Chemistry

Differential Diagnoses

FOR THE **VETSCAN® VS2**

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Improve your **chemistry differential diagnoses**

CHEMISTRY Differential Diagnoses for the VETSCAN VS2 is designed to provide you as a busy veterinary practitioner with a practical, comprehensive, and quick reference to laboratory diagnostic testing. It focuses on tests regularly used in the diagnosis and management of medical conditions of dogs and cats.

In combination with a patient history and complete physical examination, it should be used to help create a differential diagnosis and rule out list and help determine, if necessary, the next diagnostic steps.

> "Quickly locate and use the information essential to the practice of high-quality veterinary medicine."

Our objective in creating this document is to **provide patient-side information in a clear, concise, and easy-to-use format.** It is designed to help you **quickly locate and use the information essential to the practice of high-quality veterinary medicine.**

Each section also includes a **list of potential pre-analytical and analytical conditions** that may cause artifactual changes in the chemistry results from any analyzer. Clinicians are frequently presented with test results inconsistent with preconceived expectations for a given case, and unrelated to the patient's condition.

This will **serve as a quick and user-friendly guide for identification of possible pitfalls when presented with unexpected laboratory test results** for a given case. This information may also be used to forewarn you of potential test interference and to optimize results, while preventing repeat testing required by complications in sample handling or sample conditions.

A list of **additional recommended analytes** is intended to provide **clinically relevant information** that will enable you to determine quickly which other analytes are indicated in a particular case. This should help to **create or refine the differential diagnosis and rule out list.**

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VETSCAN VS2 Test Portfolio

Categories & Profiles

Comprehensive	**Comprehensive Diagnostic Profile**	ALB, ALP, ALT, AMY, BUN, Ca, CRE, GLOB*, GLU, K^+ Na^+, PHOS, TBIL, TP
	Preventive Care Profile Plus	ALB, ALP, ALT, AST, BUN, Ca, Cl^-, CRE, GLOB*, GLU, K^+, Na^+, TBIL, tCO_2, TP
Pre-Anesthetic + Critical Care	**Prep Profile II**	ALP, ALT, BUN, CRE, GLU, TP
	Electrolyte Plus	Cl^-, K^+, Na^+, tCO_2
	Critical Care Plus	ALT, BUN, Cl^-, CRE, GLU, K^+, Na^+, tCO_2
Organ Specific	**Kidney Profile Plus**	ALB, BUN, Ca, Cl^-, CRE, GLU, K^+, Na^+, PHOS, tCO_2
	Mammalian Liver Profile	ALB, ALP, ALT, BA, BUN, CHOL, GGT, TBIL
Specialty Testing	**T4/Cholesterol Profile**	CHOL, T4
	Phenobarbital Profile	ALB, ALP, ALT, AST, BUN, GGT, PHB, TBIL
Species Specific	**Avian/Reptilian Profile Plus**	ALB, AST, BA, Ca, CK, GLOB*, GLU, K^+, Na^+, PHOS, TP, UA
	Equine Profile Plus	ALB, AST, BUN, Ca, CK, CRE, GGT, GLOB*, GLU, K^+, Na^+, TBIL, tCO_2, TP
	Large Animal Profile	ALB, ALP, AST, BUN, Ca, CK, GGT, GLOB*, Mg, PHOS, TP

Discover the opportunities of on-site blood testing

TABLE OF CONTENTS

ALANINE AMINOTRANSFERASE (ALT)

Also known as SGPT: Serum Glutamic - Pyruvic Transaminase

INCREASED

Degenerative
- **Anoxia due to anemia/ shock/passive congestion** [1, 2]

Infectious
- **Canine Adenovirus**
- **Leptospirosis**
- **Leishmania**
- **Toxoplasma**
- **Neospora**
- **Hepatozoon**
- **Histoplasmosis**
- **Dirofilariosis**
- **Feline Infectious Peritonitis**
- **Bacterial Cholangiohepatitis** [1, 2]

Non-Infectious
- **Chronic Hepatitis**
- **Cholangitis**
- **Cholangiohepatitis**
- **Cirrhosis**
- **Copper storage disease**
- **Pancreatitis** [2]

Toxic (Not an exhaustive list)
- **Acetaminophen (especially cats)**
- Amiodarone
- L-Asparaginase
- Azathioprine
- **Barbiturates**
- **Carprofen**
- Clindamycin
- Cyclosporin
- Doxycycline
- Diazepam
- Erythromycin estolate
- **Glucocorticoids (dogs only)**
- Griseofulvin
- Ibuprofen
- Itraconazole
- Ketoconazole
- 6-Mercaptopurine
- Methimazole
- Methotrexate
- Nitrofurantoin
- **Phenobarbital**
- Phenytoin
- **Primidone**
- Salicylates
- Salicylazosulfapyridine
- Sulfonamides
- Tetracycline
- **Trimethoprim-sulfa drug**
- Xylitol [1]

Metabolic
- **Hepatic lipidosis**
- **Diabetes mellitus**
- **Feline hyperthyroidism**
- **Hyperadrenocorticism** [1, 2]

Neoplastic
- **Lymphoma**
- **Hepatocellular carcinoma**
- **Metastatic neoplasia** [1, 2]

Anomalous
- Portosystemic shunt (generally mild increase) [1]

Nutritional
- Copper toxicosis
- Hemochromatosis [1]

Inherited
- Copper storage disease
- Lysosomal storage disease [1]

Traumatic
- **Hit by car** (hepatocellular or skeletal muscle damage) [1]

Inflammatory / Infectious
- Myositis [3]

Inherited
- Canine musculodystrophy [1]

DECREASED

Hepatic atrophy
- Chronic congenital portosystemic shunts [3]
- Chronic liver failure
- Cirrhosis

ARTIFACT
- Hemolysis can cause mild increase [3]
- Lipemia can cause artifactual increase [3]

INTERPRET ALT WITH:
- Bilirubin
- Hepatic enzymes
- Creatine kinase

NB. Common differential diagnoses are in bold.

Ref.

