Biochemistry

ALBUMIN (ALB)

Species: All

Specimen: Plasma or serum

Container: EDTA, heparin or red-top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Helpful in the assessment of a wide variety of conditions (see below).

General information about when this test is indicated: The smallest protein in plasma which accounts for the majority of the oncotic pressure of blood and is an important carrier protein (for bile acids, free fatty acids, bilirubin, Ca, hormones, drugs). Made in liver, enters blood, catabolised by many tissues. A negative acute phase reactant in inflammation.

Major differentials:

Increase: Dehydration (globulin may also increase proportionately). Glucocorticoids may cause mild increases in dogs and possibly cats.

Decrease: Inflammation, reduced protein intake, malabsorption, maldigestion/exocrine pancreatic insufficiency, hepatic disease, protein losing enteropathy, protein losing nephropathy, haemorrhage, effusive disease, exudative disease, severe burns, haemodilution (e.g. IV fluids), hypoadrenocorticism, cachexia, hyperglobulinaemias.

Comparison with other related tests: Interpret in conjunction with total protein/globulin to help differentiate possible causes e.g. after external haemorrhage albumin and globulin are expected to reduce in parallel.

ALKALINE PHOSPHATASE (ALP)

Species: Dog, cat, avian. Not widely used in large animals

Specimen: Plasma or serum

Container: Heparin or red top tube

Collection protocol: Fasted sample preferred.

Special handling/shipping requirements: Standard.

General information about the disease: Intrahepatic and extrahepatic cholestasis, endocrine and neoplastic disorders, and others.

General information about when this test is indicated: An indicator of osteoblastic activity in all species and cholestasis in most species. In birds, it is primarily associated with osteoblastic activity, not useful for hepatobiliary disease. Several isoenzymes, but some have short half-lives and contribute little to serum ALP. Placental isoenzyme can be detected in late pregnancy in cats. Bone isoenzyme is associated with increased serum ALP in juvenile animals, bone remodelling and (probably) feline hyperthyroidism. Liver isoenzyme has a short half-life in cats (6 hours), compare with dogs (70 hours), so magnitude of increase in feline disease is lower. In dogs exogenous or endogenous steroidogenic hormones stimulate both liver and glucocorticoid isoenzymes and the largest increases in serum ALP are seen in cholestatic disorders and glucocorticoid excess, as well as some tumours. Phenobarbital induces ALP activity in the dog. In cats

phenobarbitone and glucocorticoids have little effect, but hepatic lipidosis can be associated with marked increases. Major differentials: Endocrine, cholestasis, neoplasia, breed related, drug effect.

Comparison with other related tests: Parallels, but more sensitive than GGT in canine hepatobiliary disease.

ALANINE AMINOTRANSFERASE (ALT)

Species: Dog, cat

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred.

Special handling/shipping requirements: Standard

General information about the disease: Liver (hepatocyte) injury

General information about when this test is indicated: An indicator of damage to liver cells. Also found in kidney and in cardiac and skeletal muscle. Present in hepatocyte cytosol with higher concentrations in periportal cells. Magnitude in serum correlates to number of cells affected but cannot be used to assess prognosis. Some animals with severe liver disease may have normal serum ALT due to lack of viable hepatocytes. Half-life in dogs controversial but levels usually rise rapidly within 24-48 hours and resolve over 2-3 weeks if no further injury, but pattern is highly variable. Increases seen with Phenobarbital treatment. Major differentials: Circulatory disturbances (including anaemia), hepatotoxicity, infection, inflammation, endocrine associated hepatopathy, neoplasia, drug effect.

Comparison with other related tests: More liver specific than AST

AMYLASE (AMY)

Species: Dog (less useful in other species)

Specimen: Plasma or serum (serum preferred)

Container: Heparin or red-top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Pancreatic or renal disease

