



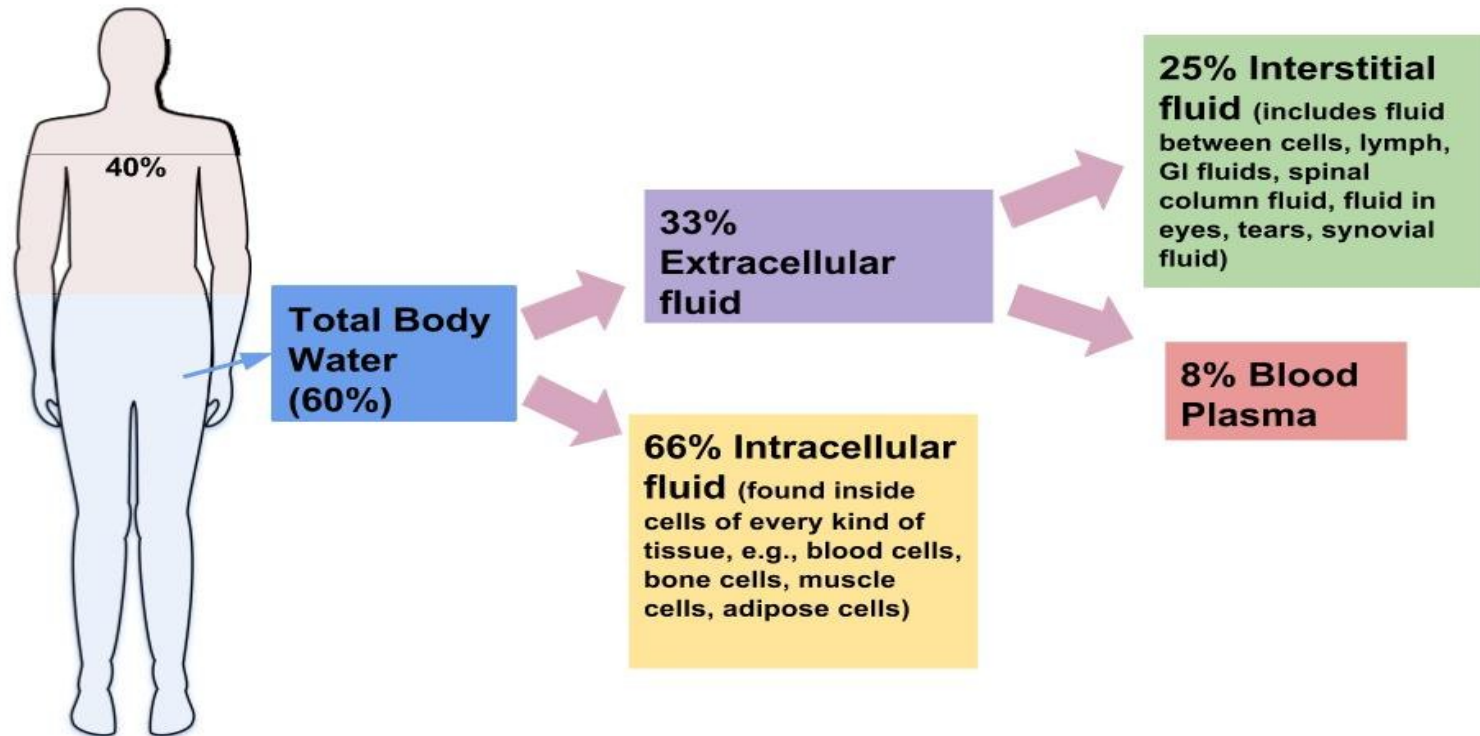
Electrolytes and acid base balance

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Total Body Water



Total Body water = 60 % Body weight



Osmolality

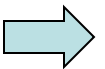
The formula to calculate **effective osmolality or tonicity** is

$$2 \times [\text{Na}^+] + \text{glucose}/18$$

(normal range, 275 to 290 mOsm/L).

When add free water ECF crosses the cell membrane into ICF to equalize ECF osmolality TBW expansion and slight reduction in osmolality.

When add isotonic saline no movement of water into the cells ECF expansion only



Osmolar gap

The osmolar gap:

measured osmolality – calculated osmolality.

Normal ≈ 10 mOsm/L.

If it is >15 mOsm/L, it means that a nondetectable agent with osmotic activity is present.

A consequent osmotic diuresis will cause $[\text{Na}^+]$ deficit with volume depletion that should be treated with saline solution

Total Body Water

Fluid loss



Osmolality

Osmoreceptors in the hypothalamus stimulate thirst and the release of antidiuretic hormone

← **Circulating blood volume, low-pressure baroreceptors in the great veins and the right atrium augment the effect on the osmoreceptors,**

urine water reabsorption and vasoconstriction.

Hyponatremia

Definition:

Serum $[Na^+]$ <138 mEq/L.

Types:

1. Hyperosmolar hyponatremia:

Plasma osmolality [POSM] >295 mOsm/kg H₂O

severe hyperglycemia (increase in plasma glucose above the normal level of 100 milligrams/dL decreases the serum $[Na^+]$ by 1.6 mEq/L).

administration of osmotic agents such as mannitol, glycerol, and maltose.

Hyponatremia

2. Iso-osmolar hyponatremia:

Plasma osmolality 275 - 295 mOsm/kg H₂O
occurs in severe hyperproteinemia or
hyperlipidemia.

Patients are asymptomatic; treatment is not
needed.

3. Hypo-osmolar hyponatremia:

Plasma osmolality <275 mOsm/kg H₂O.

Classification:

A. Hypervolemic hyponatremia.

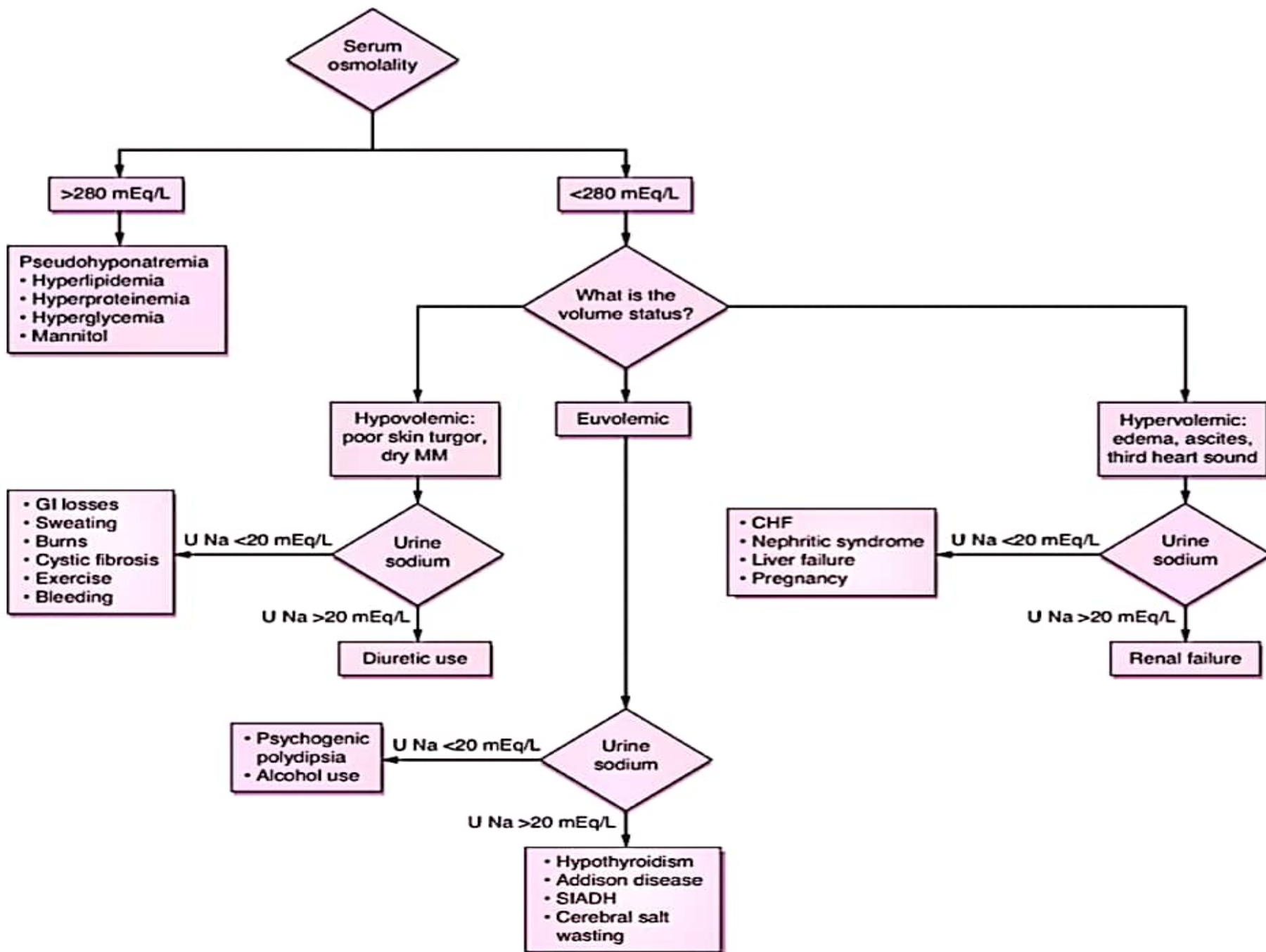
B. Normovolemic hyponatremia.

