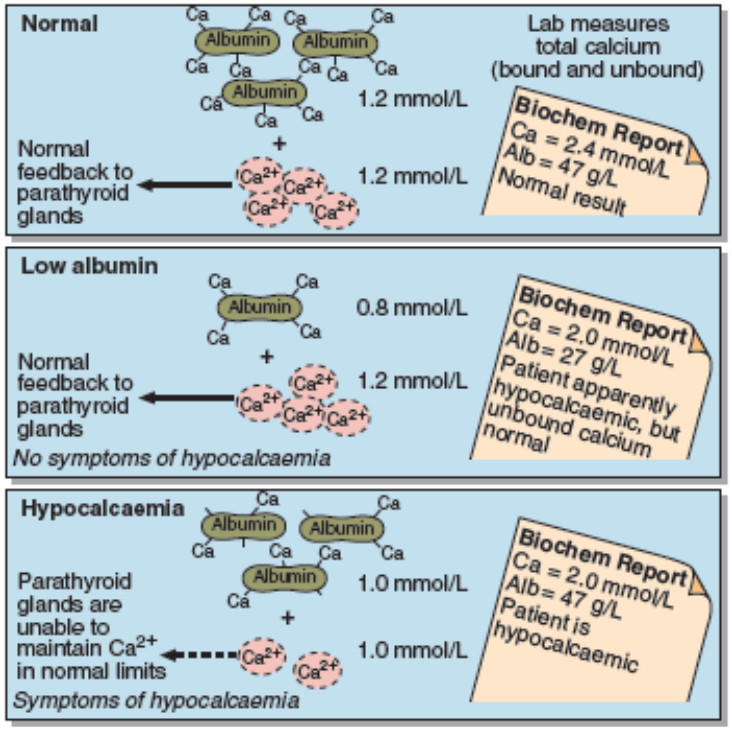
Regulation of Parathyroid gland Calcium, Magnesium and Phosphate

Calcium

- The ionized/free calcium is essential for myocardial contraction whereas protein bound and citrate-bound calcium had no effect
- It is important to maintain ionized calcium at a near normal concentration during surgery and in critically ill patients.
- Decreased ionized Ca conc. in blood can cause neuromuscular irritability which may become clinically apparer as irregular muscle spasms, called tetany.



Regulation of Calcium

- Three hormones, PTH, vitamin D, and calcitonin, are known to regulate serum calcium by altering their secretion rate in response to changes in ionized calcium

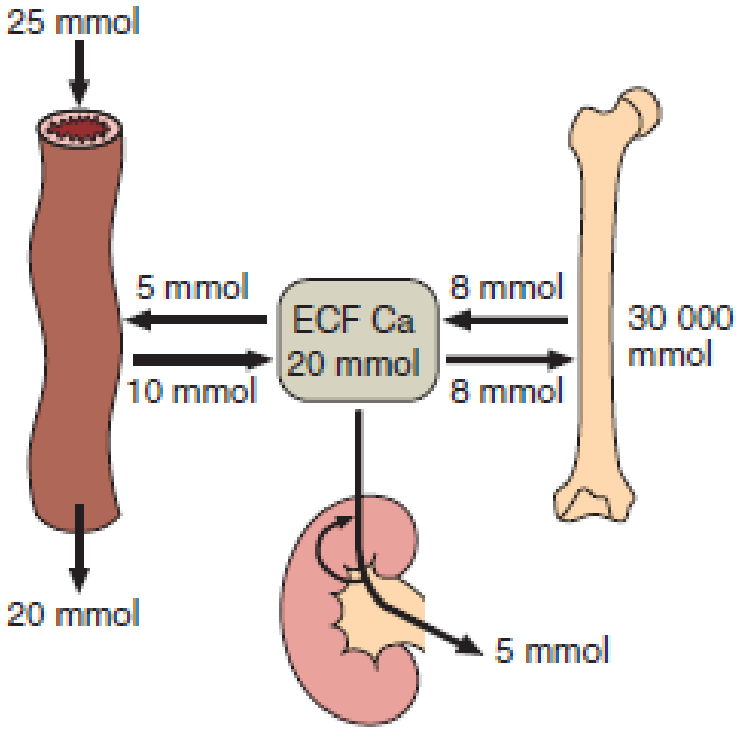
- PTH secretion in blood is stimulated by a decrease in ionized calcium and, conversely PTH secretion is stopped by an increase in ionized calcium.