General information about when this test is indicated: Amylase is a cytoplasmic enzyme that catalyses the hydrolysis of complex starches. Humans and pigs have salivary alpha amylase, but not dogs, cats, horses or cattle. In healthy dogs serum amylase may be from pancreatic and non-pancreatic tissues with up to 70% being intestinal in origin, but intestine is not considered a significant source of increased serum levels. There may be some amylase activity in the liver and hepatic inactivation of amylase is important in dogs, but hepatic disease is not generally associated with increased serum amylase. The kidneys are a route of excretion or inactivation but mechanisms are incompletely understood. Injury of pancreatic acinar cells (most commonly from pancreatitis) may increase serum amylase, and about 60% of dogs with renal failure also have increased levels. Increases have also been reported in GI disease, hepatic disease and some neoplasias (lymphoma, haemangiosarcoma). Increased serum amylase is not specific (or sensitive) for pancreatic disease. Increases >3 x the upper reference limit are more likely to be related to pancreatic injury than other causes but exceptions (both ways) occur. Half-life in dogs is about 5 hours. In experimental canine pancreatitis amylase peaks in 12-48 hours and persists for 8-14 days. Species differences: In cats with pancreatitis amylase may not increase and may decrease. Increases in horses with pancreatic injury are slight or absent, but are relatively common in equine enteritis (>50% of proximal enteritis cases). When serum amylase is not substantially increased in dogs with suspected pancreatitis it may be useful to

compare amylase activity in serum with that in peritoneal fluid. This may also be applicable in other species (cat, horse) though upper GI (duodenal) perforation could also raise amylase concentration in peritoneal fluid.

Major differentials:

Increase: Pancreatic injury, renal, pre-renal and post-renal disorders/azotaemias, GI disease, hepatic disease, pancreatic neoplasia, other neoplasia.

Decrease: Not usually clinically significant, but has been observed in some dogs with portosystemic shunt.

Comparison with other related tests: Amylase appears to be less sensitive and less specific for pancreatic activity than lipase (except in dogs receiving corticosteroid treatment).

ASPARTATE AMINO TRANSFERASE (AST)

Species: All

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Liver (hepatocyte) or muscle injury

General information about when this test is indicated: An indicator of damage to muscle (skeletal or cardiac) or liver cells. Also found in kidney. Present in cytosol and mitochondria. Increased significantly by muscle activity (e.g. seizure, or even exercise), in which case CK is also usually raised unless the injury is already resolving. In hepatocytes, found in higher concentrations in the periacinar zone. In dogs with hepatocellular injury increases tend to parallel ALT. If ALT (or GD in large animals) not raised, look for extrahepatic source. Short half-life in cats, so even small increases may be significant. Major differentials: Muscle: trauma or necrosis. Liver: circulatory disturbances (including anaemia), hepatotoxicity, infection, inflammation, neoplasia

Comparison with other related tests: See above

BILE ACIDS (BA)

Species: Dog, cat, horse, birds

Specimen: Serum, heparinised plasma

Container: Red top tube, gel tube, heparin

Collection protocol:

- Do not perform this test in an animal with increased bilirubin or in dogs with bilirubinuria – BA will be increased in these animals
- Sample 1 or single pre-prandial should be a fasted sample, avoid haemolysis
- Sample 2 obtain 2 hours after feeding a small amount of food to avoid sample lipaemia, taking care to avoid sample haemolysis

  Note: Lipaemia causes a false increase in BA and haemolysis causes a false decrease in BA

Special handling/shipping requirements: Separating serum from the clot is ideal, but not always possible

General information about the disease: None

General information about when this test is indicated:

- Used to assess liver function in cases where liver disease or failure, or portosystemic shunt are suspected
- Most animals with either congenital or acquired portosystemic shunt will have marked increases in post-prandial bile acids
- Pre-prandial values may be higher than post-prandial values in healthy animals, because of a recent meal, catching sight ore scent of food, or delayed gastric emptying
- Horses do not have a post-prandial increase in bile acids, but bile acids may be mildly increased in anorexic horses
- In parrots with BA repeatedly in excess of 200 umol/L, liver biopsy is warranted

Comparison with other related tests: N/A

BILIRUBIN

Species: All (not birds/reptiles)

Specimen: Plasma or serum

Container: EDTA, heparin or red-top tube

Collection protocol: Fasted sample preferred.

Special handling/shipping requirements: Standard

General information about the disease: Useful in the assessment of liver function and some anaemias.

General information about when this test is indicated: A by-product of haemoglobin breakdown from senescent (or diseased) erythrocytes in mammals. Released from macrophages and transported to liver where it is conjugated to a sugar group and most is excreted in bile with some going back into blood. Most of the bilirubin entering the small intestine and converted to urobilinogen is excreted in faeces, with a small amount reabsorbed into blood where it re-enters liver or is excreted in urine.