C. Hypovolemic hyponatremia:

Renal

Extrarenal

TABLE 17-4 Classification, Differential Diagnosis, and Features of Hyponatremia According to Volume Status					
	Clinical Conditions	Orthostatic Hypotension	Edema	$U_{(Na+)}$ mEq/L	U_{osm} mOsm/L
Hypervolemic hyponatremia	CHF Cirrhosis Nephrotic syndrome Acute and chronic kidney disease	Absent	Yes	Compensated: >20 Decompensated: <10	Compensated: <100 Decompensated: >100
Normovolemic hyponatremia	Psychogenic polydipsia Glucocorticoid deficit Hypokalemia Drugs SIADH	Absent	No	>20	>100
Renal hypovolemic hyponatremia	Diuretics Mineralocorticoid deficit Salt-losing nephropathy	Normally present	No	>20	>100
Extrarenal hypovolemic hyponatremia	Vomiting Diarrhea	Normally present	No	<10	>100



Hyponatraemia treatment Algorithm

Low Osmolality <285 mOsm Hypotonic Hyponatremia, "True hyponatremia"

High Osmolality >295 mOsm (Pseudohyponatremia)
E.g. Hyperglycaemia, mannitol, glycine.

Normal Osmolality 285-295 mOsm (Pseudohyponatremia)
Hyperlipidaemia, Hyperproteinaemia
Hypertriglyceridemia, Hyperglobulinemia

Hypovolaemic Hyponatraemia
Total body water ↓
Total body sodium ↓↓

Hypervolaemic Hyponatraemia
Total body water ↑↑
Total body sodium ↑

Euvolemia
Total Body Water ↑
Total Body Sodium ↔

Urine Na <20mmol/L, **Extra renal loss** -vomiting, diarrhoea, burns, bowel obstruction, pancreatitis, trauma, excessive sweating.

Urine Na > 20mmol/L, **Renal loss** -Diuretics, renal failure, mineralocorticoid deficiency, chronic pyelonephritis, nephrocalcinosis, proximal renal tubule acicosis, ketonuria, salt losing nephritis, cerebral salt wasting syndrome.

Urine Na usually <20-30 mmol/L due to activation of the renin-angiotensin- aldosterone system with secondary renal sodium conservation despite the whole-body volume overload.

Causes: CCF, cirrhosis, nephrotic syndrome

Urine osmolality <100 mOsm/kg.
Urine sodium >20 mmol/L
Primary polydipsia, Inappropriate IVI (iatrogenic) Low solute intake e.g. beer potomania.

Urine osmolality >100 mOsm/kg reflects inappropriate antidiuresis. Urine sodium > 20 mmol/L
Causes: Hypothyroidism (TSH level), Glucocorticoid deficiency; SIADH.

Treatment: Correct underlying cause
Acute (<48 hours): Consider hypertonic saline e.g. 3% NaCl. Stop diuretics, restore volume with 0.9% NaCl.
Chronic (>48 hours): 0.9% NaCl to correct fluid deficit and slowly correcting to baseline value.

Treatment:
Usually **chronic** (> 48 hours): Fluid restriction. If **acute** (<48 hours): Hypertonic saline and loop diuretic. Address underlying disease; restrict salt and water intake.

Treatment:
Fluid restrict. Treat underlying cause. Assess chronicity of disturbance and treat as below.

The chronicity of the sodium disturbance should direct treatment. Correction needs to be much slower in chronic cases. Treatment should aim to reverse symptoms and prevent cerebral oedema in acute patients but not so rapid as to pose a risk for developing osmotic demyelination in chronic patients.

Chronic- correct by 6 - 8 mmol/L/day. (Caution for patients with severe malnutrition, alcoholism, advanced liver disease - may need to target 4 - 6 mmol/L/day).
Water restriction.

Acute - Consider 100-mL bolus of 3% saline infused over 10 minutes and repeated twice as needed if severe neuro symptoms. Continue to correct sodium to normal quickly if hyponatremic for only several hours (up to 2 mmol/hour) Once symptoms have resolved correction can be slowed. If hypertonic saline is used seek expert advice and consider intensive care admission. Measure sodium every 1-2 hours. Consider loop diuretic to enhance free water clearance. Water restriction.

Clinical Features

Symptoms are effects on the brain.

Divided into:

Mild $[\text{Na}^+]$ 130-135 mEq/L

Moderate $[\text{Na}^+]$ 120-130 mEq/L

headache, nausea, disorientation,
confusion, agitation, ataxia, and
areflexia.

Sever $[\text{Na}^+]$ <120 mEq/L

intractable vomiting, seizures, coma, and
ultimately respiratory arrest due to
brainstem herniation.

TABLE 17-7**Treatment of Hyponatremia Symptomatic With Seizures or Coma**

Step 1	Assess for indication for 3% hypertonic saline: severe symptoms of hyponatremia such as seizures or coma with suspected impending brainstem herniation in setting of acute* or chronic† hyponatremia.
Step 2	Infuse 100–150 mL of 3% hypertonic saline IV over 15–20 min.†
Step 3	Measure serum sodium level after each 3% hypertonic saline infusion.
Step 4	Stop infusion when symptoms improve or a target of a 5 mEq/L (range, 4–6 mEq/L) increase in serum sodium concentration is achieved.
Step 5	May repeat 150 mL of 3% hypertonic saline up to 3 total doses, or a total of 450 mL IV of 3% hypertonic saline.
Step 6	Keep the IV line open with minimal volume of 0.9% normal saline until cause-specific treatment is started. Limit increase in sodium level to no more than 8–12 mEq/L during the first 24 h or 18 mEq/L over 48 h.

Osmotic Demyelination Syndrome

It is caused by rapid correction of hyponatremia (>12 mEq/L/24 h) as water moves from cells to ECF yielding intracellular dehydration.

Risk factors:

- $[\text{Na}^+] < 120$ mEq/L, chronic heart failure, alcoholism, cirrhosis, hypokalemia, malnutrition, and treatment with vasopressin antagonists such as tolvaptan.

Main symptoms are dysarthria, dysphagia, lethargy, paraparesis or quadriparesis, seizures, and coma.

Osmotic Demyelination Syndrome

The treatment of $[Na^+]$ overcorrection consists of:

- Giving 5% dextrose in water at 3 mL/kg/h.
- Loop diuretics.
- Desmopressin.

Hypernatremia

Definition:

Serum $[Na^+]$ >145 mEq/L and serum osmolality >295 mOsm/L.

When $[Na^+]$ and osmolality increase, normal subjects become thirsty and drink free water, and the Na^+ level returns toward normal.

Any situation that impairs the patient's sense of thirst, limits the availability of water, limits the kidney's ability to concentrate urine, or results in increased salt intake predisposes the patient to hypernatremia.