ALBUMIN (ALB)

INCREASED

Hemoconcentration
Increased ALB synthesis (or increased ALB lifespan) induced by glucocorticoid drugs [1, 2]
Hepatocellular carcinoma (rare) [3]

DECREASED

Blood Loss (Hemorrhage) [1, 2]

Protein-Losing Nephropathy [1, 2]
- **Glomerulonephritis**
- **Amyloidosis**

Protein-Losing Enteropathy [1, 2]
- **Small intestinal mucosal disease**
- **Lymphangiectasia**
- **Intestinal blood loss**

Sequestration [1]
- **Third space losses (pleural / peritoneal effusion)**
- Vasculopathy

Decreased Albumin Synthesis
- **Inflammation** [4]
- **Hepatic insufficiency (chronic hepatic disease)** [1, 2]
- **Malabsorption and maldigestion** [1]
- **Cachectic / catabolic state** [1]
- Hypergammaglobulinemia [1]

Edematous Disorders [1]
- Congestive heart failure
- Cirrhosis
- Nephrotic syndrome
- Excess Antidiuretic Hormone (ADH) secretion (syndrome of inappropriate antidiuretic hormone secretion, SIADH)

Excess Administration of Intravenous Fluid [1, 2]

ARTIFACT

- High fibrinogen levels in heparanized plasma samples used with a BCG method may cause false increases [5]
- Hemolysis or hemoglobinemia can cause false increases
- Marked lipemia or hypertriglyceridemia can cause false decreases
- Severe hypoalbuminemia can cause a falsely elevated albumin concentration (rare) [6]

INTERPRET ALB WITH:

- Total protein
- Globulins
- Blood Urea Nitrogen
- Creatinine
- Liver function tests
- Packed cell volume
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

ALKALINE PHOSPHATASE (ALP)

INCREASED

Physiologic
- **Age (young dogs with rapid bone growth)**
- Breed (Siberian Huskies – benign familial hyperphosphatasemia; Scottish Terriers) [1, 2]
- **Endogenous corticosteroid release** [3]

Biliary Tract Disease
- Cholangitis
- Bile duct neoplasia
- Cholelithiasis
- Cholecystitis
- Gall bladder mucocele
- Ruptured gallbladder
- Pancreatitis (local inflammation)
- Pancreatic neoplasia [3, 4]

Degenerative
- **Hepatocyte swelling or necrosis (leads to impaired bile flow)** [3]

Metabolic
- **Diabetes mellitus**
- **Hyperadrenocorticism**
- **Hypothyroidism (dogs)**
- **Hyperthyroidism (cats)**
- **Hyperparathyroidism (primary or secondary)**
- **Hepatic lipidosis** [3] (in the initial phase of feline hepatic lipidosis, ALP activity will be markedly increased with little to no increase in GGT activity)

Inflammatory
- **Periportal hepatitis**
- **Chronic hepatitis**
- **Cholangiohepatitis**
- **Feline infectious peritonitis**
- Copper storage disease
- **Cirrhosis/fibrosis**
- **Pancreatitis (local inflammation)** [4]

Neoplastic
- **Lymphoma**
- **Hemangiosarcoma**
- **Hepatocellular carcinoma**
- **Metastatic carcinoma** [3]

Toxic Hepatitis
- Aflatoxin
- Certain types of mushrooms
- Sago palm
- Drug induced [3]

Induction by Drugs or Hormones
(not an exhaustive list)
- Anabolic steroids/androgens
- Asparaginase
- Azathioprine
- **Barbiturates**
- Cephalosporins
- Cyclophosphamide
- Dapsone
- Erythromycin estolate
- **Estrogens** (urinary incontinence medication)
- **Glucocorticoids (dogs only)**
- Griseofulvin
- 6-Mercaptopurine
- Methimazole
- Methotrexate
- Nitrofurantoin
- **Phenobarbital**
- Phenothiazines
- **Primidone**
- Progesterone
- Testosterone
- Tetracyclines
- Thiabendazole
- Trimethoprim-sulfa combinations
- Vitamin A

Cardiogenic
- **Chronic passive congestion from right heart failure** [4]

Increased Osteoblastic Activity
- Fracture healing [3]
- Osteosarcoma [3]

Neoplasia
- Mammary neoplasia (dogs) [5]
- Osteosarcoma [3]

DECREASED

Not clinically significant

ARTIFACT

Severe hemolysis may falsely decrease ALP [6]

INTERPRET ALP WITH:

- Hepatic enzymes
- Cholestatic markers

NB. Common differential diagnoses are in bold.

Ref.

AMYLASE (AMY)

Note: Differential diagnoses also pertain to LIPASE (LIP) where stated

INCREASED

Acute Pancreatitis / Pancreatic acinar cell damage

- **Inflammation (AMY and LIP)** [1, 2]
- **Neoplasia (AMY and LIP)**

Decreased Renal Clearance / Renal Disorder

- **Dehydration** [1, 2]
- Shock (AMY and LIP) [1, 2]
- **Acute or chronic renal diseases (AMY and LIP)** [1]
- Urinary tract obstruction (AMY and LIP)
- Macroamylasemia [1]

Other Causes

- Gastrointestinal obstruction (AMY and LIP)

DECREASED

Not clinically significant

INTERPRET AMYLASE AND LIPASE WITH:

- Pancreatitis specific markers (i.e. canine pancreas-specific lipase)
- Blood Urea Nitrogen
- Creatinine
- Hepatic enzymes
- Bilirubin
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

ASPARTATE AMINOTRANSFERASE (AST)

Also known as GOT: Glutamic Oxaloacetic Transaminase

INCREASED

Hepatic Damage

- See hepatocyte damage conditions listed for increased alanine aminotransferase (ALT) activity [1, 2]

Muscular

- See skeletal muscle damage condition listed for increased creatine kinase (CK) activity [1, 2]

DECREASED

Not clinically significant

ARTIFACT

- Hemolysis will increase AST serum activity [3]
- Icterus may affect results
- Marked lipemia may interfere with spectrophotometric assays [3]
- Metronidazole may artifactually depress AST activity [3]

INTERPRET AST WITH:

- Alanine aminotransferase
- Alkaline phosphatase
- Total bilirubin
- Gamma-glutamyltransferase
- Bile acids
- Creatine kinase

NB. Common differential diagnoses are in bold.

Ref.

