Phosphorus: A Quick Reference

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MAIN FUNCTIONS OF PHOSPHORUS
The main functions of phosphorus in the body are:

- To provide along with calcium the structural integrity of bones and teeth
- To supply energy in the form of adenosine triphosphate and guanosine triphosphate
- To help in the maintenance of cell membrane structure.

Distribution of phosphorus in the body is:

Inorganic (Pi)
- 85% in the inorganic matrix of bone
- 14% to 15% intracellular
- Less than 1% in the extracellular fluid and serum
  - 10% to 20% is bound to protein
  - The remainder circulates as free anion or is complexed to sodium, magnesium, or calcium

Organic
- The majority (two-thirds) is in the form of phospholipids

In its regulation, phosphorus is:

- Under the influence of parathyroid hormone, calcitriol, and calcitonin
- Absorbed from the small intestine (primarily duodenum)
  - Intestinal phosphorus absorption is increased with calcitriol
  - Intestinal phosphorus absorption is decreased with glucocorticoids, increased dietary magnesium, and hypothyroidism
- Excreted primarily by the kidneys
  - Normally, 80% to 90% of the filtered load of phosphorus is reabsorbed by the proximal tubules of the kidneys
  - Parathyroid hormone decreases phosphorus reabsorption and is the most important regulator of renal phosphate transport.
ANALYSIS

Indications
Serum phosphorus concentration is commonly measured in systemic diseases characterized by anorexia, vomiting, diarrhea, or in patients with hemolysis, diabetes mellitus, renal disease, or hypercalcemia.

Typical Reference Range
The concentration of serum phosphate is generally expressed in terms of serum phosphorus mass (mg/dL). One mg/dL of phosphorus is equal to 0.32 mmol/L of phosphate. Normal serum phosphorus concentration is 2.5 mg/dL to 5.5 mg/dL (0.8 mmol/L–1.8 mmol/L) in dogs and 2.5 mg/dL to 6.0 mg/dL (0.8 mmol/L–1.9 mmol/L) in cats. These values may vary among laboratories and analyzers. They also fluctuate with age (they are higher in young animals) and dietary intake.

Danger Values
Values below 1 mg/dL are associated with hemolysis and rhabdomyolysis. Severe hyperphosphatemia leads to hypocalcemia and metabolic acidosis (for each 1-mg/dL increase in phosphorus there is approximately a 0.55-mEq/L decrease in bicarbonate concentration).

Artifacts
Phosphorus concentration may be increased postprandially. Lipemia, hyperproteinemia, and hemolysis may falsely increase phosphorus concentration, whereas mannitol may falsely lower it.

Drug Effects
Antacids decrease absorption because calcium, aluminum, and magnesium bind phosphorus into insoluble complexes. Insulin and bicarbonate shift phosphorus inside the cell and may lead to hypophosphatemia. Glucose administration may lead to hypophosphatemia by inducing insulin release. Anabolic steroids and calcitriol can increase phosphorus concentration.

HYPOPHOSPHATEMIA

Causes
Hypophosphatemia may result from decreased intestinal absorption (eg, anorexia, malabsorption, vomiting, and diarrhea), increased renal excretion (eg, diabetes mellitus and diuretic administration), or from transcellular shifts (eg, insulin or bicarbonate administration). The most important causes of hypophosphatemia in dogs and cats are presented in Box 1.

Signs
Hypophosphatemia may be clinically silent in many animals. Clinical signs associated with hypophosphatemia are vague, with mild to moderately decreased phosphorus (1 mg/dL–2 mg/dL) and include weakness, disorientation, anorexia, and joint pain. Clinical signs are typically life-threatening when
phosphorus is less than 1 mg/dL with hemolysis, secondary to osmotic fragility, acute respiratory failure, seizures, and coma.

**HYPERPHOSPHATEMIA**

**Causes**

Hyperphosphatemia may result from increased intestinal absorption (eg, vitamin D toxicity, increased dietary phosphorus), decreased renal excretion (eg, renal failure, urinary obstruction), or from transcellular shifts (eg, hemolysis, tumor cell lysis). The most important causes of hyperphosphatemia in dogs and cats are presented in Box 2.
Signs
Hyperphosphatemia usually does not cause clinical signs. However, hyperphosphatemia may lead to hypocalcemia and its associated neuromuscular signs. Hyperphosphatemia is also a risk for soft tissue mineralization.

Stepwise Approach
An algorithm for the differential diagnosis of hyperphosphatemia is presented in Fig. 1.

**Box 2: Common rule-outs for hyperphosphatemia**

- Increased gastrointestinal absorption
  - Vitamin D toxicosis
  - Cholecalciferol rodenticides
  - Psoriasis creams: calcipotriene
  - Phosphate containing enema

- Decreased excretion
  - Renal
    - Prerenal
      - Hypoadrenocorticism
    - Renal
      - Acute
      - Chronic
    - Postrenal
      - Uroabdomen
      - Urinary obstruction
  - Hypoparathyroidism
  - Acromegaly
  - Hyperthyroidism

- Transcellular Shifts
  - Tumor cell lysis
  - Rhabdomyolysis or tissue trauma
  - Hemolysis

- Physiologic
  - Young growing dog
  - Postprandial

- Laboratory error
  - Lipemia
  - Hyperproteinemia

*Most important causes in small animal practice.*
**Hyperphosphatemia**

Azotemia?

Yes

↓ Renal Excretion

- Pre-renal
- Renal
- Post-renal

Normal Calcium Concentration?

Yes

Transcellular Shift

- Tumor cell lysis
- Rabdomyolysis
- Hemolysis

Others

- Hyperthyroidism
- Acromegaly
- Physiologic

Hypocalcemia

Hypercalcemia

↑ GI Absorption

- Phosphate enema toxicity

Others

- Primary hypoparathyroidism

↑ GI Absorption

- Vitamin D toxicity
- Nutritional hyperparathyroidism

**Fig. 1.** Algorithm for evaluation of patients with hyperphosphatemia.

**Further Readings**