TABLE 17-9 Hypermnatremia Classification and Features According to Volume Status				
Volume Status	Clinical Conditions	Diagnosis	U_{Osm} , mOsm/kg H_2O	$U_{\text{Na+}}$, mEq/L
Hypervolemic hypernatremia	Cushing's syndrome Primary hyperaldosteronism Salt water intake Iatrogenic Hemodialysis	Cortisol test History of hypertension and hypokalemia Psychiatric disorder Hypertonic saline, enteral feeding, bicarbonate infusion Clinical history	>100	>20
Normovolemic hypernatremia	Central DI Partial DI Nephrogenic DI Hypodipsia Medications	History of CNS lesion, urinary concentration after desmopressin History of nephrotoxic drugs, no response to desmopressin History of poor oral intake Amphotericin, aminoglycosides, lithium, phenytoin	Central DI <300 Partial DI >300 but <800 <200 >100 <200	>20
Renal hypovolemic hypernatremia	Osmotic diuretics Loop diuretics Postobstructive diuresis	Hyperglycemia. High sodium level after correction Clinical history Clinical history	>100	>20
Extrarenal hypovolemic hypernatremia	Vomiting Diarrhea GI fistulas Sweating Burns	Clinical history	>800	<10

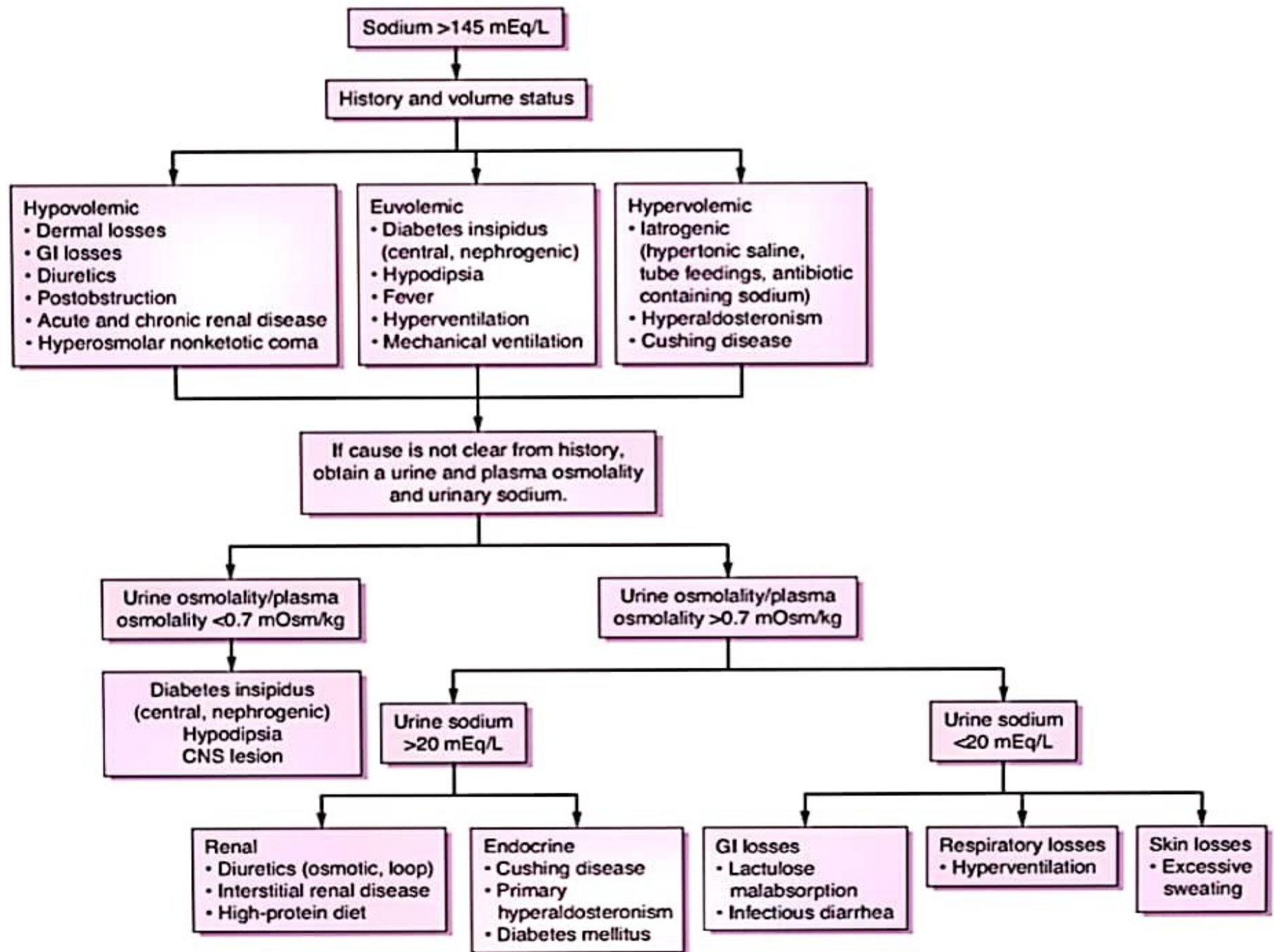


TABLE 17-11**Treatment of Hyponatremia**

Treatment	Indication and Comments
Isotonic (0.9%) saline	Use for correction of volume deficits.
Etiology-specific therapy	Treat fever with antipyretics, vomiting with antiemetics, and diabetes insipidus with desmopressin (see “Diabetes Insipidus” section).
D ₅ W or oral free water to replace free water deficit over 2–3 d	In cases of chronic hyponatremia, it is suggested that correcting (lowering) the sodium level should occur at a rate of no more than 0.5 mEq/L/h or 10–12 mEq/24 h.
0.45% normal saline at 100 mL/h	Correct volume deficits first. A commonly used infusion for mild to moderate hyponatremia, but this therapy adds sodium to total body.
D ₅ W to replace free water deficit over 1–2 h	Reserved only for those cases where acuity is known to be <6 h and the salt load is known to be lethal (0.75–3.0 grams/kg of body weight).
Hemodialysis	An alternative or as a supplement to D ₅ W to replace free water deficit in life-threatening acute cases of salt ingestion.

Hypokalemia

Definition

serum $[K^+]$ of <3.5 mEq/L.

TABLE 17-13	Causes of Hypokalemia
Transcellular shifts	Alkalosis* Increased plasma insulin (treatment of diabetic ketoacidosis) β -Adrenergic agonists Hypokalemic periodic paralysis (congenital) Thyrotoxic hypokalemic periodic paralysis
Decreased intake	Fasting Alcoholism (worsened by hypomagnesemia) Eating disorders
GI loss	Vomiting*, nasogastric suction Diarrhea* (including laxative, enema abuse) Malabsorption Ureterosigmoidostomy Enteric fistula Villous adenoma
Renal loss	Diuretics (carbonic anhydrase inhibitors, loop diuretics, and thiazide-like diuretics)* Primary hyperaldosteronism Secondary hyperaldosteronism Licorice ingestion Excessive use of chewing tobacco Renal tubular acidosis Postobstructive diuresis Osmotic diuresis Bartter's syndrome (mimics loop diuretic use) Gitelman's syndrome (mimics thiazide diuretic use) Apparent mineralocorticoid excess and related syndromes (Conn's, Liddle's) Drugs and toxins (aminoglycosides, echinocandins, carbenicillin, penicillins, amphotericin B, levodopa, lithium, thallium, cesium, barium, toluene, theophylline, chloroquine, steroids, etc.)
Sweat loss	Heavy exercise Heatstroke Fever
Other	Hypomagnesemia Acute leukemia and lymphomas IV hyperalimentation Recovery from megaloblastic anemia Hypothermia (accidental or induced)

TABLE 17-14**Symptoms and Signs of Hypokalemia**

Cardiovascular	- Hypertension - Orthostatic hypotension - Potential of digitalis toxicity - Dysrhythmias (usually tachyarrhythmias) - T-wave flattening, QT prolongation, U waves, ST depression
Neuromuscular	- Malaise, weakness, fatigue - Hyporeflexia - Cramps - Paresthesias - Paralysis - Rhabdomyolysis
GI	- Nausea, vomiting - Abdominal distention - Ileus
Renal	- Increased ammonia production - Urinary concentrating defects - Metabolic alkalemia, paradoxical aciduria - Nephrogenic diabetes insipidus
Endocrine	Glucose intolerance

