**Determination of Serum Uric Acid**

Uric acid is a waste product of purine metabolism in man. It has been that the healthy adult human body contain about 1.1 gm of uric acid and about one sixth of this is present in the blood, and the remainder being distributed in other tissues. Two purines, adenine and guanine are important constituents of nucleic acid, and of free nucleotides such as ATP, cAMP and GTP. Xanthine and hypoxanthine are other body purines. Purines can be synthesized in the body or can be ingested. Food stuffs which contain abnormal amounts of nuclear proteins such as liver and pancreas, contains large quantities of purines. The stimulants in coffee and tea (caffeine) and in cocoa (theobromine) are xanthines. The serious consequences of abnormal uric acid metabolism depend in part upon the insolubility of uric acid and its sodium monurate salt. The former crystallizes in the kidney and urinary tract while the latter in cartilage and other tissues around the joints in gout.

Plasma uric acid is filtered by the glomerulus's and is subsequently reabsorbed to about 90% by the tubules. Uric acid concentration in serum are greatly affected by extra renal as well as renal factors.

**Principle:**

Uric acid is oxidized to allantion and carbon dioxide by a phosphotungstic acid regent in alkaline solution. Phosphotungstic acid is reduced in this reaction to tungsten blue which is measured at 710 nm. Protein have been removed by precipitation with tungstic acid, TCA, phosphotungstic acid and heat coagulation. Other oxidizing agents have included arsenotungstic acid, arslenophosphotungstic acid, arsenomolybdic acid, and potassium ferricyanide. Urea-cyanide was latter used as the alkaline reagent, but this modification did not required the isolation of U.A. from the filtrate.
**Clinical Significance:**

There are 3 major causes for elevated level of uric acid: Gout, increased nuclear breakdown and renal diseases.

Gout is a diseases condition found primarily in males and usually first diagnosed between the ages of 40-50 yr, patients have pain and inflammation of the joints caused by precipitation of Na urates owing to the high levels of u.a. found in extracellular fluids. In 25 % - 30 % of these patients hyperuricemia has been shown to be due to overproduction of U. A the plasma UA levels in these patients is usually between 6.5-10 mg/100ml. Use of their high U.A. levels patients with gout are already highly susceptible development of renal calculi.

Another common cause of elevated plasma U.A, levels is in increased breakdown of cell nuclei such as that which occur in patients on chemotherapy for proliferation diseases such as leukemia, lymphomas multiple myeloma or polycythemia. Chronic renal diseases, will also cause elevated levels of U.A.

Hyperuricemia is also a common feature of toxemia of pregnancy and lactic acidosis. The cause of increased plasma levels have is apparently competition for binding sites in the renal tubules.

Elevated levels may also be found after ingestion of a diet rich in purine, or a marked decrease in total dietary intake, resulting in increased tissue breakdown.

In general hyperuricemia and hypouricemia are associated with following clinical disorders.

1 – Hyperuricemia;
- Acute and chronic Nephritis.
- Urinary obstruction
- Gout
• Diabetic ketoacidosis
• High purine diet
• Leukemia
• Malignant tumors especially with extensive necrosis
• Acute infections
• Alcohol ingestion and certain toxins and some diuretics
• Elevate uric acid levels

2- Decreased serum uric acid levels are associated with:

• Pernicious anemia
• Acute yellow atrophy of the liver
• Salicylate and cinchophen therapy.