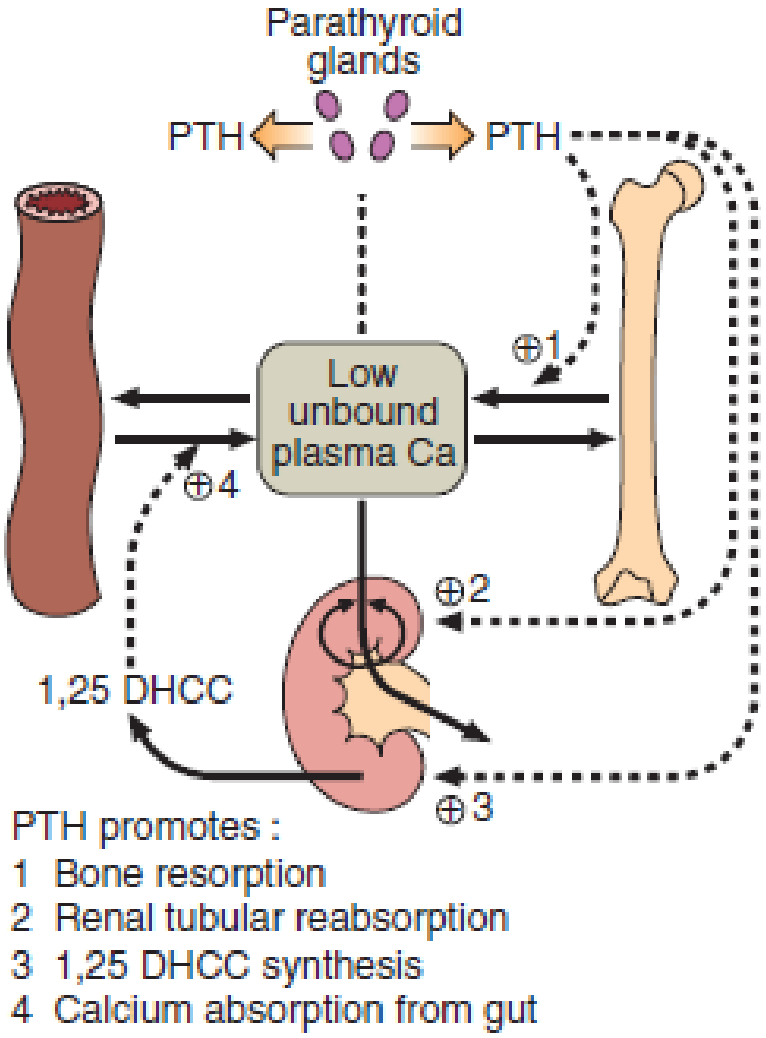
- PTH exerts three major effects on both:
  - Bone: activates bone resorption, break down osteoclast and release Ca to ECF
  - Kidney: conserves calcium by increasing tubular reabsorption of calcium ions and stimulates renal production of active vitamin D.

Regulation of Calcium

- Vitamin D3, a cholecalciferol, is obtained from the diet or exposure of skin to sunlight, hydroxylated in liver to 25-OH- (inactive form), activated in the kidney by 1-α-hydroxylase to form (1,25-OH)2-D3, the biologically active form which will:
  - Increase calcium absorption in the intestine and
  - Enhance the effect of PTH on bone resorption.
- Calcitonin, which originates in the medullary cells of the thyroid gland, is secreted when the concentration of Ca in blood increases (not at normal levels)
- Calcitonin reduces Ca by inhibiting the actions of both PTH and vitamin D

Regulation of calcium





Distribution

- About 99% of calcium in the body is part of bone.
- The remaining 1% is mostly in the blood and other ECF. Little is in the cytosol of most cells.
- The concentration of ionized calcium in blood is 5,000-10,000 times higher than in the cytosol of cardiac or smooth muscle cells.
- Maintenance of this large gradient is vital to maintain the essential rapid inward flux of calcium ions
- Calcium in blood: 45% circulates as free calcium ions (ionized calcium), 40% is bound to albumin, and 15% is bound to anions (bicarbonate, citrate, phosphate, and lactate)
- Distribution can change in disease as conc. Of citrate, bicarbonate, lactate, phosphate and albumin can change dramatically during surgery or critical care. This calcium cannot be reliably calculated from total calcium measurement (acutely ill patients)

