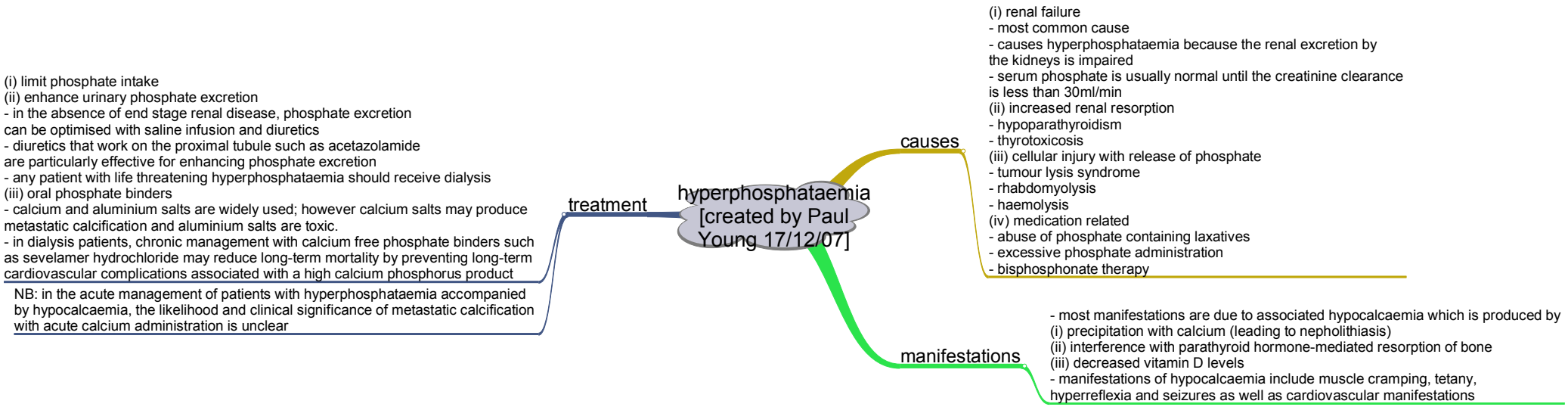
hyperphosphataemia [created by Paul Young 17/12/07]
causes
(i) renal failure
- most common cause
- causes hyperphosphataemia because the renal excretion by the kidneys is impaired
- serum phosphate is usually normal until the creatinine clearance is less than 30ml/min
(ii) increased renal resorption
- hypoparathyroidism
- thyrotoxicosis
(iii) cellular injury with release of phosphate
- tumour lysis syndrome
- rhabdomyolysis
- haemolysis
(iv) medication related
- abuse of phosphate containing laxatives
- excessive phosphate administration
- bisphosphonate therapy
manifestations
- most manifestations are due to associated hypocalcaemia which is produced by
(i) precipitation with calcium (leading to nepholithiasis)
(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
treatment
(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