Increased serum bilirubin can occur from:

1. Increased haemoglobin production (haemolysis, especially extravascular, internal haemorrhage) termed prehepatic, haemolytic, or retention hyperbilirubinaemia;
2. Reduced uptake or conjugation by hepatocytes (reduced blood flow, reduced hepatocyte numbers, defective uptake or conjugation) termed hepatic or, again, retention hyperbilirubinaemia;
3. Fasting (most pronounced effect is in horse)
4. Disruption of bile flow (intra- or extra- hepatic) causing cholestasis (inflammation, neoplasia, choleliths, lipidosis, endocrinopathy associated hepatopathy) termed posthepatic or cholestatic hyperbilirubinaemia.

Species differences are notable: Most birds and reptiles have little biliverdin reductase and therefore do not produce bilirubin in health. Biliverdin is the main bile pigment in these species and bilirubin is rarely diagnostically useful. Dogs, especially male, have a low renal threshold, so hyperbilirubinuria may precede hyperbilirubinaemia. In horses most serum bilirubin is unconjugated regardless of cause and very high levels can occur, e.g. in anorexia. In ruminants, liver disease may not always raise bilirubin and high levels are most commonly associated with haemolysis.

Major differentials:

Increase: Haemolysis, cholestasis, hepatocyte injury, biliary system disease, reduced functional hepatic mass.

Decrease: Not usually clinically significant, possibly reduced red cell production.

Comparison with other related tests: N/A

CHOLESTEROL

Species: All, including birds

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Used mainly to assist with assessment of liver function and some endocrinopathies.

General information about when this test is indicated: A sterol lipid found in animal tissue which can be absorbed from diet or made in liver (and other tissues). In fasting samples most will have been made by hepatocytes. Hypercholesterolaemia can result from increased production, reduced lipolysis/processing of lipoproteins, and some other processes. Hypocholesterolaemia can result from reduced production and some other processes. Liver diseases may result in increases or decreases in cholesterol, depending on the pathogenesis.

Major differentials:

Increase - Postprandial, nephrotic syndrome, hypothyroidism, diabetes mellitus, excess glucocorticoids/ hyperadrenocorticism, pancreatitis, idiopathic hyperlipidaemia (breed related), and cholestasis.

Decrease - Hepatic dysfunction/shunt, low fat diet, protein losing enteropathy/GI disease/malabsorption, hyperthyroidism, hypoadrenocorticism.

Comparison with other related tests: N/A

CK (CREATINE KINASE)

Species: All

Specimen: Plasma or Serum (serum preferred)

Container: EDTA (K only), heparin (Li or NH4 only) or red-top tube.

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Muscle injury or muscle catabolism

General information about when this test is indicated: An indicator of damage to muscle (skeletal or cardiac) or, particularly in cats, muscle catabolism. Also found in smooth muscle, brain and nerves but damage to these tissues does not usually affect serum CK. Present in cytoplasm and leaks from damaged cells, increasing rapidly after muscle injury, peaking in about 6-12 hours and returning to normal within 24-48 hours after injury has stopped. Half-life <2 to 4 hours in most common domestic species. Can be increased significantly by muscle activity, including exercise – particularly relevant to racing animals. Species and age differences: Puppies have higher serum CK than adult dogs and canine serum CK may be higher than plasma CK due to presence of CK in canine platelets. Bovine uterus has enough CK to produce increased serum CK in endometritis.

Major differentials:

Increase: Muscle damage from physical trauma, hypoxia, thrombosis, exertion, seizure, recumbency, VitE/Se deficiency, inflammatory myositis (Neospora, Toxoplasma, bacteria etc), toxin, inherited disease, neoplasia. Muscle catabolism, especially in anorexic cats. Haemolysis (artefact).

Decrease: Not usually clinically significant. Can be seen in some animals with reduced muscle mass.

Comparison with other related tests: After muscle injury CK increases more rapidly than AST and returns to normal earlier.

CREATININE

Species: All (but not very useful in birds/reptiles)

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Used as a convenient, though poorly precise/sensitive, indicator of renal function/ glomerular filtration rate (GFR).