Causes of hypocalcemia

- Primary hypoparathyroidism-glandular aplasia, destruction, or removal
- Hypomagnesemia (in hospitalized patients)
- Hypermagnesemia causes hypocalcemia by three mechanisms:
  - it inhibits the glandular secretion of PTH across the membrane parathyroid gland
  - it impairs PTH action at its receptor site on bone
  - It causes vitamin D resistance “elevated Mg levels may inhibit PTH release and target tissue response. Perhaps leading to hypocalcemia and hypercalciuria
- Hypoalbuminemia (total calcium only, ionized not affected): chronic liver disease, nephrotic syndrome, malnutrition: for each 1g/dL decrease in serum albumin, there is a 0.2 mmol/L (0.8 mg/dL) decrease in total calcium levels

Causes of hypocalcemia

- **Acute pancreatitis:** the cause appears to be a result of increased intestinal biding of calcium as increased intestinal lipase activity occurs

- **Vitamin D deficiency** and **malabsorption** can cause decreased absorption, which leads to increased PTH production or secondary hyperparathyroidism.

- **Renal disease** (Altered concentrations of calcium, phosphate, albumin, magnesium and hydrogen ion (pH):
  - In chronic renal disease, secondary hyperparathyroidism frequently develops as the body tries to compensate for hypocalcemia caused either by hyperphosphatemia (phosphate binds and lowers ionized calcium) or altered vitamin D metabolism

Causes of hypocalcemia

- **Pseudohypoparathyroidism:** a rare hereditary disorder in which PTH target tissue response is decreased (end organ resistance)

- PTH production responds normally to loss of calcium, however, without normal response (decreased cAMP(Adenosine 3’,5’-cyclic phosphate) production), calcium is lost in the urine or remains in the bone storage pool

- Patients often have common physical features, including short stature, obesity. Shortened metacarpals and metatarsals, and abnormal calcification.

- **Rhabdomyolysis:** as with major crush injury and muscle damage, may cause hypocalcemia as a result of increased phosphate release from the cells, which binds to calcium ions.

Surgical and intensive care

- Controlling calcium concentrations may be critical in **open heart surgery** when the heart is restarted and during **liver transplantation** because large volumes of citrated blood are given.

- Ionized Ca measurements are the measurement of greatest clinical value.

- Hypocalcemia occurs commonly in critically ill patients, that is, those with sepsis, thermal burns, renal failure, or cardiopulmonary insufficiency (abnormalities of acid-base regulation and losses of protein and albumin)

Neonatal monitoring

- Typically blood-ionized calcium concentrations in neonates are high at birth and then rapidly decline by 10-20% after 1-3 days. After about 1 week, ionized calcium concentrations in the neonate stabilize at levels slightly higher than in adults.

- The concentration of ionized calcium may decrease rapidly in the early neonatal period because the infant may lose calcium rapidly and not readily reabsorb it.

- Several possible etiologies have been suggested: abnormal PTH and vitamin D metabolism, hypercholesterolemia, hyperphosphatemia, and hypomagnesemia.

Symptoms of hypocalcemia

- Neuromuscular irritability and cardiac irregularities are the primary groups of symptoms that occur with hypocalcemia.
- Neuromuscular symptoms include parasethesia, muscle cramps, tetany, and seizures.
- Cardiac symptoms may include arrhythmia or heart block.
- Symptoms usually occur with severe hypocalcemia, in which total calcium levels are below 1.88 mmol/L (7.5 mg/dL).

Treatment of hypocalcemia

- Oral or parenteral calcium therapy may occur, depending on the severity of the decreased level and the cause.
- Vitamin D may sometimes be administered in addition to oral calcium to increase absorption.
- If hypomagnesemia is a concurrent disorder, magnesium therapy should also be provided

Hypercalcemia

- Although either total or ionized calcium measurements serious cases, ionized calcium is more frequently elevated in asymptomatic hyperparathyroidism.

