Serum Phosphate

**Indications**

Muscle weakness, bone pain, suspected bone disease, renal disease (including nephrolithiasis), alcohol abuse, suspected vitamin D deficiency. Phosphate is the major intracellular anion and its metabolism is closely linked to that of calcium.

A low serum phosphate can be caused by:

- poor diet,
- malabsorption (caused by vitamin D or calcium malabsorption, or, for example, antacids binding phosphate in the gut),
- redistribution between the intracellular and extracellular fluids (phosphate moves into cells with glucose) as occurs with athletics and body-building, as well as insulin therapy,
- alkalosis (activation of phosphofructokinase leading to increased glycolysis),
- hepatic disease,
- increased excretion (for example diuretic therapy)
- hypomagnesaemia
- increased PTH secretion
- Fanconi syndrome
- X-linked hypophosphataemic rickets etc.
- ethanol abuse
- the recovery phase after burn injuries

Mild hypophosphatemia is relatively common, but a serum low phosphate can have a profound effect on all organ systems, for example:

1. Muscle - myopathy, rhabdomyolysis
2. Blood - haemolysis, tissue hypoxia, reduced white cell and platelet function
3. Nervous system - malaise, confusion, coma
4. Skeleton - rickets, osteomalacia

A raised serum phosphate can be caused by:

- excessive intake (for example phosphate-containing laxatives and enemas),
- excessive absorption (caused by high vitamin D intake),
- redistribution after tissue destruction (for example after chemotherapy),
- acidosis (phosphate ions move out of cells into the extra-cellular fluid),
- low insulin activity – diabetes mellitus,
- poor excretion (renal failure)
- hypoparathyroidism (in which there is increased tubular reabsorption of phosphate).

Females subject values are generally slightly higher than those of males. Serum phosphate levels are higher in children and neonates than they are in adults, but otherwise values are constant throughout life.

**Interpretation**

Phosphate is absorbed from the small intestine under the control of PTH and vitamin D. The excretion of phosphate is controlled in the kidney. Phosphate is reabsorbed in the proximal tubule via a sodium-dependent transporter, with fine control by PTH (which inhibits phosphate reabsorption) in the distal tubule. There is a circadian rhythm in serum phosphate. Results are highest in the late morning and higher in summer than in winter.

**P.T.O.**
**Reference values:**
Adult: serum or plasma 0.70-1.50 mmol/l
Paediatric (Sheffield Childrens’ Hospital): Infant 1.00-2.60 mmol/l, Child 1.20-2.30 mmol/l.
Urine 12.9-42.0 mmol/24 hrs (Literature derived)

**Patient preparation**
No special preparation is required and the patient can continue to take nutritional supplements and medication before the collection of the sample.

**Specimen requirements**
Serum or lithium heparin plasma.

“Old” blood samples, in which the serum has been in prolonged contact with cells, increase their phosphate level due to the action of phosphatases on the organic phosphates that are released. Postal samples must reach Biolab within 24 hours of collection.

Haemolysed, grossly lipaemic or icteric samples will not be analysed since these factors can produce false elevation in results (by more than 50%).

Urine samples, either 24 hour collections or random samples, should be acidified with 6M HCL to pH < 2 following collection to ensure that all the urine phosphate is in solution.

**Turn around time**
2-3 working days.

**References**

**Suggested further reading**