General information about when this test is indicated: Creatinine is formed from muscle creatine at a relatively constant daily rate, not as affected by extra-renal factors as urea. Minor dietary and postprandial effects (red meat consumption increases and eating a meal decreases serum urea). Excreted almost exclusively through glomerular filtration in kidneys. Patient may experience significant reduction in functional nephron number/GFR (>75%) before serum creatinine is significantly increased. Reductions in GFR from prerenal, renal, or post-renal causes cannot be distinguished as all may increase serum creatinine. Post-renal causes (e.g. lower urinary tract obstruction) are usually associated with the largest and fastest increases. Decreased results are rarely clinically significant, though animals with lower muscle mass tend to have lower creatinine.

Major differentials include reduced renal perfusion (dehydration, shock, cardiovascular disease) urinary tract obstruction or rupture, renal disease.

Comparison with other related tests: Tends to parallel serum urea in renal disease or reduced renal perfusion. Affected less by extra-renal factors. Should be interpreted in conjunction with urinalysis.

GLOBULIN (GLOB)

Species: All

Specimen: Plasma (includes fibrinogen) or serum (has no fibrinogen)

Container: EDTA, heparin or red-top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Helpful in the assessment of a wide variety of conditions (see below). Calculated by subtraction (TP-ALB).

General information about when this test is indicated: A group of large, variably sized proteins. Classified by electrophoretic mobility into alpha (includes transport proteins for thyroxine, cortisol, lipids, copper, and haemoglobin binder, thrombin inhibitor, insulin binder and trypsin inhibitor proteins) – produced in liver; beta (includes transport proteins for lipids and iron, C-reactive protein, complement components C3 and C4, plasminogen and, in plasma, fibrinogen) – produced mostly in liver; and gamma (immunoglobulins) – produced in lymphoid tissue in response to antigenic stimulation. Some immunoglobulins may migrate into the beta fraction in electrophoresis. Fibrinogen is converted to fibrin when blood clots so is not present in serum, only plasma. Hepatic failure may reduce globulin, but chronic hepatic disease may cause increase (especially in horses).

Major differentials:

Increase: Dehydration, inflammation (includes infectious, neoplastic, traumatic, immune mediated etc.), B-lymphocyte neoplasia, nephrotic syndrome.

Decrease: Haemorrhage, hepatic disease, protein losing enteropathy, protein losing nephropathy, effusive disease, exudative disease, severe burns, haemodilution (e.g. IV fluids), failure of passive transfer in neonates, inherited or acquired immune deficiency.

Comparison with other related tests: Interpret in conjunction with total protein/albumin to help differentiate possible causes e.g. after external haemorrhage albumin and globulin are expected to reduce in parallel. Differentiation of fractions by electrophoresis may be useful, especially for identifying monoclonal peaks (which usually signify B-lymphocyte neoplasia).

FRUCTOSAMINE

Species: Dog, cat

Specimen: Serum

Container: Red top tube

Collection protocol:

- Fasted sample, particularly if other biochemical tests will be run
- Levodopa at therapeutic doses can cause a false increase in fructosamine
- Hyperthyroid cats may have decreased serum fructosamine

Special handling/shipping requirements: Standard

General information about when this test is indicated: Fructosamine is used for the diagnosis and monitoring of diabetes mellitus in cats and dogs. It is particularly useful in cats, where stress hyperglycaemia can be marked and may lead to glucosuria and interference with the performance of blood glucose curves during therapy.

Generally fructosamine concentrations provide a reflection of blood glucose concentrations during the previous 1-3 weeks but it is important to note that other conditions may affect this analyte. Examples include hypoalbuminaemia/ hypoproteinaemia, hyperlipidemia, azotemia and in cats, hyperthyroidism.

Serum fructosamine may also be useful for demonstrating prolonged hypoglycemia in animals presenting with a suspicion of an insulinoma/ beta cell tumour. Further workup to exclude other causes of hypoglycaemia and demonstrate inappropriate insulin secretion however would still be required to confirm a diagnosis of insulinoma. Used to monitor response to insulin therapy

Comparison with other related tests: N/A

GAMMA GLUTAMYL TRANSFERASE (GGT)

Species: All

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Cholestasis/biliary disease (especially intrahepatic), colostrum ingestion.