- In general, ionized calcium measurement elevated in 90-95% of cases of hyperparathyroidism whereas total calcium is elevated in 80-85% of the cases

Causes of hypercalcemia

- Primary hyperparathyroidism (in old women)-adenoma or glandular hyperplasia
- hyperthyroidism, because of the proximity of the parathyroid gland to the thyroid gland, hyperthyroidism can sometimes cause hyperparathyroidism.
- Benign familial hypocalciuria
- Malignancy:
  - Ca is sole biochemical marker for many tumors.
  - Many tumors produce PTH-related peptide (PTH-rP), which binds to normal PTH receptors and causes increased calcium levels
  - Assays to measure PTH-rP are available because this abnormal protein is not detected by most PTH assays

Causes of hypercalcemia

- **Thiazide diuretics** increase calcium reabsorption
- **Prolonged immobilization** may cause increased bone resorption. This cause is further compounded by renal insufficiency
- **Multiple myeloma**
- **Increased vitamin D**

Symptoms of hypercalcemia

- A mild hypercalcemia (2.62-3.00 mmol/L) is often asymptomatic.
- Moderate or severe calcium elevations include:
  - Neurologic symptoms: mild drowsiness or weakness, lethargy, and coma depression,
  - GI: constipation, nausea, vomiting, anorexia, and peptic ulcer disease.
  - Hypercalcemia may cause renal symptoms of nephrolithiasis and nephrocalcinosis
- Hypercalciuria can result in nephrogenic diabetes insipidus, which causes polyuria and results in hypovolemia, which further aggravates the hypercalcemia.
- Hypercalcemia can also cause symptoms of digitalis toxicity.

Treatment of hypercalcemia

- Depends on the level of hypercalcemia and the cause.
- Often people with primary hyperparathyroidism are asymptomatic. Postmenopausal women with primary hyperparathyroidism may have estrogen deficiency
- **Parathyroidectomy** may be necessary in some hyperparathyroidic patients
- Often, **estrogen replacement therapy** reduces calcium levels.
- Patients with moderate to severe hypercalcemia are treated by:
  - Encouraging salt and water intake to increase calcium excretion and avoid dehydration
  - Thiazide diuretics should be discontinued.
  - Biphosphanate (a derivative of pyrophosphate) are the main drug class used to lower calcium levels by preventing bone resorption, achieved by its binding action to bone.

Determination of Calcium

- For total calcium determinations is either **serum or lithium heparin** plasma collected without venous stasis.
- Anticoagulant such as **EDTA or oxalate** bind calcium tightly and interfere with the measurement, so, their use should be **avoided**
- The proper collection of samples for ionized calcium measurements requires greater care. Because loss of CO2 will increase **pH**, samples must be collected anaerobically.
- Although heparinized whole blood is the preferred sample, serum from sealed evacuated blood collection tubes may be used if clotting and centrifugation are done quickly (<30 minutes) and at room temperature.

Determination of Calcium

- No liquid heparin products should be used. Most heparin anticoagulants (sodium, lithium) partially bind to calcium and lower ionized calcium concentrations

- Dry heparin products are available titrated with small amounts of Ca or Zn ions that essentially eliminates the interference by heparin.

- For analysis of calcium in urine, an accurately timed urine collection is preferred, acidified with approximately 1 mI of HCl (6M) for each 100 mI of urine

Method

- The two commonly used methods for total calcium analysis are:
- Use of ortho-cresolphthalein complexone (CPC) or arsenzo III dye to form a complex with calcium.
  - Prior to the dye binding reaction, calcium is released from its protein carrier and complexes by acidification of the sample
  - The CPC method uses 8- hydroxyquinoline to prevent magnesium interference
- Use ISEs:
  - Use membranes impregnated with special molecules that selectively, but reversibly, bind calcium ion
  - As calcium ions bind to these membranes, an electric potential develops across the membrane that is proportional to the ionized calcium concentration.
- AAS remains the reference method for total calcium

Reference Ranges

TOTAL CALCIUM (SERUM, PLASMA)	
Child	2.20–2.70 mmol/L (8.8–10.8 mg/dL)
Adult	2.15–2.50 mmol/L (8.6–10.0 mg/dL)
IONIZED CALCIUM (SERUM)	
Neonate	1.20–1.48 mmol/L (4.8–5.9 mg/dL)
Child	1.20–1.38 mmol/L (4.8–5.5 mg/dL)
Adult	1.16–1.32 mmol/L (4.6–5.3 mg/dL)
Urine (24-hour)	2.50–7.50 mmol/day (100–300 mg/day), varies with diet