BILE ACIDS (BA)

Panel With Fasting and Post-Prandial Samples Preferred

INCREASED

Decreased Functional Hepatic Mass

- **Diffuse hepatocellular disease** [1]

Decreased Portal Blood Flow to Liver

- **Congenital and acquired portosystemic shunts** [1]
- **Hepatic microvascular dysplasia** [2]

Hepatic Cholestasis (Obstructive)

- Hepatic lipidosis
- Diabetes mellitus
- Steroid hepatopathy
- Lymphoma
- Histoplasmosis
- Cytauxzoonosis
- **Cirrhosis**
- **Cholangitis/Cholangiohepatitis**
- **Periportal hepatitis**
- Pyrrolizidine alkaloid toxicosis [1]

Post-hepatic Cholestasis

- **Cholangitis**
- **Bile duct carcinoma**
- **Liver fluke**
- **Cholelithiasis**
- **Cholecystitis**
- **Pancreatitis**
- **Pancreatic carcinoma** [1]

Functional Cholestasis

- Sepsis-associated cholestasis [1]

DECREASED

Physiologic

- Prolonged fasting [1]

Enterohepatic

- Delayed gastric emptying
- Intestinal malabsorption
- Rapid gastrointestinal transit
- Ileal resection [2]

ARTIFACT

Decreased BA value

- Hemolysis [3]
- Heparin BA [3]
- Lipid-clearing agents [3]
- Incomplete gallbladder contraction after feeding may result in a lower value than expected [2]
- Cholestryamine binds BAs and prevents their resorption [3]

Increased BA value

- Lipemia (spectrophotometry)[3]
- Spontaneous gallbladder contraction (without feeding) [2]
- Treatment with ursodiol (a synthetic BA) [3]

INTERPRET BILE ACIDS WITH:

- Pancreatic specific markers (canine or feline pancreas-specific lipase)
- Hepatic enzymes
- Cholestatic markers
- Bilirubin
- Ammonia

NB. Common differential diagnoses are in bold.

Ref.

BLOOD UREA NITROGEN (BUN)

INCREASED

Pre-Renal Conditions

- Hypovolemia
  - **Dehydration**
  - **Hypoadrenocorticism**
  - **Shock**
  - **Blood loss**
- Decreased cardiac output
  - **Cardiac insufficiency**
  - **Shock**
  - **Hypoadrenocorticism**
- Shock
  - **Hypovolemic**
  - **Cardiogenic**
  - **Anaphylactic**
  - **Septic**
  - **Neurogenic** [1, 2]

Renal Conditions

- Inflammatory
  - **Glomerulonephritis**
  - **Pyelonephritis (e.g. leptospirosis)**
  - Tubular-interstitial nephritis [1, 2]
- Amyloidosis
- Toxic nephrosis
  - **Hypercalcemia**
  - **Ethylene glycol intoxication**
  - Myoglobin
  - Gentamicin
  - **NSAID intoxication** [1]
- Renal ischemia or hypoxia
  - **Poor renal perfusion**
  - **Infarction** [1]
- Congenital hypoplasia or aplasia [1]
- Hydronephrosis
- Neoplasia (renal or metastatic) [1, 2]

Post-Renal Conditions

- Urinary tract obstruction
  - **Urolithiasis**
  - **Urethral plugs in cats**
  - **Neoplasia**
  - Prostatic disease
  - **Uroabdomen** [1, 2]

Increased Production

- **Hemorrhage into the gastrointestinal system**
- **High protein diet**
- **Fever**
- **Burns**
- **Corticosteroid administration**
- **Starvation (increased protein catabolism)**
- Exercise [1, 2]

DECREASED

Decreased BUN Synthesis

- **Hepatocellular disease**
- **Portosystemic shunts (congenital or acquired)**
- Urea cycle enzyme deficiencies [1, 2]

Increased Renal Excretion of Urea

- Impaired proximal tubular resorption of urea: glucosuria [1]
- Central or nephrogenic diabetes insipidus (polyuria/polydipsia) [1]

ARTIFACT

- Severe hemolysis will increase BUN concentration [1, 3]
- Severe icterus may increase BUN concentrations [1]
- Severe lipemia may decrease BUN concentrations
- Contamination of the sample with ammonium (NH_4) [1, 3] (e.g. benzalkonium chloride disinfectants) can increase BUN results obtained by reflectance spectrophotometry

INTERPRET BLOOD UREA NITROGEN WITH:

- Creatinine
- Total protein
- Albumin
- Electrolytes
- Anion gap
- Calcium
- Phosphate
- Hepatic function tests
- Hematology
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

CALCIUM (CA)

INCREASED

PHYSIOLOGIC

- **Healthy young, fast growing dogs** (young dog < 6 months, large or giant breed) [1, 2]

INCREASED PRIMARY HYPERPARATHYROIDISM (PTH) OR PARATHYROID HORMONE-RELATED PROTEIN (PTHrp) ACTIVITY

Primary Hyperparathyroidism (PTH)

- **Parathyroid neoplasia** [1]

Hypercalcemia of Malignancy / Humoral Hypercalcemia (PTHrp)

- **Lymphoma**
- **Apocrine gland adenocarcinoma** [1, 2]
- **Carcinoma**
  - **Squamous cell**
  - **Mammary**
  - **Bronchogenic**
  - **Prostate**
  - **Thyroid**
  - **Nasal cavity** [1, 2]
  - **Thymoma** [3, 4]
- Local Osteolysis
  - **Lymphoma**
  - **Multiple myeloma**
  - **Squamous cell carcinoma**
  - **Osteosarcoma**
  - **Fibrosarcoma** [1, 2]

Humoral Hypercalcemia of Benign Disorders

- Canine schistosomiasis [1]

Increased Vitamin D Activity (Hypervitaminosis D)

- Exogenous Vitamin D
  - **Rodenticides containing cholecalciferol**
  - Tacalcitol or calcipocriol
  - Plants containing ergocalciferol (vitamin D_2)
  - Excess dietary supplementation [1, 2]
- Endogenous Vitamin D
  - Granulomatous inflammation
    - **Blastomycosis**
    - **Histoplasmosis**
    - **Cryptococcosis**
    - **Pulmonary angiostrongylosis**
    - **Feline infectious peritonitis** [1, 2]
  - Neoplasm-associated hypervitaminosis D [1]

Bone Neoplasia

- Osteosarcoma
- Myeloma
- Lymphoma
- Metastatic neoplasm [1, 2]

Decreased Urinary Excretion of Calcium

- **Renal failure** [1, 2]
- Hypoadrenocorticism
- Primary hyperparathyroidism
- Humoral hypercalcemia of malignancy
- Thiazide diuretics [1]

Increased Protein-Bound Calcium

- Hyperglobulinemia in multiple myeloma
- Hyperalbuminemia [1]