General information about when this test is indicated: Membrane bound glycoprotein associated with bile ducts/canaliculi. Serum increases associated with intrahepatic bile duct obstruction and biliary disease (e.g. sporidesmin toxicity in ruminants). Glucocorticoids stimulate production in dogs, similar to ALP, but anticonvulsants such as phenobarbital have only mild effects. Produced abundantly by mammary gland with high levels in colostrum (not in the horse). Neonates of most species (not cats) have high serum levels. This test has been used to assess adequacy of colostral transfer in some species (e.g. in calves it has been suggested that levels should be >780 U/L at 1 day, >520 U/L at 3 days, >169 U/L at 5-10 days and >85 U/L at 10-15 days)

Major differentials include cholestasis, biliary disease, colostrum ingestion, endocrine.

Comparison with other related tests: More useful than ALP for detecting cholestasis in large animals (horses, ruminants) due to narrower reference intervals.

GLUTAMATE DEHYDROGENASE (GDH, GLDH)

Species: All but most commonly used in large animals (horses, ruminants)

Specimen: Plasma or serum

Container: EDTA, heparin or red top tube

Collection protocol: Fasted sample preferred (in companion animals)

Special handling/shipping requirements: Standard

General information about the disease: Liver (hepatocyte) injury, especially acute.

General information about when this test is indicated: An indicator of damage to liver cells. Mitochondrial leakage enzyme in cytoplasm. In humans more activity in centrilobular than periportal hepatocytes. More liver specific than AST. Generally, less sensitive than GGT for detecting liver damage in horses but has a better positive predictive value. Magnitude in serum generally correlates to number of cells affected but cannot be used to assess prognosis. Half-life approximately 14 hours in bovine. Increases sometimes with Phenobarbital treatment. Major differentials: Circulatory disturbances (including anaemia), hepatotoxicity, infection, inflammation, endocrine associated hepatopathy, neoplasia, drug effect.

Comparison with other related tests: More liver specific than AST and generally, less sensitive than GGT for detecting liver damage in horses but has a better positive predictive value.

LIPASE (LIP)

Species: Dog (less useful in other species)

Specimen: Plasma or serum (serum preferred)

Container: Heparin or red-top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Pancreatic or renal disease

General information about when this test is indicated: Lipase is a cytoplasmic enzyme that catalyses the hydrolysis of triglycerides. It is present in pancreas, adipose tissue, gastric and duodenal mucosa, liver and other tissue. The source of serum lipase in healthy dogs is controversial. The kidneys are a route of

excretion or inactivation but mechanisms are incompletely understood. Exogenous corticosteroid treatment can significantly increase serum lipase in dogs (mechanism unclear). Injury of pancreatic acinar cells (most commonly from pancreatitis) may increase serum lipase, and about 50% of dogs with renal failure also have increased levels. Increases have also been reported in GI disease, hepatic disease and some neoplasias (lymphoma, haemangiosarcoma) as well as steatitis/panniculitis. Increased serum lipase is not specific (or sensitive) for pancreatic disease. Increases >2 x the upper reference limit are more likely to be related to pancreatic injury than other causes but exceptions (both ways) occur and a notable exception is with exogenous steroid treatment (see above). Half- life in dogs is short (2-6 hours). In experimental canine pancreatitis lipase changes at a similar rate to amylase (peaks within first 2-4 days). Species differences: In cats with pancreatitis lipase is commonly not increased. Usefulness in horses and other species is not well defined. When serum lipase is not substantially increased in dogs with suspected pancreatitis it may be useful to compare lipase activity in serum with that in peritoneal fluid. This may also be applicable in other species (cat, horse) though upper GI (duodenal) perforation could also raise lipase concentration in peritoneal fluid.

Major differentials:

Increase: Corticosteroid therapy, pancreatic injury, renal, pre-renal and post-renal disorders/azotaemias, steatitis/panniculitis, GI disease, pancreatic neoplasia, other neoplasia, possibly hepatopathy.

Decrease: Not usually clinically significant.

Comparison with other related tests: Lipase appears to be more sensitive and specific for pancreatic activity than amylase, except when the dog is receiving corticosteroid therapy.

TOTAL PROTEIN (TP)

Species: All

Specimen: Plasma (includes fibrinogen) or serum (has no fibrinogen)

Container: EDTA, heparin or red-top tube.