Magnesium

- The average human body (70 kg) contains 1 mole (distributed as 24 g) of magnesium
  - 53% in bone
  - 46% in muscle and other organs and soft tissue
  - less than 1% in serum and RBC's (1/3 bound to albumin)

- Similar to calcium, it is the free ion that is physiologically active in the body

- The role magnesium in the body is:
  - It is an essential cofactor of more than 300 enzymes
  - The most significant findings are the relationship between abnormal serum magnesium levels and cardiovascular, metabolic, and neuromuscular disorders. Although serum levels may not reflect total body stores of Mg, serum level is useful in determining acute changes in the ion

Regulation

- Rich sources of Mg in the diet: raw nuts, dry cereal, and “hard” drinking water.
- Other sources include vegetables, meats, fish, and fruit
- Processed foods have low levels of magnesium that may cause an inadequate intake
- The small intestine may absorb 20-65% of the dietary Mg, depending on the need and intake.
- The overall regulation of body magnesium is controlled largely by the kidney which can reabsorb magnesium in deficiency states or readily excrete excess magnesium in overload states.
- Henle loop is the major renal regulatory site, where 50-60% of filtered Mg is reabsorbed in the ascending limb

Regulation

- The renal threshold for magnesium is about 0.60-0.85 mmol/L (close to normal serum conc.), so slight excesses of magnesium in serum are rapidly excreted by the kidneys
- Normally only about 6% of filtered Mg is excreted in the urine per day
- Magnesium regulation appears to be related to that of calcium and sodium
- Parathyroid hormone (PTH) increases the renal reabsorption of magnesium and enhances the absorption of magnesium in the intestine.
- Changes in ionized calcium have a far greater effect on PTH secretion.
- Aldosterone and thyroxine apparently have the opposite effect of PTH in the kidney, increasing the renal excretion of magnesium.

Hypomagnesemia

- Most frequently observed in hospitalized individuals in intensive care units of those receiving diuretic therapy or digitalis therapy
- Hypomagnesemia is rare in nonhospitalized individuals
- There are many causes of hypomagnesemia
  - Reduced intake Poor diet/starvation, Prolonged magnesium-deficient IV therapy, chronic alcoholism
  - Decreased absorption: due to GI disorders as malabsorption syndrome, surgical resection of the small intestine, nasogastric suction, pancreatitis, prolonged vomiting, diarrhea, laxative abuse, neonatal (due to surgical procedure), primary (due to selective malabsorption of the ion), congenital (autosomal recessive disorder)
    - A chronic congenital hypomagnesemia with secondary hypocalcemia occurs due to specific transport protein defect in the intestine

Causes of hypomagnesemia

- Increased Excretion
  - **Renal:** Tubular disorder, Glomerulonephritis, Pyelonephritis
  - **Endocrine:** Hyperparathyroidism (increased calcium) (Hyperaldosteronism (increase of Mg excretion and water retention (pseudohypomagnesemia), hyperthyroidism (increase excretion cause intracellular shift of ions), hypercalcemia, diabetic ketoacidosis (increase renal loss due to glycosuria)
  - **Drug induced** (increase renal loss of Mg): diuretics, antibiotics (gentamicin), cyclosporine, and cisplatin (nephrotoxic), digitalis (interfere with Mg reabsorption)
- **Miscellaneous**: Excess lactation (loss in milk)), Pregnancy (may cause a hyperexcitable uterus, anxiety and insomnia)

Symptoms of hypomagnesemia

- A patient who is hypomagnesemic may be asymptomatic until serum levels fall below 0.5 mmol/L.
- A variety of symptoms can occur. The most frequent involve cardiovascular, neuromuscular, psychiatric, and metabolic abnormalities

Cardiovascular	**Psychiatric**
Arrhythmia	Depression
Hypertension	Agitation
Digitalis toxicity	Psychosis
Neuromuscular	**Metabolic**
Weakness	Hypokalemia
Cramps	Hypocalcemia
Ataxia	Hypophosphatemia
Tremor	Hyponatremia
Seizure	
Tetany	
Paralysis	
Coma	

Symptoms of hypomagnesemia

- Cardiac:
  - Symptoms result primarily from the ATPase enzyme's Mg. requirement for Mg
  - Mg loss leads to decreased intracellular K levels because of a faulty NaK pump (ATPase)
  - This change in cellular RMP causes increased excitability that may lead to cardiac arrhythmia and digitalis toxicity

- Normal nerve and muscle cell stimulation:
  - Requires magnesium and ATPase for normal calcium uptake following contraction
  - Requires magnesium to assist with the regulation of acetylcholine, a potent neurotransmitter

- Metabolic disorders:
  - Mg deficiency can impair PTH release and target tissue response, resulting in hypocalcemia. Mg therapy alone may restore both ions levels to normal
  - Serum levels of the ions must be monitored during treatment.