Iatrogenic Disorders

- Excessive calcium supplementation (Intravenous) [1]
- Excessive oral phosphate buffers [1]
- Calcipotriene [2]

Other or Unknown Mechanisms

- Hemoconcentration
- Hypothyroidism (juvenile onset) [1]
- Retained fetus and endometritis in dog (rare) [5]
- **Idiopathic hypercalcemia in cats** [6]

DECREASED

Hypoalbuminemia

- **Hypoproteinemia** [1, 2]

Primary Hypoparathyroidism

- Naturally acquired
- Post-thyroidectomy [1]

Pseudo-Hypoparathyroidism

- Decreased PTH receptor responsiveness [1]

Hypovitaminosis D

- Chronic renal disease or failure
- Protein-losing enteropathy in dogs
- Dietary vitamin D deficiency (rare) [1, 2]

Pregnancy, Parturient, or Lactational Hypocalcemia

- Postpartum / periparturient hypocalcemia / puerperal tetany
- **Eclampsia** [1, 2]

Hypercalcitonism

- Thyroid C-cell neoplasia
- Iatrogenic (calcitonin therapy) [1]

Excess Urinary Eexcretion of Calcium

- **Ethylene glycol toxicosis** (dogs and cats)
- Intravenous HCO_3^- infusions
- Furosemide treatment [1]

Calcium binding anticoagulants

- EDTA, citrate, oxalate (in vivo or in vitro) [1]

Other or unknown mechanisms

- Exocrine pancreatic insufficiency (dogs) [1, 2]
- Vitamin D-receptor defect rickets [1]
- Nutritional hypocalcemia (rare) [1]
- Oxalate toxicity [2]
- Tetracycline administration [1]
- Calcium deposition during fracture healing
- **Acute pancreatitis in dogs and cats**

NB. Common differential diagnoses are in bold.

Ref.

CALCIUM (CA)

Other or Unknown Mechanisms continued

- **Urinary tract obstruction**
- Acute and chronic renal failure
- Phosphate enema
- Sepsis
- Acute tumor lysis syndrome
- Nutritional secondary hyperparathyroidism [1, 2]

ARTIFACT

- Total Ca is falsely increased by lipemia and hemolysis [7]
- Total Ca can be decreased by marked bilirubinemia [7]
- Prolonged occlusion during phlebotomy may mildly increase Ca [7]
- Use of an inappropriate anticoagulant (EDTA, citrate anticoagulants) may cause falsely decreased results [7]

INTERPRET CA WITH:

- Albumin
- Ionized Calcium
- Phosphate
- Blood Urea Nitrogen
- Creatinine
- Urinalysis

Note: Frequency of hypercalcemia due to listed neoplastic processes may differ by species and/or breed

NB. Common differential diagnoses are in bold.

CHLORIDE (Cl^-)

INCREASED

Inadequate Water Intake
- **Water deprivation**
- Defective thirst response (hypothalamic defect)

Water Loss
- Pure Water Loss
  - **Insensible loss: Panting, hyperventilation, or fever**
  - Diabetes Insipidus
- Renal
  - Osmotic diuresis
  - Proximal renal tubular acidosis
  - Hypoadrenocorticism

Excessive Loss of Sodium Relative to Chloride
- **Small bowel diarrhea**

Gastrointestinal (GI) Loss/ Sequestration of Cl^-/HCl
- **Vomiting (biliary, pancreatic fluids)**
  - Upper GI obstruction
- **Secretory (osmotic) diarrhea**
- Osmotic sequestration
- Phosphate enema[1]

Excessive Gain of Chloride Relative to Sodium
- Salt poisoning
- Fluid therapy (e.g. 0.9% sodium chloride, hypertonic saline, potassium chloride-supplemented fluids)
- Therapy with chloride salts: KCl, or NH_4Cl [2]

Decreased Renal Excretion of Na and Cl
- Hyperaldosteronism[1]

Alimentary Loss of Bicarbonate
- **Bicarbonate loss / Small bowel diarrhea**
- **GI loss / sequestration (diarrhea)**

Renal Chloride Retention
- **Renal failure**
- Proximal renal tubular acidosis
- Distal renal tubular acidosis
- **Diabetes mellitus**[3]
- Hypoadrenocorticism[3]
- Drug-induced: acetazolamide, spironolactone[3]
- Compensatory response to chronic respiratory alkalosis
  - Hyperventilation or hypocapnea
  - Hypoxemia
  - Primary pulmonary disease
  - Pain[2]

Bicarbonate Consumption (Titration Acidosis)
- **Lactic acidosis**
- **Ketosis (diabetes mellitus)**
- Decreased excretion of non-carbonic acid:
  - Sulfates
  - Phosphates
  - **Renal failure**
  - **Toxicity (ethylene glycol, salicylate, methanol)**[1]

DECREASED

Gastrointestinal
- Loss or sequestration of chloride rich fluid
  - **Vomiting / diarrhea**
  - **Sequestration**
  - **Pyloric obstruction**
  - **Gastric rupture**
  - **Gastric Dilation-Volvulus**
  - Trichuriasis

Renal Loss
- Hypoadrenocorticism
- Osmotic diuresis (diabetes mellitus)
- Proximal renal tubular dysfunction (prolonged diuresis)
- Hypoaldosteronism
- Hyperadrenocorticism[3]
- Glucocorticoid administration[3]
- Ketonuria
- Sodium-wasting nephropathies
- Compensatory response to chronic respiratory acidosis
- Furosemide therapy
- Thiazide therapy

Third Space Losses
- Pancreatitis
- Peritonitis
- Uroabdomen
- Chylothorax with repeated pleural fluid drainage[2]
- Acute internal hemorrhage
- Acute exudation
- Cutaneous (sweating)

Metabolic Acidosis
- **Ketoacidosis**
- **Lactic acidosis**
- **Ingestion of foreign substances generating strong anions (ethylene glycol)**

Edematous Disorders
- Congestive heart failure
- Hepatic disease / hepatic cirrhosis
- Nephrotic syndrome
- Advanced renal failure[1]

Expanded Extracellular Fluid Volume (without edema)
- Excess sodium-poor fluid administration (parenteral)[1]
  - Fluid therapy with 5 % dextrose, 0.45 % saline solution, or hypotonic fluids[2]
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- Antidiuretic drugs (e.g. heparin solutions containing chlorbutol, vincristine, cyclophosphamide, nonsteroidal anti-inflammatory drugs)[2]

NB. Common differential diagnoses are in bold.