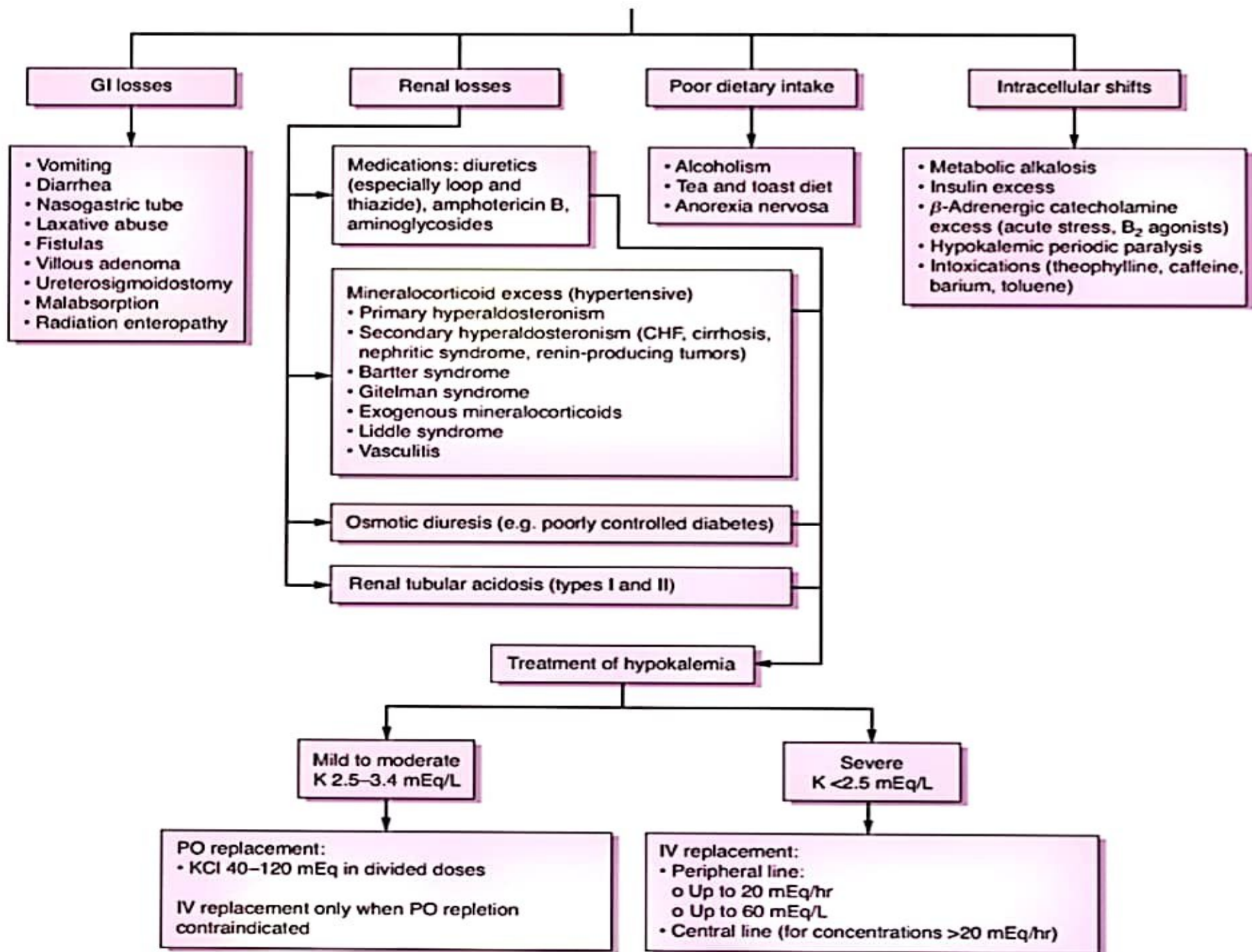
Treatment

general principles in hypokalemia correction:

1. Use potassium chloride and avoid administering K^+ in glucose solutions.
2. Maximum recommended $[K^+]$ in 500 mL of a saline solution is 40 mEq, over 4 to 6 hours in a peripheral line).

Higher concentrations use a central line, but not exceed 20 mEq/h.

3. Reassessing serum $[K^+]$
4. ECG monitoring
5. Add magnesium (20 to 60 mEq/24 h) to the infusion for optimize tubular reuptake of potassium



Hyperkalemia

Definition:

measured serum $[K^+]$ of >5.5 mEq/L.

Due to release of intracellular potassium caused by hemolysis.

Clinical manifestations of hyperkalemia result from disordered membrane polarization.

Cardiac manifestations are the most serious.

TABLE 17-17

Causes of Hyperkalemia

Pseudohyperkalemia	Tourniquet use Hemolysis (in vitro)* Leukocytosis Thrombocytosis
Intracellular to extracellular potassium shift	Acidosis* Heavy exercise β -Blockade Insulin deficiency Digitalis intoxication Hyperkalemic periodic paralysis
Potassium load	Potassium supplements Potassium-rich foods IV potassium Potassium-containing drugs Transfusion of aged blood Hemolysis (in vivo) GI bleeding Cell destruction after chemotherapy Rhabdomyolysis/crush injury* Extensive tissue necrosis
Decreased potassium excretion	Renal failure* Drugs—potassium-sparing diuretics,* β -blockade, NSAIDs, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, cyclosporine, tacrolimus Aldosterone deficiency* Selective defect in renal potassium excretion (pseudohypoaldosteronism, systemic lupus erythematosus, sickle cell disease, obstructive uropathy, renal transplantation, type IV renal tubular acidosis)

K >5.5 mEq/L

Common causes: renal failure, hyperglycemia, specimen delay and hemolysis, renal insufficiency, acidosis, rhabdomyolysis, insulin deficiency, adrenal insufficiency, medications, massive blood transfusions, tumor lysis, ischemic bowel

Weakness, nausea, paresthesias, ileus, flaccid paralysis

ECG changes: peaked T waves, ↑ PR interval, ↑ QRS width, sine wave pattern, PEA

Repeat test.

No

Normal

Pseudohyperkalemia:
IVF with K, hemolyzed sample

Abnormal

Check medications.

Treat as acute hyperkalemia.

Yes

No suspicious medications

ABGs, GFR, Cr, BUN, glucose, electrolytes, digoxin levels, CPK, urine K+

ACEI, ARBs, β -blockers, NSAIDs, penicillin K, TMP-SMX, digitalis, K-sparing diuretics, heparin, cyclosporine, tacrolimus, pentamidine, succinylcholine

Discontinue medication or adjust dose.

Urine K >30 mEq/L:
transcellular shift

**Acidosis
Rhabdomyolysis
Hyperglycemia
Burns**

Urine K <30 mEq/L:
impaired renal excretion

**Renal insufficiency
Adrenal insufficiency
Hyporeninemic
hypoaldosteronism**

Treatment

Based on the action mechanism can be divided into:

- Membrane stabilization (crucial for cardiac tissue, must be done immediately),

- Intracellular shift of K^+ ,

- Removal/excretion of K^+ from the body.

TABLE 17-19 Emergency Therapy of Hyperkalemia				
Therapy	Dose and Route	Onset of Action	Duration of Effect	Mechanism
Calcium chloride (10%)*	5–10 mL IV	1–3 min	30–50 min	Membrane stabilization
Calcium gluconate (10%)*	10–20 mL IV	1–3 min	30–50 min	Membrane stabilization
NaHCO ₃ [†]	50–150 mEq IV if accompanying metabolic acidosis	5–10 min	1–2 h	Shifts [K ⁺] into cell
Albuterol (nebulized)	10–20 milligrams in 4 mL of normal saline, nebulized over 10 min	15–30 min	2–4 h	Upregulates cyclic adenosine monophosphate, shifts [K ⁺] into cell
Insulin [‡] and glucose [‡]	5–10 units regular insulin IV Glucose 25 grams (50% solution) IV	30 min	4–6 h	Shifts [K ⁺] into cell
Furosemide	40–80 milligrams IV	Varies	Varies	Renal [K ⁺] excretion
Sodium polystyrene sulfonate	25–50 grams PO or PR	1–2 h	4–6 h	GI [K ⁺] excretion
Hemodialysis	—	Minutes	Varies	Removes [K ⁺]

Hypocalcemia

[Ca²⁺] level < 8.8 mg/dL or Ionized
[Ca²⁺] level < 2.0 mEq/L (< 4 mg/dL or
< 1.1 mmol/L).

TABLE 17-24

Selected Causes of Hypocalcemia

Cause	Mechanism(s)
Decreased calcium absorption	
Vitamin D deficiency Decreased oral intake Decreased intestinal absorption Decreased production of 25(OH)D ₃ Decreased synthesis of 1,25(OH ₂)D ₃ Malabsorption syndromes	Malnutrition Intestinal bypass, gastrectomy Liver failure Renal failure, hyperphosphatemia Malabsorption
Increased calcium excretion/reduced bone resorption	
Alcoholism	Hypomagnesemia causing inhibition of PTH secretion, PTH resistance to bone resorption
Hypoparathyroidism	Genetic, autoimmune, surgical, tumoral
Pseudohypoparathyroidism	Resistance to PTH action
Hypomagnesemia	Inhibition of PTH secretion, PTH resistance to bone resorption
Drugs (Table 17-25)	
Malignancy	Pseudohypocalcemia, hyperphosphatemia, hypomagnesemia, vitamin D or PTH deficiency, osteoblastic metastasis
Sepsis	
Acute pancreatitis	Fatty acids combine with [Ca ²⁺] to form insoluble Ca ²⁺ soaps and lead to a reduction of serum [Ca ²⁺]
Massive transfusions	
Rhabdomyolysis	

TABLE 17-26**Symptoms and Signs of Hypocalcemia**

Muscular	Weakness, fatigue Spasms, cramps
Neurologic	Seizures Tetany Chvostek sign, Trousseau sign Circumoral and digital paresthesias Impaired memory, confusion Hallucinations, dementia Extrapyramidal disorders
Dermatologic	Hyperpigmentation Coarse, brittle hair Dry, scaly skin
Cardiovascular	Heart failure Ventricular arrhythmias, QT _c prolongation leading to torsades de pointes Vasoconstriction
Skeletal	Osteodystrophy Rickets Osteomalacia
Miscellaneous	Dental hypoplasia Cataracts Decreased insulin secretion

Treatment

elemental Ca^{2+} (500 to 3000 mg) orally daily, in one dose or up to three divided doses.

