUNCTION**.
Mg ↔ K CORRECTION CLUE	**↓ Mg → kidney K wasting** → low K may persist	Mg deficiency makes it harder for the kidneys to retain K.	K stays low despite replacement; combined deficits increase rhythm risk.	**K not improving? CHECK Mg.** Monitor **ECG + kidney function**.
Kidneys ↔ all PUT IT TOGETHER	**Impaired regulation / excretion** → several abnormalities at once	Kidneys regulate electrolytes, acid-base and fluid. With kidney failure, K, Mg and phosphate may rise; Ca may fall.	Rhythm, reflex, weakness, fluid and pH changes may occur together.	**THINK: KIDNEYS + GI LOSSES + MEDS + FLUIDS.** Review BUN/Cr, urine output, I/O, meds, orders + trends.

PATTERNS TO RECOGNIZE ON AN EXAM

METABOLIC ACIDOSIS
K tends OUT; effect depends on cause
DKA: total-body K is depleted

METABOLIC ALKALOSIS
K tends IN → K ↓
Ionized Ca ↓ → tetany risk

Ca / PHOSPHATE
Often opposite
High phosphate can produce low-Ca findings

LOW Mg + LOW Ca
Excitability ↑
Cramps / hyperreflexia / tetany

VOMITING / NG
H+ + Cl lost → alkalosis
K often ↓

DIARRHEA
HCO3 lost → acidosis
Cl may ↑; K often ↓ from GI loss

INSULIN / DKA
Insulin moves K into cells
Serum K may fall; trend closely

RENAL DYSFUNCTION
K / Mg / phosphate may build up
Check creatinine + urine output

Course basis: NUR 122 electrolyte and acid-base lectures/objectives. Clinical clarifications preserve course intent while distinguishing metabolic K shifts and total-body K loss in DKA.

Review: Lippincott Ch. 10, 17 and 48 • ATI Ch. 45-46 • Exam 1: Days 1-2 exemplars.