Treatment of hypomagnesemia

- The preferred treatment of hypomagnesemia by oral intake is Mg-lactate, Mg oxide, MgCl or an antacid that contains Mg.

- In severely ill patients, a $MgSO_4$ solution is given parenterally

- Before initiation of therapy; renal function must be evaluated to avoid inducing hypermagnesemia during treatment

Hypermagnesemia and its causes

- less frequently than hypomagnesemia
- The most common cause is **renal failure** (GFR <30 severe elevations are usually a result of the combine effects of decreased renal function and increased intake of commonly prescribed magnesium-containing medication, such as antacid, enemas, or cathartics.
- Nursing home patients are at greatest risk for this occurrence.
- **Decreased excretion**: acute or chronic renal failure, hypothyroidism, hypoaldosteronism, hypopituitarism (IGH)
- **Increased intake**: Antacids, enemas, cathartics, therapeutic-eclampsia, cardiac arrhythmia
- **Miscellaneous:** dehydration (pseudohypermagnesemia, corrected by dehydration), bone carcinoma, bone metastases (high Mg due to bone loss)

Causes of hypomagnesemia

- **Endocrine disorders:** Thyroxine and growth hormone cause a decrease in tubular reabsorption of Mg and of either hormone may cause a moderate elevation in serum Mg.
- **Adrenal insufficiency** may cause a mild elevation as a result of decreased renal excretion of Mg
- MgSO4 may be used therapeutically with preeclampsia, cardiac arrhythmia, or myocardial infarction
- Mg is a vasodilator, and can decrease uterine hyperactivity in eclampsic states and increase uterine blood flow (maternal hypermagnesemia)
- Neonatal hypermagnesemia due to the immature kidney of the newborn (Premature infants are at great risk)

Symptoms of hypermagnesemia

- Hypermagnesemia typically do not occur until the serum level exceeds 1.5 mmol/L.
- Most frequent symptoms involve cardiovascular, dermatological, GI, neurologic, neuromuscular, metabolic, and hemostatic abnormalities.
- Mild to moderate symptoms may occur when serum levels are 1.5-2.5mmol/L: hypotension, bradycardia, skin flushing, increased skin temperature, nausea, vomiting, and lethargy
- Life-threatening symptoms, such as ECG changes, heart block, asystole, sedation, coma, respiratory depression or arrestand paralysis, can occur when serum levels reach 5.0 mmol/L

Symptoms of hypermagnesemia

- Elevated Mg levels may inhibit PTH release and target tissue response. This may lead to hypocalcemia and hypercalciuria

- Normal hemostasis is a calcium-dependent process that may be inhibited as a result of competition between increased levels of magnesium and calcium ions. Thrombin generation and platelet adhesion are two processes in which interference may occur.

Treatment of hypermagnesemia

- If Mg excess associated with increased intake one should discontinue the source of Mg.
- Severe symptomatic hypermagnesemia requires immediate supportive therapy for cardiac, neuromuscular, respiratory or neurologic abnormalities.
- Patients with renal failure require hemodialysis.
- Patients with normal renal function may be treated with a diuretic and IV fluids

Determination of magnesium

- **Specimen:**
- Nonhemolyzed serum or lithium heparin plasma may be analyzed.
- Mg is intracellular cation so hemolysis should be avoided and the serum should be separated from the cells as soon as possible
- Oxalate, citrate, and EDTA anticoagulants are unacceptable because they will bind with magnesium.
- A 24-hour urine is preferred for analysis and must be acidified with HCl to avoid precipitation

Method

- The three most common methods for measuring total serum Mg are colorimetric:
  - Calmagite: Mg binds with calmagite to form a reddish-violet complex that is read at 532 nm
  - Formazen dye: Mg binds with the dye to form a colored complex that is read at 660 nm
  - Methylthymol blue: Mg binds with the chromogen to form a colored complex
- Most methods use a calcium shelter to prohibit interference from Ca.
- The reference method for measuring magnesium is AAS.