Ref.

CHLORIDE (CL)

Expanded Extracellular Fluid Volume Continued
- Myxedema coma of hypothyroidism (rare)[2]
- Psychogenic polydipsia[2]

Extracellular Translocation of Water
- Hyperglycemia
- Mannitol infusion (intravenously)[1]

Intracellular Translocation of Na (Cl follows)
- Hypokalemia (to maintain the intracellular electronegativity)
- Acute muscle injury[1]

Extravascular Fluid Translocation of Na (Cl follows)
- Uroperitoneum (ruptured bladder, or abdominal urethra)[1]

ARTIFACT
- Lipemia causing pseudo-hypochloremia (ion-exclusion effect when using the titrimetric methods) or causing pseudo-hyperchloremia (using the colorimetric method)[2]
- Potassium bromide therapy will falsely increase the reported chloride concentration (common and important)[2]
- Hyperviscosity may cause problems in analyzers that dilute samples before analysis[2]

INTERPRET CHLORIDE WITH:
- Electrolytes (Na^+, K^+)
- Urinalysis
- Total carbon dioxide / Bicarbonate
- Anion gap
- Acid-base analysis

NB. Common differential diagnoses are in bold.

Ref.

CHOLESTEROL (CHOL)

INCREASED

Postprandial Hypercholesterolemia [1, 2, 5]

Secondary Hypercholesterolemia

- **Hypothyroidism**
- **Diabetes mellitus** [1, 5]
- **Nephrotic syndrome or protein-losing nephropathy** [1, 2, 5]
- **Cholestasis** [1, 5]
- **Acute pancreatitis** [3, 5]
- **Hyperadrenocorticism or excess iatrogenic glucocorticoids**
- Hepatic Insufficiency [2]

Primary Hypercholesterolemia

- Idiopathic hyperlipoproteinemia (Miniature Schnauzers and other breeds) [1, 2, 5]
- Hypercholesterolemia in Briards (dog) [5]
- Idiopathic hyper-chylomicronemia (cat) [2]
- Lipoprotein lipase deficiency (cat) [1, 2, 5]
- Idiopathic hypercholesterolemia [1, 2]

Drug Induced Hypercholesterolemia

- Megestrol acetate (cat) [2]
- Glucocorticoids [2]

DECREASED

Severe Malnutrition [5]

Malabsorption / Maldigestion

- Protein-losing enteropathy [2]
- Lymphangiectasia [5]
- Exocrine pancreatic insufficiency [5]

Decreased Cholesterol Production

- Portosystemic shunt
- Chronic liver disease [1]
- Liver failure [5]

Altered Metabolism

- Inflammatory cytokines [1]

Increased Lipoprotein Uptake

- Rapidly proliferating neoplastic cells (histiocytic sarcoma, multiple myeloma) [3, 4]

Other Causes of Likely Multiple Mechanism

- Hypoadrenocorticism [5]

ARTIFACT

- Hemolysis and hyperproteinemia artifactually increase results [5]
- Bilirubin and ascorbic acid negatively interfere with enzymatic assays [5]
- Postprandial cholesterol increase may be mistaken for metabolic disease [5]

INTERPRET CHOLESTEROL WITH:

- Glucose
- Bood urea nitrogen
- Creatinine
- Hepatic enzymes
- Bilirubin
- Triglycerides
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

CREATINE KINASE (CK)

Also known as Creatine Phosphokinase (CPK)

INCREASED

SKELETAL MUSCLE DAMAGE

Degenerative

- **Hypoxia caused by exertion or seizures, exertional Rhabdomyolisis, saddle thrombus** [1, 2]

Neoplastic

- Metastatic neoplasia with striated muscle involvement

Nutritional

- White muscle disease (vitamin E-selenium deficiency), polioencephalomalacia [1, 2]

Inflammatory

- Myositis caused by *Neospora*, *Toxoplasma*, bacteria or other agents [1]

Toxic

- Monensin, ricin (castor bean), mycotoxin, snake-bite [1]

Traumatic

- **Intramuscular injections, hit by car, prolonged recumbency, seizures, exertion, post-surgical** [1]

Inherited

- Muscular dystrophy (Cavalier King Charles Spaniel dystrophin-deficient muscular dystrophy)
- Hyperkalemic periodic paralysis
- Malignant hyperthermia [1]

Other pathologies with uncertain pathogeneses

- Critically ill anorectic cat [3]

DECREASED

Not clinically significant

ARTIFACT

- May increase the measured CK activity
  - Hemolysis [1, 2]
  - Muscle penetration during venipuncture [1]
  - Underfilling of lithium heparin tube [4]

NB. Common differential diagnoses are in bold.

Ref.

CREATININE (CRE)

INCREASED

Pre-Renal Conditions

- Hypovolemia
  - **Dehydration**
  - **Hypoadrenocorticism**
  - **Shock**
  - **Blood loss**
- Decreased cardiac output
  - **Cardiac insufficiency**
  - **Shock**
  - **Hypoadrenocorticism**
- Shock
  - **Hypovolemic**
  - **Cardiogenic**
  - **Anaphylactic**
  - **Septic**
  - **Neurogenic** [1]

Renal Conditions

- Inflammatory
  - **Glomerulonephritis**
  - **Pyelonephritis**
  - Tubular-interstitial nephritis
  - Amyloidosis
- Toxic nephrosis
  - **Hypercalcemia**
  - **Ethylene glycol intoxication**
  - Myoglobin
  - Aminoglycosides
  - **NSAID intoxication**
- Renal ischemia or hypoxia
  - **Poor renal perfusion**
  - **Infarction**
- Congenital hypoplasia or aplasia
- Hydronephrosis
- Neoplasia (renal or metastatic) [1, 2]

Post-Renal Conditions

- **Urolithiasis**
- **Urethral plugs in cats**
- Neoplasia
- Prostatic disease
- **Leakage of urine from the urinary tract within the abdominal cavity: trauma, neoplasia** [1, 2]

Physiologic Increase

- **Heavily-muscled dogs (Greyhounds)** [1]
- Post-protein meal [1]

DECREASED

Not a clinically significant finding

Physiologic

- **Young dogs** [3]
- **Small breed dog** [4]
- **Decreased muscle mass** [1, 2]