IV Ca^{2+} is recommended only in cases of symptomatic or severe hypocalcemia (ionized $[\text{Ca}^{2+}] < 1.9 \text{ mEq/L}$ or $< 0.95 \text{ mmol/L}$).

IV Ca^{2+} gluconate is preferred over IV calcium chloride (CaCl_2)

(10 to 30 mL of 10% Ca^{2+} gluconate) given IV over 10 to 20 minutes and repeated every 60 minutes until symptoms resolve or followed by a continuous IV infusion of 0.02 to 0.08 mL/kg/h

Hypocalcemia is difficult to correct if hypomagnesemia is also present.

Hypercalcemia

[Ca²⁺] >10.5 mg/dL or an ionized [Ca²⁺] level >2.7 mEq/L.

Mild 10.5 - 11.9 mg/dL;

Moderate 12 - 13.9 mg/dL;

Severe >14 mg/dL

Each 1 g/dL reduction in the serum albumin concentration will lower the total calcium concentration by approximately 0.8 mg/dL (0.2 mmol/L) without affecting the ionized calcium concentration and, therefore, without producing any symptoms or signs of hypocalcemia

TABLE 17-27 Causes of Hypercalcemia

Cause	Mechanism
<i>Hypercalcemia due to increased bone Ca^{2+} resorption</i> Primary hyperparathyroidism Malignancy Pseudohyperparathyroidism Renal failure Addison's disease Hyperthyroidism Immobilization	$\uparrow$ PTH Osteolysis, PTH-related protein (PTHrP) production PTH from non-parathyroid tissue source Secondary and tertiary hyper-PTH due to chronic hypocalcemia $\uparrow$ Albumin, bone resorption Increased bone resorption Osteoclast activation
<i>Hypercalcemia due to decreased urinary Ca^{2+} excretion</i> Familial hypercalcemic hypocalciuria Thiazides	Mutation of CaSR Increased kidney Ca^{2+} reabsorption in proximal tubule
<i>Hypercalcemia due to increased GI Ca^{2+} absorption</i> Granulomatous diseases (sarcoidosis, tuberculosis, coccidioidomycosis, histoplasmosis) Milk (calcium)-alkali syndrome Vitamin D intoxication	1α -Hydroxylase activity $\uparrow$ Ca^{2+} intake (calcium carbonate) and absorption Increased calcium absorption and bone resorption

TABLE 17-28**Signs and Symptoms of Hypercalcemia****General**

Malaise, weakness

Polydipsia, dehydration

Neurologic

Confusion

Apathy, depression, stupor

Decreased memory

Irritability

Hallucinations

Headache

Ataxia

Hyporeflexia, hypotonia

Mental retardation (infants)

Metastatic calcification

Band keratopathy

Conjunctivitis

Pruritus

Skeletal

Fractures

Bone pain

Deformities

Cardiovascular

Hypertension

Dysrhythmias

Vascular calcifications

ECG abnormalities

QT shortening

Coving of ST-T wave

Widening of T wave

Digitalis sensitivity

GI

Anorexia, weight loss

Nausea, vomiting

Constipation

Abdominal pain

Peptic ulcer disease

Pancreatitis

Urologic

Polyuria, nocturia

Renal insufficiency

Nephrolithiasis

Common causes: lab error, chronic renal failure, postsurgical hypoparathyroidism, hypoalbuminemia, hypomagnesemia, hyperphosphatemia, medication, PTH deficiency or resistance, vitamin D deficiency or resistance

Check labs: Repeat serum calcium, ionized calcium, electrolytes, BUN, creatinine, magnesium, phosphorus, albumin, LFTs, PT, PTT, PTH.

Serum calcium
>8.5 mg/dL

Probable false-
positive result

Calcium <8.5 mg/dL,
ionized or corrected calcium
low, albumin normal or
slightly low

If ionized calcium not
available, correct calcium for
albumin; calcium concentration
falls by 0.8 mg/dL for every
1 g/dL fall in albumin.

Magnesium low

Hypomagnesemia

Phosphorus low

Hypoparathyroidism
Rickets
Vitamin D
deficiencies

PTH high

Pseudohypoparathyroidism
Abnormalities of vitamin D
metabolism

PTH low

Hypoparathyroidism

Calcium <8.5 mg/dL,
ionized or corrected calcium
normal, albumin
low

If ionized calcium not
available, correct calcium for
albumin; calcium concentration
falls by 0.8 mg/dL for every
1 g/dL fall in albumin.

Hypoalbuminemia

Cirrhosis
Nephrotic syndrome
Malnutrition
Burns
Chronic illness
Sepsis

Treatment

Symptomatic patients or asymptomatic patients with $[Ca^{2+}]$ levels >14 mg/dL should receive treatment starting with volume repletion.

Administer **0.9% normal saline at 500 to 1000 mL/h for 2 to 4 hours as tolerated by the patient.**

3 to 4 L should be given over the first 24 hours, then 2 to 3 L per 24 hours until a urine output of 2 L/d is achieved

Treatment

Furosemide is recommended to promote a diuresis of 150 to 200 mL/h, which increases the calciuric effect, with an initial dose of 20 to 40 milligrams.

In very severe cases, it will be necessary to receive **hemodialysis** to quickly remove Ca^{2+} from blood.

for hypercalcemia associated with malignancy, IV bisphosphonates are now considered (Zoledronic acid, Calcitonin).

Hypomagnesemia

serum magnesium concentration below
1.2 mEq/L,

TABLE 17-20	Causes of Hypomagnesemia	
Redistribution	IV glucose Correction of diabetic ketoacidosis IV hyperalimentation Refeeding after starvation Acute pancreatitis Postparathyroidectomy (hungry bone syndrome) Osteoblastic metastasis (hungry bone syndrome)	
Extrarenal loss	Nasogastric suction (infrequent) Lactation Profuse sweating, burns, sepsis Intestinal or biliary fistula Diarrhea	
Decreased intake	Alcoholism (cirrhosis) Malnutrition, poor intake Small bowel resection Malabsorption (steatorrhea)	
Renal loss	Ketoacidosis Saline or osmotic diuresis Potassium depletion Phosphorus depletion Familial hypophosphatemia Tubulointerstitial renal disease	
Drugs	Loop diuretics Aminoglycosides Amphotericin B Vitamin D intoxication Alcohol Cisplatin Theophylline Proton pump inhibitors Calcineurin inhibitors (cyclosporine, tacrolimus)	
Endocrine disorders	Syndrome of inappropriate antidiuretic hormone secretion Hyperthyroidism Hyperparathyroidism Hypercalcemic states Primary or secondary aldosteronism	

TABLE 17-21

Symptoms and Signs of Hypomagnesemia

Neuromuscular	Tetany Muscle weakness Chvostek and Trousseau signs Cerebellar (ataxia, nystagmus, vertigo) Confusion, obtundation, coma Seizures Apathy, depression Irritability Paresthesias
GI	Dysphagia Anorexia, nausea
Cardiovascular	Heart failure Dysrhythmias Hypotension
Miscellaneous	Hypokalemia Hypocalcemia Anemia

Treatment

Hypokalemia, hypocalcemia, hypophosphatemia are present with hypomagnesemia.

Hypocalcemia develops when $\text{Mg}^{2+} < 1.2 \text{ mg/dL}$.

For asymptomatic patients magnesium supplements should be administered orally.

For severe and symptomatic hypomagnesemia, urgent IV magnesium sulfate (MgSO_4).

In life-threatening conditions (torsades de pointes, eclampsia), 1 to 4 grams or 8 to 32 mEq diluted in at least 100 mL over 10 to 60 minutes under continuous monitoring.

Spironolactone is effective in maintaining $[\text{Mg}^{2+}]$ homeostasis as well as in reducing the incidence of arrhythmias in congestive heart failure patients

Hypermagnesemia

serum magnesium concentration above 2.2 mg/dL.