Limitations of Mg determination

- Although the measurement of total Mg conc in serum remains the usual diagnostic test for the detection of magnesium abnormalities, it has limitations:

- Because approximately 25% of magnesium is protein bound, total magnesium may not reflect the physiologically active free ionized magnesium.

- Because magnesium is primarily an intracellular ion, serum concentration will not necessarily reflect the status of intracellular magnesium (depletion of 20% of cellular Mg, serum magnesium concentrations may remain normal)

TABLE 13-15. REFERENCE RANGE FOR MAGNESIUM

Serum, plasma	0.63–1.0 mmol/L (1.2–2.1 mEq/L)

Phosphate

- Found everywhere in living cells: DNA, RNA, in most coenzymes
- The most important reservoirs of biochemical energy are ATP, creatine, phosphate, and phosphoenolpyruvate.
- Phosphate deficiency can lead to ATP depletion, which is ultimately responsible for many of the clinical symptoms observed
- Alteration in the concentration of 2,3-bisphosphoglycerate (2,3-BPG) in red blood cells affect the affinity of hemoglobin for oxygen, the concentration of inorganic phosphate indirectly affects the release of oxygen from hemoglobin
- Transcellular shifts of phosphate are a major cause of hypophosphatemia in blood. Once phosphate is taken up by the cell, it will be used in the synthesis of phosphorylated compounds. As these phosphate compounds are metabolized, Pi slowly leaks into the blood, where it is regulated principally by the kidney

Regulation

- Phosphate in blood may be absorbed in the intestine from dietry sources, released from cells into blood and lost from bones . In healthy individuals, all these processes are relatively constant and easily regulated by renal excretion or reabsorption of phosphate

- Many factors can alter phosphate concentrations in the blood:
  - The loss of regulation by the kidneys will have the most profound effect
  - The most important factor is PTH, which overall lowers blood conc. By increasing renal excretion
  - Vitamin D acts to increase phosphate in the blood by increasing both phosphate absorption in the intestine and phosphate reabsorption in the kidney.
  - Growth hormone, which helps regulate skeletal growth, can affect circulating concentration of phosphate
  - Excessive secretion or administration of growth hormone, phosphate concentrations in the blood may increase because of decreased renal excretion of phosphate.
  - Calcitonin, acid-base status, can also affect renal regulation of Phosphate

Distribution

- Although the concentration of all phosphate compounds in blood is about 12 mg/dl (3.9 mmol/L), only about 3-4 mg/dL is inorganic phosphate.

- Phosphate is the predominant intracellular anion, with variable concentrations depending on the type of cell.

- About 80% of the total body pool of phosphate is contained in bone, 20% in soft tissues, and less than 1% is active in the serum/plasma.

Hypophosphatemia

- Hypophosphatemia occurs in about 1-5% of hospitalized patients.
- The incidence of hypophosphatemia increases to 20-40% in patients with:
- diabetic ketoacidosis, chronic obstructive pulmonary disease (COPD), asthma, malignancy, long-term treatment with total parenteral nutrition (TPN), inflammatory bowel disease, anorexia nervosa, and alcoholism.
- The incidence increases to 60-80% in:
  - ICU patients with sepsis.
  - increased renal excretion
  - Hyperparathyroidisim
  - Decreased intestinal absorption
  - vitamin D deficiency
  - Antacid use
- Although most cases are moderate and seldom cause problemsm severe hypophosphartemia (<1mg/dl or 0.3 mmol/L) requires monitoring and possible replacement therapy

Hyperphosphatemia

- Patients at greatest risk for hyperphosphatemia are those with acute or chronic renal failure
- An increased intake of phosphate or increased release of cellular phosphate may also cuse hyperphosphatemia
- Neonates: not developed mature PTH and vitamin D metabolism, hyperphosphatemia is caused by increased intake such as from cow's milk or laxatives.
- Increased breakdown of the cells as with severe infections, intensive exercise, neoplastic disorders, or intravascular hemolysis
- Because immature lymphoblasts have about 4 times the phosphate content of mature lymphocytes, patients with lymphoplastic leukemia are especially susceptible to hyperphosphatemia.