Decreased Production

- Starvation
- Cachexia
- Hepatic insufficiency: hepatocellular disease
- Portosystemic shunts (congenital or acquired) [5]

ARTIFACT

- Presence of acetoacetate, glucose, vitamin C, uric acid, pyruvate, cephalosporins and amino acids in the sample [1]
- Lidocaine: increases values (dry chemistries) [6]
- Nitrofurantoin: increases values (Jaffe reaction) [6]
- Cefoxitin: increases values (Jaffe reaction) [6]
- Dobutamine: decreases values [6]
- Proline from hyperalimentation fluids: increases values [6]

INTERPRET CREATININE WITH:

- Blood urea Nitrogen
- Hepatic enzyme activity
- Creatine kinase
- Lactate dehydrogenase
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

GAMMA-GLUTAMYLTRANSFERASE (GGT)

INCREASED

Biliary Tract Abnormalities (same as Alkaline Phosphatase, ALP)[1, 2]

Hepatic Parenchyma Disease / Condition

- Degenerative, metabolic, inflammatory, neoplastic (same as ALP)[1, 2]

Induction by Drugs or Hormones

- Corticosteroids endogenous or exogenous (dog)[1]
- Phenobarbital, Phenytoin, Primidone[1]

DECREASED

- Not clinically significant

ARTIFACT

- Hemolysis or icterus may decrease the measured GGT activity[3]
- Underfilling of lithium heparin tube may increase GGT activity[4]

INTERPRET GGT WITH:

- Hepatic enzymes
- Markers of cholestasis

NB. Common differential diagnoses are in bold.

GLOBULIN (GLOB)

Calculation using measured parameters (Total Protein and Albumin)

INCREASED

HEMOCONCENTRATION

- **Dehydration** [1, 2]

IATROGENIC

- **Corticosteroids (dogs)**

INFLAMMATION

- Acute phase reactant response
- Nephrotic syndrome (α-globulins) [1]
- Inflammation (acute, chronic)
  - Active liver disease
  - Nephrotic syndrome (β-globulins) [1]

POLYCLONAL GAMMOPATHY

Infections

- Bacterial
  - **Brucellosis**
  - **Pyoderma (suppurative dermatopathies)**
  - **Bacterial endocarditis** [1, 2]
  - **Rickettsial**
    - **Ehrlichiosis** [2]
- Viral
  - **Feline infectious peritonitis (FIP)**
  - **Feline immunodeficiency virus (FIV)**
  - **Feline leukemia virus (FeLV)** [1, 2]
- Fungal
  - Systemic fungal infections
    - **Blastomycosis**
    - **Histoplasmosis**
    - **Cryptococcosis**
    - **Coccidioidomycosis** [1, 2]
- Parasitic
  - **Dirofilariasis**
  - **Demodicosis**
  - **Scabies** [1, 2]

Immune-Mediated Disease

- Infections (immune complex)
  - **Feline cholangitis/ cholangiohepatitis**
  - **Pyometra** [2]
- Systemic lupus erythematosus (SLE)
  - Glomerulonephritis
  - Immune-mediated hemolytic anemia (IMHA), thrombocytopenia (IMT), and polyarthritis [2]
- Immune mediated hemolytic anemia (IMHA) [2] and Immune-mediated thrombocytopenia (IMT)- not because of SLE [2]
- Pemphigus complex, bullous pemphigoid [2]
- Rheumatoid arthritis [2]
- Neoplasia [1, 2]

MONOCLONAL GAMMOPATHY

Infection

- Ehrlichiosis
- Leishmaniosis
- Feline infectious peritonitis (rare)
- Idiopathic
- Benign monoclonal gammopathy [2]

Neoplasia

- **Multiple myeloma**
  - Macroglobulinemia
  - Lymphoma
  - Extramedullary plasmacytoma (rare)
  - Chronic lymphocytic leukemia
  - Waldenström's macroglobulinemia [2]

Miscellaneous

- Cutaneous amyloidosis [3]
- Lymphocytic-plasmacytic gastroenterocolitis

DECREASED

- **Blood Loss (hemorrhage)** [2]
- **Protein Losing Enteropathy (PLE)** [2]
- Acquired
  - Chemotherapy, radiation therapy or other compounds (e.g. toxins, drugs)
- Inherited
  - IgM deficiency (Dobermans) [1]
  - Primary severe combined immunodeficiency (Jack Russell Terrier, Basset Hounds, Cardigan Welsh Corgis, Dachshunds) [1, 3]
  - IgA deficiency (Shar Pei, Beagle, Airedale Terriers, and German Shepherd Dogs) [1]
  - Transient hypogammaglobulinemia (dogs) [1]
- Infectious
  - Viral: FeLV, FIV, canine parvovirus, canine distemper virus
- Plasma Loss
  - Intestinal mucosa (protein-losing enteropathy- PLE) [2]
  - Markedly damaged glomeruli (protein losing nephropathy- PLN) [2]
  - Skin (plasma exudation)

INTERPRET GLOBULINS WITH:

- Albumin
- Packed cell volume
- Leukogram

NB. Common differential diagnoses are in bold.

Ref.

GLUCOSE (GLU)

INCREASED

Physiologic
- **Post-prandial**
- **Excitement, fright**
- **Steroid-associated**
- **Diestrus / pregnancy** [1, 2]

Type 1 Diabetes Mellitus
- **Idiopathic diabetes mellitus**
- Immune-mediated diabetes mellitus [1]

Type 2 Diabetes Mellitus
- **Pancreatic insular amyloidosis (mostly cats)**
- Obesity

Primary Pancreatic Condition
- **Pancreatitis**
- **Pancreatic carcinoma** [1]

Endocrine
- **Hyperadrenocorticism**
- Glucagonoma
- Acromegaly
- Hyperpituitarism
- Hyperthyroidism (transient, cats)
- Hypothyroidism (dogs)
- Pheochromocytoma
- Hepatocutaneous syndrome (dogs) [1]

Infectious diabetes mellitus
- Sepsis (initial phase, transient) [1]

Other
- Anti-insulin antibodies [1]

Pharmacologic or Toxicological Hyperglycemia (Iatrogenic)
- **Intravenous glucose administration**
- **Steroids (glucocorticoids)**
- Megestrol acetate
- Ketamine [1, 2]
- Glucagon
- Thyroxine
- **Ethylene glycol**
- Alpha-2 agonists (xylazine, detomidine, medetomidine, dexmedetomidine)
- Propanolol
- **Insulin (Somogyi effect)**
- Morphine
- Progestins [1]