TABLE 17-22

Causes of Hypermagnesemia

Renal Failure	Acute or Chronic
Increased magnesium load	Magnesium-containing laxatives, antacids, or enemas* Treatment of preeclampsia/eclampsia (mothers and neonates) Diabetic ketoacidosis (untreated)* Tumor lysis Rhabdomyolysis*
Increased renal magnesium absorption	Hyperparathyroidism Familial hypocalciuric hypercalcemia Hypothyroidism Mineralocorticoid deficiency, adrenal insufficiency (Addison's disease)

TABLE 17-23**Symptoms and Signs of Hypermagnesemia**

Magnesium Level (mEq/L)	Clinical Manifestations
2.0–3.0	Nausea
3.0–4.0	Somnolence
4.0–8.0	Loss of deep tendon reflexes
8.0–12.0	Respiratory depression
12.0–15.0	Hypotension, heart block, cardiac arrest

Treatment

Furosemide (40 to 80 mg IV) with fluids may be indicated (no R.F).

Calcium directly antagonizes the cardiac effects of magnesium.

10 mL of 10% calcium chloride IV over 2 to 3 minutes.

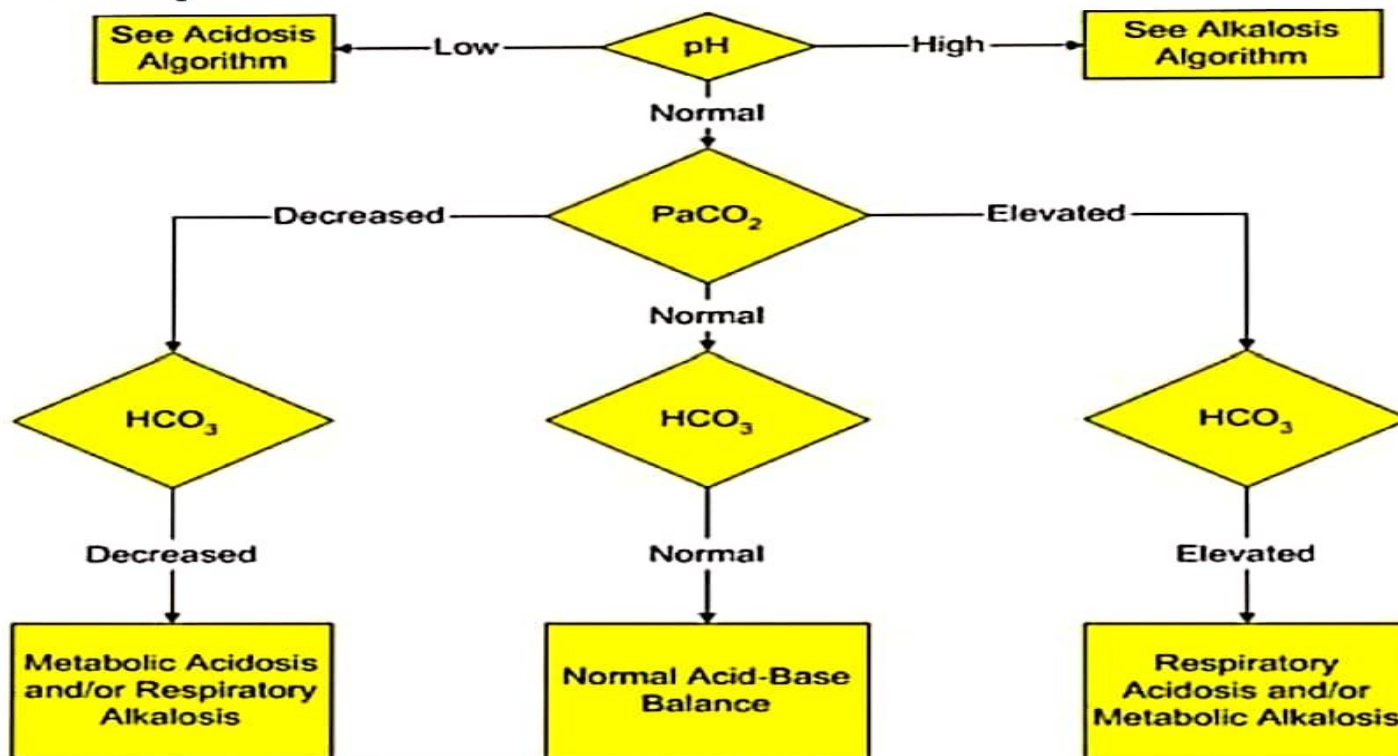
Dialysis for patients with renal failure.

HYPO natremia ↓Na <135 "SALT LOSS"			HYPO kalemia ↓K <3.5 "slow and low /A SIC WALT"		
S/S muscle spasm weakness urine loss (↓output) shallow respiration ↓DTR orthostatic hypotension ↑bowel motility			S/S Alkalosis irritability lethargy shallow respirations ↓ breath sounds ↑BP lethal cardiac Δs thread pulse ↓bowel constipation confusion		
Causes Na excretion (renal, NG suction, V/D, diuretics, sweating) SIADH ↓aldosterone diabetes insipidus Fluid overload or FVD low intake			Causes Drugs anorexia N NPO fluid loss ↑H2O intake cushings disease ↑aldosterone		
HYPER natremia ↑Na >145 "no FRIED food"/think dehydrated				HYPER kalemia ↑K >5.0 "MURDER"	
S/S Fever, flushed skin dry mouth/skin Restless agitated ↑fluid retention, edema confusion		S/S Muscle spasm/ cramps, twitching weakness Seizures urine loss, ↓output ↓BP Shallow resp. weak pulse rhythmΔs			
Causes Hypercortisolism (cushings) hyperventilation IncR intake (oral/IV) hypoaldosteronism GI tube w/o adequate H2O intake Hypertonic solutions thirst impairment Reduced excretion corticosteroids Infection, fever, sweating, D loss of fluids		Causes Cellular mvmt ICF→ECF renal failure Excess intake addisons (adrenal insuff.) Drugs (K-sparing diuretic, ace inhibitors, NSAIDS)			
↑P	HYPO calcemia ↓Ca <8.5 "cramps"	=	HYPO magnesemia ↓Mg <1.5 "twitching" (neuro excitability)		
S/S + trousseaus, chvosteks tetany, spasm seizures ↑DTR confusion arrhythmias		S/S + trousseaus, chvosteks tetany Tourdes de pointes cardiac Δs seizures ↑DTR ↑BP ↓RR ↓bowel motility			
Causes Low PTH celiac/crohns low Vit. D Acute pancreatitis chronic kidney issues Inadequate intake (alcohol, bulimia) ↑Phos. Wound drainage (esp GI) meds ↓mobility		Causes "LOW MAG" Limited intake Other electrolyte issues (hypOcalcemia, hypOkalemia) Wasting Mg Malabsorption Alcohol Glycemic issues (DKA, insulin)			
↓P	HYPER calcemia ↓Ca >10 "body is weak"	=	HYPER magnesemia ↑Mg >2.5 "body system is lethargic"		
S/S Muscle weakness, lethargy EKG Δs absent or ↓DTR confused Abdominal distention d/t constipation Ca deposits kidney stone formation		S/S Muscle weakness → resp. arrest EKG Δs → cardiac arrest Absent or ↓DTR N/V ↓BP			
Causes hyperPTH hyperthyroidism ↓excretion (renal failure, thiazides) bone cancer ↑Ca /Vit D intake lithium glucocorticoids (suppress Ca) addisons (adrenal insuff.)		Causes Mg rich antacids/laxatives (Maalox, Mylanta) Addisons (adrenal insuff) Glomerular filtration insuff.			



TAke A BREATH

Acid-Base protocol



Normal pH: 7.35-7.45
 Acidemia: pH < 7.35
 Alkalemia: pH > 7.45
 Normal PaCO₂: 35-45 mm Hg
 Normal HCO₃: 22-26 meq/L
 Anion Gap: Na - (Cl + HCO₃)
 Normal Anion Gap: 8-12
 Excess AG = Observed AG - Normal AG
 Delta HCO₃ = Normal HCO₃ - Measured HCO₃

Diagnosis of Triple Acid-Base Disorders:

Rule 1: Triple acid-base disorders involve metabolic acidosis and metabolic alkalosis with one primary respiratory disorder. Respiratory acidosis and respiratory alkalosis can not co-exist.