Determination of inorganic phosphorus

- Specimen. Serum or lithium heparin plasma is acceptable for analysis.
- Oxalate, citrate, or EDTA anticoagulants should not be used because they interfere with the analytic method.
- Hemolysis should be avoided because of the higher concentrations inside the red cells
- Circulating phosphate levels are subject to circadian rhythm, with highest levels in late morning and lowest in the evening. Urine analysis for phosphate requires a 24-hour sample collection because of significant diurnal variations.

Methods and reference ranges

- Phosphorus determination methods involve the formation of an ammonium phosphomolybdate complex. This colorless complex can be measured by ultraviolet absorption at 340 nm or can be reduced to form molybdenum blue, a stable blue chromophore, which is read between 600 and 700 nm.

- Normal ranges:

SERUM, PLASMA	
Neonate	1.45–2.91 mmol/L (4.5–9.0 mg/dL)
Child	1.45–1.78 mmol/L (4.5–5.5 mg/dL)
Adult	0.87–1.45 mmol/L (2.7–4.5 mg/dL)
Urine (24-hour)	13–42 mmol/day (0.4–1.3 g/day)

CASE STUDY 13-2

A 60-year-old man entered the emergency department after 2 days of "not feeling so well." History revealed a myocardial infarction 5 years ago, when he was prescribed digoxin. Two years ago, he was prescribed a diuretic after periodic bouts of edema. An electrocardiogram at time of admission indicated a cardiac arrhythmia. Admitting lab results are shown in Case Study Table 13-2.1.

Questions

1. Because the digoxin level is within the therapeutic range, what may be the cause for the arrhythmia?
2. What is the most likely cause for the hypomagnesemia?
3. What is the most likely cause for the decreased potassium and ionized calcium levels?
4. What type of treatment would be helpful?

CASE STUDY TABLE 13-2.1. LABORATORY RESULTS

Venous Blood
Digoxin: 1.4 ng/mL, therapeutic 0.5–2.2 (1.8 nmol/L, therapeutic 0.6–2.8)
Na^{+}: 137 mmol/L
K^{+}: 2.5 mmol/L
Cl^{-}: 100 mmol/L
HCO^{-}: 25 mmol/L
Mg^{+2}: 0.4 mmol/L
Ion/free Ca^{+2}: 1.0 mmol/L

CASE STUDY 13-3

An 84-year-old nursing home resident was seen in the emergency department with the following symptoms: nausea, vomiting, decreased respiration, hypotension, and low pulse rate (46). Physical exam showed the skin was warm to the touch and flushed. Admission lab data are found in Case Study Table 13-3.1.

Questions

1. What is the most likely cause for the patient's symptoms?
2. What is the most likely cause for the hypermagnesemia?
3. What could be the cause for the hypocalcemia?

CASE STUDY TABLE 13-3.1. LABORATORY RESULTS

		RESULT	REFERENCE RANGE
Serum	Total protein	5.6 g/dL	6.0–8.0 g/dL
	Albumin	3.0 g/dL	3.5–5.0 g/dL
	Total calcium	8.2 g/dL	8.6–10.0 g/dL
	BUN	45 mg/dL	5–20 mg/dL
	Creatinine	2.3 mg/dL	0.7–1.5 mg/dL
	Magnesium	4.0 mmol/L	0.63–1.0 mmol/L
Plasma	Na^+	129 mmol/L	136–145 mmol/L
	K^+	5.3 mmol/L	3.4–5.0 mmol/L
	Cl^-	96 mmol/L	
	HCO_3^-	16 mmol/L	

CASE STUDY 13-4

Consider the following laboratory results from three adult patients:

Questions

1. Which set of laboratory results (Case A, B, or C) is most likely associated with each of the following diagnoses:
   - Primary hyperparathyroidism
   - Malignancy
   - Hypomagnesemic hypocalcemia

CASE STUDY TABLE 13-4.1. LABORATORY RESULTS

	REFERENCE RANGES				
CASE	ION Ca^{+2} 1.16–1.32 mmol/L	TOTAL Mg^{+2} 0.63–1.0 mmol/L	PO_4^- 0.87–1.45 mmol/L	HEMATOCRIT 35–45%	INTACT PARATHYROID HORMONE 13–64 ng/L
A	1.44	0.90	0.85	42	100
B	1.08	0.50	0.90	40	25
C	1.70	0.98	1.43	30	12