### Causes of Hyperphosphatemia

<table>
<thead>
<tr>
<th>Pseudohyperphosphatemia</th>
<th>Increased endogenous loads</th>
<th>Reduced urinary excretion</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple myeloma</td>
<td>Tumor lysis syndrome</td>
<td>Renal failure</td>
<td>Fluoride poisoning</td>
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<td>Extreme hypertriglyceridemia</td>
<td>Rhabdomyolysis</td>
<td>Hypoparathyroidism</td>
<td>β-Blocker therapy</td>
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<td>In vitro hemolysis</td>
<td>Bowel infarction</td>
<td>Hereditary</td>
<td>Verapamil</td>
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<td>Increased exogenous phosphorus load or absorption</td>
<td>Malignant hyperthermia</td>
<td>Acquired</td>
<td>Hemorrhagic shock</td>
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<td>Phosphorus-rich cow's milk in premature neonates</td>
<td>Heat stroke</td>
<td>Pseudohyoparathyroidism</td>
<td>Sleep deprivation</td>
</tr>
<tr>
<td>Vitamin D intoxication</td>
<td>Acid-base disorders</td>
<td>Vitamin D intoxication</td>
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</tr>
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<td>PO₃⁴-containing enemas</td>
<td>Organic acidosis</td>
<td>Growth hormone</td>
<td></td>
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<td>Intravenous phosphorus supplements</td>
<td>Lactic acidosis</td>
<td>Insulin-like growth factor-1</td>
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<td>White phosphorus burns</td>
<td>Ketoadiposis</td>
<td>Glucocorticoid withdrawal</td>
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<tr>
<td>Acute phosphorus poisoning</td>
<td>Respiratory acidosis</td>
<td>Mg²⁺ deficiency</td>
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<td></td>
<td>Chronic respiratory alkalosis</td>
<td>Tumoral calcinosis</td>
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</tbody>
</table>

**FIGURE 7-25**
Causes of hyperphosphatemia. (From Knochel and Agarwal [5]; with permission.)

### Clinical Manifestations of Hyperphosphatemia

- Consequences of secondary changes in calcium, parathyroid hormone, vitamin D metabolism and hypocalcemia:
  - Neuromuscular irritability
  - Tetany
  - Hypotension
  - Increased QT interval

- Consequences of ectopic calcification:
  - Periarticular and soft tissue calcification
  - Vascular calcification
  - Ocular calcification
  - Conduction abnormalities
  - Pruritus

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**FIGURE 7-26**
Clinical manifestations of hyperphosphatemia.

**FIGURE 7-27**
Treatment of hyperphosphatemia.

### Treatment of Hyperphosphatemia

- **Acute hyperphosphatemia in patients with adequate renal function**
  - Saline diuresis that causes phosphaturia

- **Chronic hyperphosphatemia in patients with end-stage renal disease**
  - Dietary phosphate restriction
  - Phosphate binders to decrease gastrointestinal phosphate reabsorption

**FIGURE 7-27**
Treatment of hyperphosphatemia.
Disorders of Phosphate Balance

Periarticular calcium phosphate deposits in a patient with end-stage renal disease who has severe hyperphosphatemia and a high level of the product of calcium and phosphorus. Note the partial resolution of calcific masses after dietary phosphate restriction and oral phosphate binders. Left shoulder joint before (A) and after (B) treatment. (From Pinggera and Popovtzer [17]; with permission.)

Resolution of soft tissue calcifications. The palms of the hands of the patient in Figure 7-28 with end-stage renal disease are shown before (A) and after (B) treatment of hyperphosphatemia. The patient has a high level of the product of calcium and phosphorus. (From Pinggera and Popovtzer [17]; with permission.)
7.12 Disorders of Water, Electrolytes, and Acid-Base

**FIGURE 7-30**
A, B, Bone sections from the same patient as in Figures 7-28 and 7-29, illustrating osteitis fibrosa cystica caused by renal secondary hyperparathyroidism with hyperphosphatemia.

**FIGURE 7-31**
Roentgenographic appearance of femoral arterial vascular calcification in a patient on dialysis who has severe hyperphosphatemia. The patient has a high level of the product of calcium and phosphorus.

**FIGURE 7-32** (see Color Plate)
Microscopic appearance of a cross section of a calcified artery in a patient with end-stage renal disease undergoing chronic dialysis. The patient has severe hyperphosphatemia and a high level of the product of calcium and phosphorus. Note the intimal calcium phosphate deposit with a secondary occlusion of the arterial lumen.

**FIGURE 7-33**
Massive periarticular calcium phosphate deposit (around the hip joint) in a patient with genetic tumoral calcinosis. The patient exhibits hyperphosphatemia and increased renal tubular phosphate reabsorption. Normal parathyroid hormone levels and elevated calcitriol levels are present. The same disease affects two of the patient’s brothers.