Rule 2: For high anion gap acidosis, the serum HCO₃ will fall by 1 meq/L for each meq increase in anion gap. This relationship helps make diagnosis of mixed metabolic acidosis and alkalosis:

Calculate Delta Anion gap

Calculate Delta HCO₃

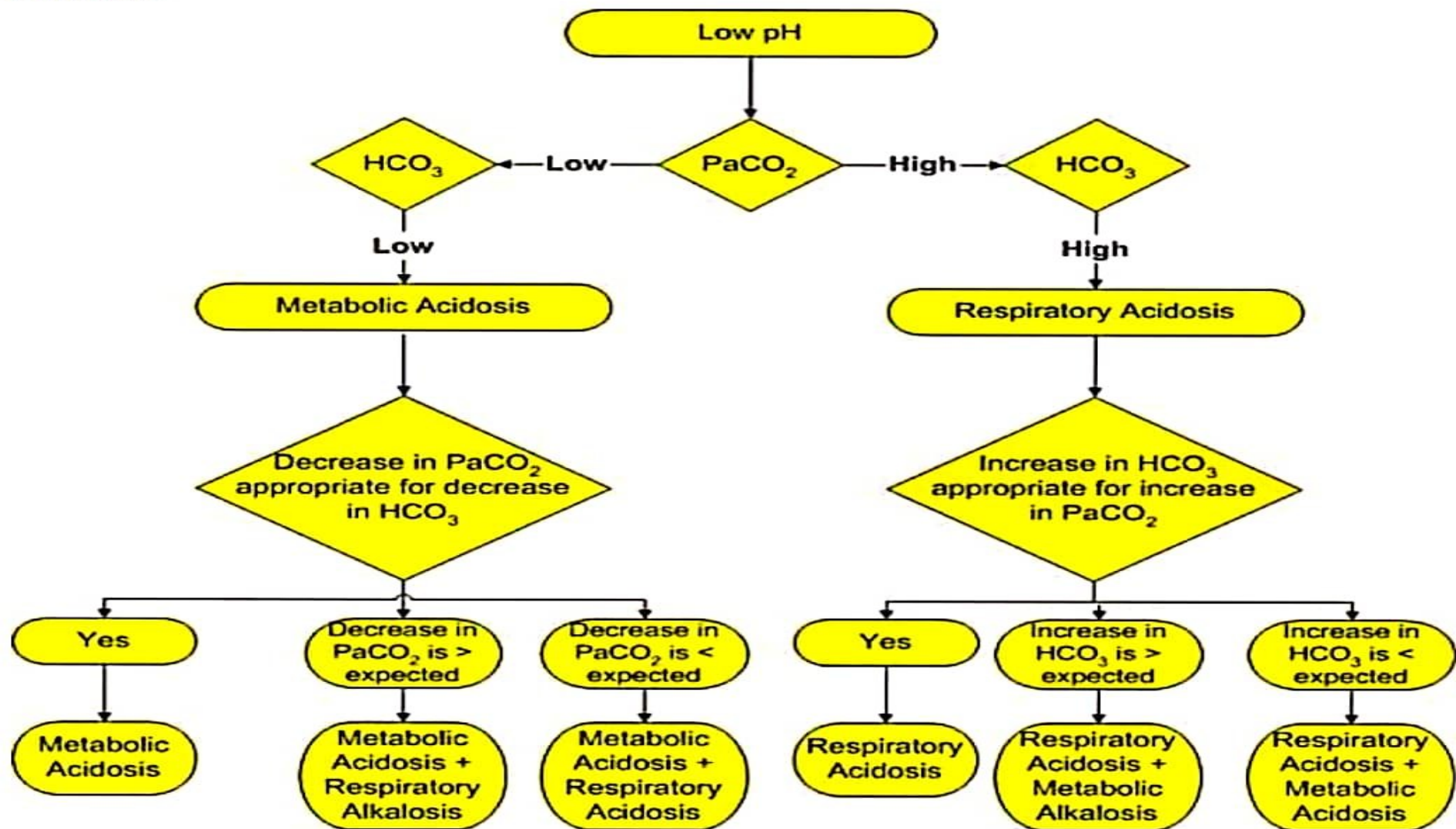
If Excess AG is > Delta HCO₃: Patient has superimposed metabolic alkalosis in addition to primary high AG metabolic acidosis

If Excess AG is < Delta HCO₃, suspect a combined high AG acidosis plus normal AG acidosis.

Rule 3: If AG is > 35, suspect a combined metabolic acidosis plus metabolic alkalosis.

Rule 4: In a patient with metabolic acidosis, if excess AG + Measure HCO₃ is > 30, there is underlying metabolic alkalosis; if < 23, there is associated non AG acidosis.

Acidosis



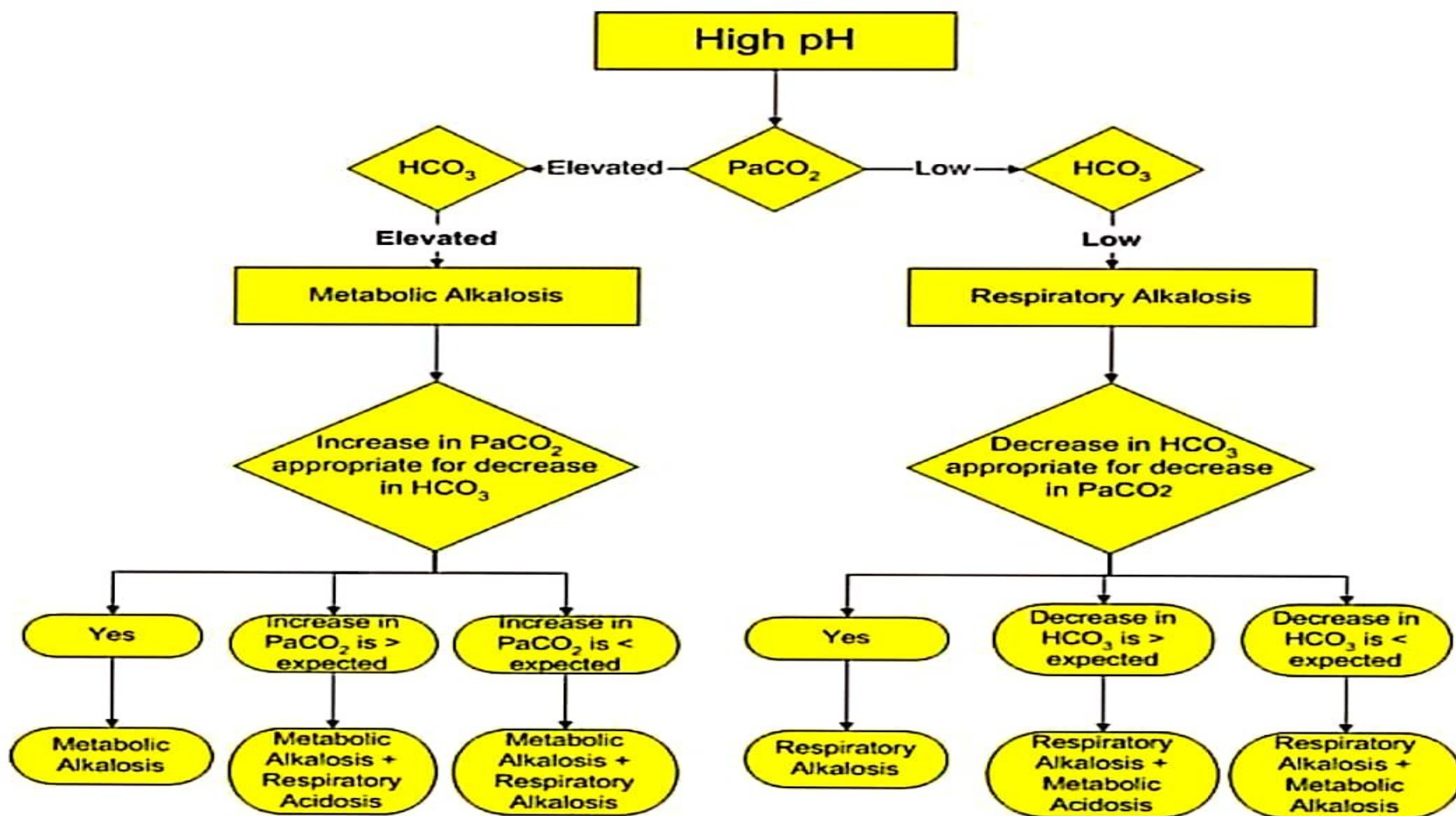
Compensation for Metabolic Acidosis

For each meq/L decrease in HCO₃, PaCO₂ decreases by 1.2
Expected PaCO₂ = $1.5 \times \text{HCO}_3 + 8$ (+/- 2)
PaCO₂ = Last 2 digits of pH