Trauma
- Head trauma [2]

DECREASED

Increased Insulin Secretion
- **Pancreatic β-cell neoplasia (insulinoma)**
- **Xylitol toxicosis (dogs)** [2]

Decreased Insulin Antagonists
- **Hypoadrenocorticism (decreased cortisol)**
- Growth hormone deficiency
- Hypopituitarism [1]

Decreased Gluconeogenesis
- **Hepatic insufficiency / failure (acquired, congenital)**
- Porto-systemic shunt
- **Hypoadrenocorticism (decreased cortisol)**
- Starvation, malabsorption and severe malnutrition [1, 2]

Decreased Glycogenolysis
- Glycogen storage diseases (rare) (e.g. Pompe's disease, von Gierke's disease) [1]

Increased Glucose Utilization
- Exertional hypoglycemia ("hunting dog hypoglycemia") [1, 2]

Pharmacologic or Toxicologic Hypoglycemia
- **Insulin therapy**
- Sulfonylurea compounds (glipizide, glyburide)
- Ethanol [1, 2]

Uncertain Pathogeneses
- **Sepsis (especially with endotoxemia)** [1]
- Non- β-cell neoplasia neoplasms
  - Hepatocellular carcinoma, hepatoma, leiomyosarcoma, leiomyoma, hemangiosarcoma etc. [2]
- Pregnancy hypoglycemia
- Malonic aciduria (Maltese dogs) [1]
- Idiopathic hypoglycemia
  - Neonatal hypoglycemia
  - Juvenile hypoglycemia (especially toy breeds) [2]

ARTIFACT
- Delayed analysis of blood sample / failure to remove serum or plasma from blood cells can cause a decrease in glucose concentration [3]
- Sample bacterial contamination / bacteremia (parasitemia) can cause a decrease in glucose concentration (increased glycolysis) [4]
- Extreme leukocytosis can cause a decrease in glucose concentration (increased glycolysis) [1, 2]

...

NB. Common differential diagnoses are in bold.

Ref.

GLUCOSE (GLU)

- Extreme erythrocytosis can cause a decrease in glucose concentration (increased glycolysis)[1]
- Bromide (KBr) interference with glucose in some of the oxidase activity used to measure glucose concentration[1]
- Stressed / struggling patients, particularly cats during sample collection may have transient hyperglycemia[5]

INTERPRET GLUCOSE CONCENTRATION WITH:

- Ketones (serum, urine)
- Hepatic enzymes serum activity
- Urinalysis
- Fructosamine

NB. Common differential diagnoses are in bold.

PHOSPHORUS (PHOS)

INCREASED

Physiologic
- **Post-prandial**
- **Young fast-growing dog (generally large breed dog)** [1, 2]

Renal (Decreased Renal PHOS Excretion)
- **Decreased glomerular filtration rate (GFR)**
- **Pre-renal azotemia**
- **Renal failure**
- **Post-renal obstruction**
- Urinary bladder rupture or urine leakage into tissues
- Decreased parathyroid hormone (PTH) concentration or activity (hypoparathyroidism)
- Hyperthyroidism
- Acromegaly [1, 2]

Intestinal (Increased Intestinal PHOS Absorption)
- Increased vitamin D
  - Ingestion of cholecalciferol rodenticides and plants (e.g. *Cestrum diurnum*, *Solanum sp.*)
- Phosphate enema or ingestion of phosphate urinary acidifier
- Ischemic intestinal lesions (shift intracellular fluid to extracellular fluid)
- Granulomatous disease
  - Fungal, parasitic
- Humoral hypercalcemia of malignancy
- Diet with a low calcium / phosphorus ratio (rare) [1]

Bone
- Osteolytic bone lesions (neoplasia) [2]

Other or Unknown Mechanisms
- Malignant hyperthermia [1, 2]
- Acute tumor lysis syndrome [1, 2]
- Hyperthyroidism in cats
- Metabolic acidosis
- Hyperadrenocorticism in dogs [1, 2]

DECREASED

Renal (Increased Renal PHOS Excretion)
- Prolonged diuresis [1]
- Increased PTH or PTHrp activity
- Primary hyperparathyroidism (parathyroid neoplasia) [1, 2]
- Humoral hypercalcemia of malignancy [1, 2]
- Eclampsia
- Fanconi syndrome (dogs) [1]
- Hyperadrenocorticism / iatrogenic steroid administration [2, 3]

Intestinal (Decreased Intestinal PHOS Absorption)
- **Prolonged anorexia or phosphorus-deficient diet**
- Vomiting / diarrhea
- Phosphorus-binding agents
- Hypovitaminosis D
- Intestinal malabsorption / steatorrhea [1, 2]

Increased Loss
- Vomiting / diarrhea
- Diabetes
- Diabetes ketoacidosis [3]

Defective Mobilization of Phosphorus From Bone
- Puerperal tetany
- Eclampsia [1]

Iatrogenic
- **Treatment of diabetes ketoacidosis** [3]
- Glucose infusion [1]
- Sodium bicarbonate administration
- Parenteral glucose administration
- Aggressive parenteral fluid therapy [2]

Other Causes
- Respiratory alkalosis [1]
- Monoclonal gammopathy [4]
- Hyperinsulinism (endogenous or exogenous)
- Hepatic lipidosis (cats)
- Re-feeding syndrome [3]

ARTIFACT
- Drugs or Substances That May Cause Increased Serum PHOS [5]
  - Bilirubin (icterus)
  - Hemoglobin (hemolysis)
  - Lipemia
  - Aminosalicylic acid
  - Detergents contaminating glassware
  - Fat emulsions

NB. Common differential diagnoses are in bold.

Ref.

PHOSPHORUS (PHOS)

- Drugs or Substances That May Cause Increased Serum PHOS[5] continued
  - Methotrexate
  - Naproxen
  - Rifampin

- Drugs or Substances That May Cause Decreased Serum PHOS[5]
  - Phenothiazine
  - Cefotaxime
  - Citrates
  - Mannitol
  - Oxalate
  - Promethazine

INTERPRET PHOSPHORUS CONCENTRATION WITH:

- Calcium
- Blood Urea Nitrogen
- Creatinine
- Urinalysis

NB. Common differential diagnoses are in bold.

