hyperphosphataemia

(i) renal failure
- most common cause
- causes hyperphosphataemia because the renal excretion by the kidneys is impaired
- serum phosphate is usually normal until the creatinine clearance is less than 30ml/min
(ii) increased renal resorption
- hypoparathyroidism
- thyrotoxicosis
(iii) cellular injury with release of phosphate
- tumour lysis syndrome
- rhabdomyolysis
(iv) medication related
- abuse of phosphate containing laxatives
- excessive phosphate administration
- bisphosphonate therapy

(i) limit phosphate intake
(ii) enhance urinary phosphate excretion
- in the absence of end stage renal disease, phosphate excretion can be optimised with saline infusion and diuretics
- diuretics that work on the proximal tubule such as acetazolamide are particularly effective for enhancing phosphate excretion
- any patient with life threatening hyperphosphataemia should receive dialysis
(iii) oral phosphate binders
- calcium and aluminium salts are widely used; however calcium salts may produce metastatic calcification and aluminium salts are toxic.
- in dialysis patients, chronic management with calcium free phosphate binders such as sevelamer hydrochloride may reduce long-term mortality by preventing long-term cardiovascular complications associated with a high calcium phosphorus product

NB: in the acute management of patients with hyperphosphataemia accompanied by hypocalcaemia, the likelihood and clinical significance of metastatic calcification with acute calcium administration is unclear

(i) precipitation with calcium (leading to nephrothiasis)
(ii) interference with parathyroid hormone-mediated resorption of bone
(iii) decreased vitamin D levels
- manifestations of hypocalcaemia include muscle cramping, tetany, hyperreflexia and seizures as well as cardiovascular manifestations