Collection protocol: Fasted sample preferred.

Special handling/shipping requirements: Standard

General information about the disease: Helpful in the assessment of a wide variety of conditions (see below).

General information about when this test is indicated: Includes albumin and globulins. Most produced in liver (albumin, most alpha and beta globulins) with some produced in lymphoid tissue in response to antigenic stimulation (gamma globulins/immunoglobulins). Fibrinogen (in beta fraction) is converted to fibrin when blood clots so is not present in serum, only plasma. TP may remain in normal limits even when albumin or globulin is not, so evaluate all three.

Major differentials:

Increase: Dehydration, inflammation (includes infectious, neoplastic, traumatic, immune mediated etc.), B-lymphocyte neoplasia, nephrotic syndrome.

Decrease: Inflammation, haemorrhage, reduced protein intake, malabsorption, maldigestion/exocrine pancreatic insufficiency, hepatic disease, protein losing enteropathy, protein losing nephropathy, effusive disease, exudative disease, severe burns, haemodilution (e.g. IV fluids), hypoadrenocorticism, cachexia, failure of passive transfer in neonates, inherited or acquired immune deficiency.

Comparison with other related tests: Interpret in conjunction with albumin and globulin to help differentiate possible causes. E.g. after external haemorrhage albumin and globulin are expected to reduce in parallel.

TRYPSIN-LIKE IMMUNOREACTIVITY (TLI)

Species: Canine

Specimen: Serum

Container: Red top tube or gel serum tube

Collection protocol: Fasted sample required. Post prandial samples may have elevated TLI values.

Special handling/shipping requirements: Standard

General information about the disease: Exocrine pancreatic insufficiency (EPI) is typically diagnosed once 90% of exocrine function has been lost. Causes of EPI in dogs include pancreatic acinar atrophy, which is an immune-mediated disease most often seen in young adult German Shepherds as well as some Rough Collies and English Setters. EPI may also occur with chronic pancreatitis and with pancreatic tumours.

General information about when this test is indicated: TLI is an accurate and specific indicator of pancreatic function and the test of choice for dogs with EPI. The assay is species specific and measures both trypsin and trypsinogen. Clinical findings with EPI are characterised by fat malabsorption and include weight loss with fatty, foul smelling faeces (steatorrhea) and polyphagia. Hypoproteinaemia is not typically associated with EPI.

Comparison with other related tests:

Plasma lipase is often normal in these cases due to extra pancreatic sources.

Feline EPI is uncommon, however feline TLI can be requested, and is referred overseas to Texas A&M University.

Interpretation:

A diagnosis of EPI is supported with very low levels of TLI (<2.5 ug/L).

- Increased TLI values may also occur with active pancreatitis, marked reductions in glomerular filtration rate.
- The assay does not cross react with pancreatic enzyme supplementation.

UREA

Species: All (but less useful in birds/reptiles)

Specimen: Plasma or serum

Container: EDTA, lithium heparin or red top tube

Collection protocol: Fasted sample preferred

Special handling/shipping requirements: Standard

General information about the disease: Used as a traditional but poorly precise/sensitive indicator of renal function/Glomerular filtration rate (GFR).

General information about when this test is indicated: Most of the urea produced by the body is excreted through the kidneys. Reduced GFR increases serum urea (BUN), but BUN is also affected by many extra-renal factors. Increased dietary protein, upper GI bleeding, and increased protein catabolism can increase BUN, and conversely low protein intake and reduced hepatic function can reduce it. Also affected by tubular flow rate (so hydration status is relevant). Patient may experience significant reduction in functional nephron number/GFR (>75%) before serum urea is significantly increased. Reductions in GFR from pre-renal, renal, or post-renal causes cannot be distinguished as all may increase serum urea.

Major differentials:

Increase: Reduced renal perfusion (dehydration, shock, cardiovascular disease, hypoadrenocorticism), urinary tract obstruction or rupture, renal disease. Extra-renal (dietary protein, GI haemorrhage, catabolic state)

Decrease: Hepatic dysfunction/shunt, protein malnutrition/malabsorption, fluid therapy, polydipsia.

Comparison with other related tests: Tends to parallel serum creatinine in renal disease or reduced renal perfusion. When only urea is increased look for extra-renal cause. Should be interpreted in conjunction with urinalysis.

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