Compensation for Respiratory Acidosis

Acute: For every 10 mmHg increase in PaCO₂, HCO₃ increases by 1 meq/L
Chronic: For every 10 mmHg increase in PaCO₂, HCO₃ increase by 3.5 meq/L

Alkalosis



Compensation for Metabolic Alkalosis

For each meq/L increase in HCO₃, PaCO₂ increases by 0.6 mm Hg

Compensation for Respiratory Alkalosis

Acute: For every 10 mm decrease in PaCO₂, HCO₃ decreases by 2 meq/L
Chronic: For every 10 mm decrease in PaCO₂, HCO₃ decrease by 4-5 meq/L

The

7

*Habits of
Highly
Successful
Blood Gas
Analysts*

ABG Interpretation

Step 1

Look at the pH

Is the patient
or

acidemic
alkalemic

$\text{pH} < 7.35$
 $\text{pH} > 7.45$



< 7.35

Acidosis

> 7.45

Alkalosis

$7.35 - 7.45$

Normal
Mixed
disorder

• Step 2

- *Is it a metabolic or respiratory disturbance ?*
- Acidemia: With $\text{HCO}_3^- < 20 \text{ mmol/L}$ = metabolic
- With $\text{PCO}_2 > 45 \text{ mm Hg}$ = respiratory
- Alkalemia: With $\text{HCO}_3^- > 28 \text{ mmol/L}$ = metabolic
- With $\text{PCO}_2 < 35 \text{ mm Hg}$ = respiratory



Step 3

If there is a primary respiratory disturbance, is it acute?

Expect $\Delta \text{pH} = 0.08 \times \Delta \text{PCO}_2 / 10$

• Step 4

- ***For a respiratory disorder is renal compensation OK?***
- Respiratory acidosis: <24 hrs: $\Delta [\text{HCO}_3] = 1/10 \Delta \text{PCO}_2$
- >24 hrs: $\Delta [\text{HCO}_3] = 3/10 \Delta \text{PCO}_2$
- Respiratory alkalosis: 1- 2 hrs: $\Delta [\text{HCO}_3] = 2/10 \Delta \text{PCO}_2$
- >2 days: $\Delta [\text{HCO}_3] = 6/10 \Delta \text{PCO}_2$

Primary disorder	Primary defect	Compensatory response
Respiratory acidosis	$\uparrow$ PCO ₂	$\uparrow$ HCO ₃
Respiratory Alkalosis	$\downarrow$ PCO ₂	$\downarrow$ HCO ₃

- **Step 5**

- **If the disturbance is metabolic is the respiratory compensation appropriate?**

- For metabolic acidosis:

- Expect $\text{PCO}_2 = (1.5 \times [\text{HCO}_3]) + 8 \pm 2$

- (Winter's equation)

- For metabolic alkalosis:

- Expect $\text{PCO}_2 = (0.7 \times [\text{HCO}_3]) + 21 \pm 1.5$

- If not:

- actual $\text{PCO}_2 > \text{expected}$: hidden respiratory acidosis

- actual $\text{PCO}_2 < \text{expected}$: hidden respiratory alkalosis

Primary disorder	Primary defect	Compensatory response
Metabolic Acidosis	$\downarrow \text{HCO}_3^-$	$\downarrow \text{PCO}_2$
Metabolic alkalosis	$\uparrow \text{HCO}_3^-$	$\uparrow \text{PCO}_2$

- Remember.....

- ✓ *Respiratory compensation*

*is always **FAST** ...12-24 hrs*

- ✓ *Metabolic compensation*

- *is always **SLOW** ...5 -7 days*



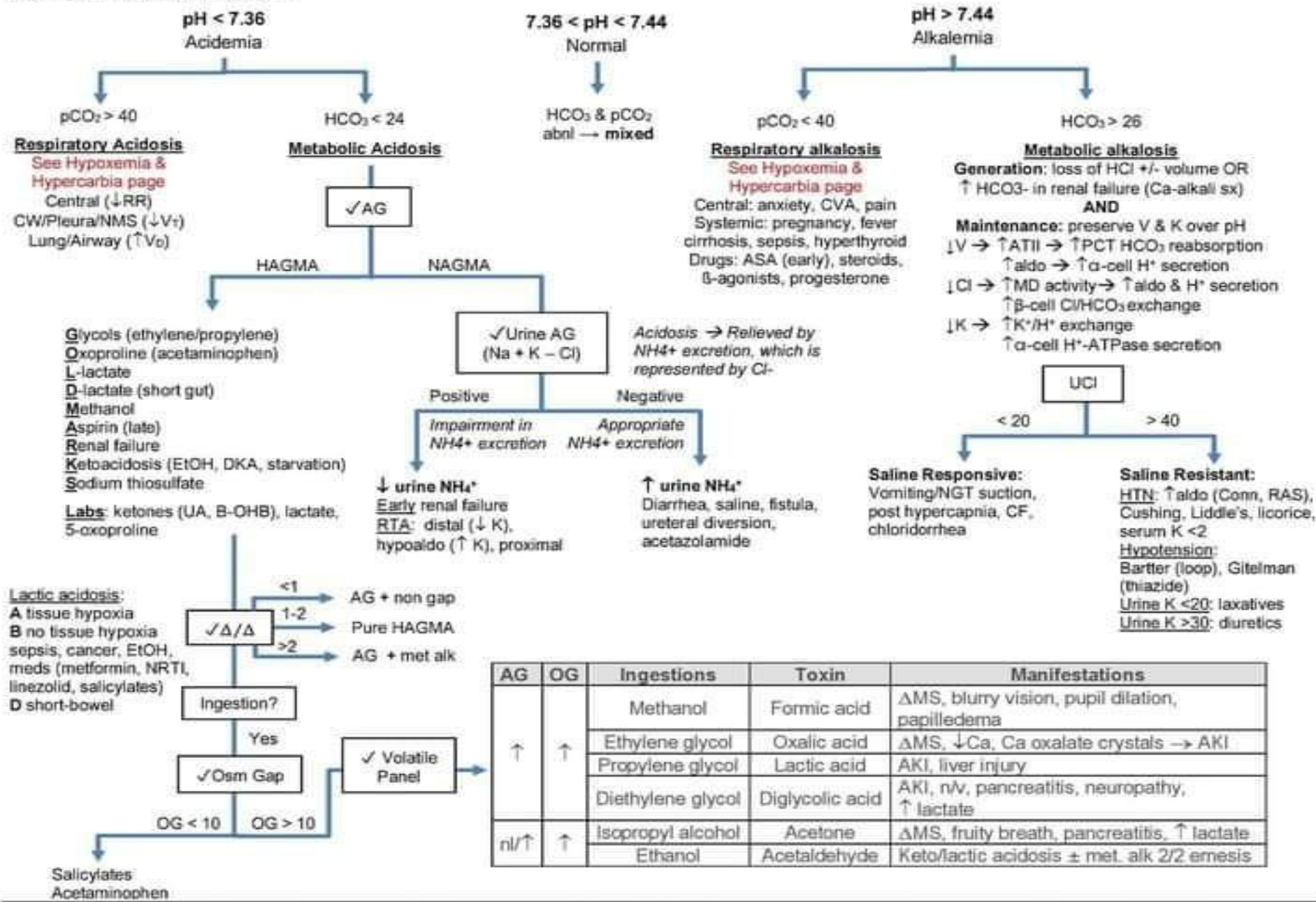
- Step 6

- *If there is metabolic acidosis, is there an anion gap?*
- $\text{Na} - (\text{Cl} + \text{HCO}_3^-) = \text{Anion Gap}$ usually <12
- **Normal AG** - (loss of HCO_3^- , increase in chloride) – Diarrhoea, RTA, carbonic anhydrase inhibitor use.
- **High AG** - If >12 , **Anion Gap Acidosis** : Methanol
- (Decreased excretion of acids) Uremia
- Diabetic Ketoacidosis
- Paraldehyde
- Infection (lactic acid)
- Ethylene Glycol
- Salicylate

• Step 7

- *Does the anion gap explain the change in bicarbonate?*
- *(to rule out co-existence of 2 acid-base disorders)*
- Δ anion gap (Anion gap -12) Delta Gap
- Delta Gap + $[\text{HCO}_3^-] = 22-26 \text{ mmols/l}$
- If Delta anion gap is greater(>26); consider additional metabolic alkalosis
- If Δ anion gap is less(<22); consider additional nonanion gap metabolic acidosis

ALGORITHMIC APPROACH



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Thank you