POTASSIUM (K^+)

INCREASED

Decreased Renal Excretion

- **Urinary tract obstruction**
- **Ruptured bladder / ureter (uroabdomen)**
- **Renal insufficiency or failure (primarily oliguric or anuric patients)**
- Hypoaldosteronism
  - **Hypoadrenocorticism (pathologic)**
  - Angiotensin-converting enzyme inhibitors (iatrogenic)
  - Hyporeninemic hypoaldosteronism (with diabetes or renal failure (rare)[1]

Increased Intake

- Administration of potassium-rich fluid

Drugs

- Angiotensin-converting enzyme inhibitors (e.g. enalapril)
- Potassium-sparing diuretics (e.g. spironolactone, amiloride, triamterene)
- Prostaglandin inhibitors
- Heparin
- Non-specific beta blockers (e.g. propranolol)[2]

Metabolic

- **Metabolic acidosis by accumulation of inorganic acid (NH_4Cl, HCl etc.)**[3]

Hypertonicity

- **Diabetes mellitus**
- Mannitol infusion
- Massive intravascular hemolysis with potassium-rich erythrocytes
- Massive tissue damage
  - Acute tumor lysis syndrome
  - Reperfusion of extremities after aortic thromboembolism in cats with cardiomyopathy
  - Crush injuries[2]
  - Hyperkalemic myopathy (Rhabdomyolysis or other muscle damage)[1]

Other / Unknown Mechanism

- Repeated drainage of chylous thoracic effusions[2]
- Peritoneal effusions in cats[2]

Iatrogenic

- IV fluids or IV fluid line contamination with potassium supplementation
- Use of the wrong anticoagulant (K^+ EDTA)

DECREASED

Decreased Intake

- **Anorexia**

Gastrointestinal Loss

- **Vomiting or sequestration of H^+ and Cl^- causing metabolic alkalosis**
- **Diarrhea**[1]

Renal Loss

- **Chronic renal failure in cats**
- **Post-obstructive diuresis**
- Increased fluid flow in distal nephron (collecting tubule)
  - **Osmotic (diabetes)**
  - **Sodium- losing nephropathies**[1]

Increased renal excretion of anions

- **Ketonuria**
- **Lactaturia**
- **Bicarbonaturia**
- Distal (type I) renal tubular acidosis (rare)[2]
- Proximal (type II) renal tubular acidosis after $NaHCO_3^-$ treatment (rare)[2]
- Hyperaldosteronism (primary)[1]

Drugs

- **Loop diuretics (e.g. furosemide)**
- Thiazide diuretics (e.g. chlorothiazide, hydrochlorothiazide)
- Amphotericin B
- Penicillins (rare)
- Albuterol overdose (rare)[2]
- Inadequate fluid therapy
  - Inadequate potassium supplementation
  - Potassium-free fluids (e.g. 0.9% NaCl, 5% dextrose in water)[2]
- **Glucose-containing fluids ± insulin**[2]

Other and Unknown Mechanisms

- **Metabolic / respiratory alkalosis with alkalemia**[2]
- Catecholamine release[2]
- Endotoxemia[1]
- Third space loss (body cavity effusion)[1]
- **Hypokalemic renal failure in cats**
- Hypokalemic myopathy of Burmese kittens

NB. Common differential diagnoses are in bold.

Ref.

POTASSIUM (K)

ARTIFACT

- Lipemia may cause a decreased measured K^+ concentration (ionic exclusion phenomenon)[3]

- Serum K^+ is slightly higher than plasma K^+ of healthy animals
  - Release of K^+ from platelets during clotting[2]

- Elevations in K^+ levels
  - Potassium oxalate or K_2 or K_3 EDTA anticoagulants[3]
  - Severe bilirubinemia: slight increase (ion-selective electrodes)[2]
  - Marked thrombocytosis[1, 2]
  - Marked leukocytosis (physiologic or neoplastic)[3]
  - In vitro hemolysis of K-rich erythrocytes in the Akita and Shiba Inu breeds[2]
  - In vivo hemolysis in phosphofructokinase deficiency in predisposed canine breeds[2, 3]
    - English Springer Spaniels, American Cocker Spaniels

INTERPRET POTASSIUM CONCENTRATION WITH:

- Electrolytes
- Blood Urea Nitrogen
- Creatinine
- Total Carbon Dioxide
- Anion gap
- Acid-Base analysis
- Urinalysis

NB. Common differential diagnoses are in bold.

SODIUM (Na^+)

INCREASED

Inadequate Water Intake
- **Water deprivation (inadequate access to water)**
- Primary adipsia / hypodipsia (defective thirst response secondary to hypothalamic disease or lesion)[1]

Excess Pure Water Loss
- Diabetes insipidus
- Central
- Nephrogenic
- **Panting**
- **Fever**
- **Hyperventilation**[1]

Renal Water Loss
- Osmotic
- **Diabetes mellitus**
- Mannitol infusion
- Chemical diuresis (pharmacologic)
- **Chronic renal failure**
- Post-obstructive diuresis
- Non-oliguric renal failure[2]

Extra-Renal Water Loss
- **Vomiting**
- **Osmotic diarrhea**
- **Osmotic sequestration (small intestinal obstruction)**
- Third space water losses
- **Peritonitis, pancreatitis, cavitary effusions**[2]
- Cutaneous water losses
- Burn lesions

Excess Intake of Sodium
- Salt poisoning (with concurrent water deprivation)
- Administration of hypertonic fluid
  - Hypertonic saline
  - Sodium bicarbonate
  - Parenteral nutrition
  - Sodium phosphate enema

Increased Renal Sodium Retention
- Hyperaldosteronism[2]

Other / Unknown Mechanism
- Hyperadrenocorticism[2]

Therapeutics
- Administration of hypertonic saline or sodium bicarbonate

DECREASED

Gastrointestinal Sodium Loss
- **Vomiting / diarrhea**
- **Sequestration**
- Canine whipworm infestation[1]

Renal Sodium Loss
- **Hypoadrenocorticism**
- **Osmotic diuresis (diabetes mellitus)**
- Proximal renal tubule dysfunction (prolonged diuresis)
- **Hypoaldosteronism**
- **Ketonuria**
- **Sodium-wasting nephropathies**[1]

Third Space Sodium Loss
- **Pancreatitis**
- **Peritonitis**
- **Uroabdomen**
- Chylothorax with repeated pleural fluid drainage[2]
- Acute internal hemorrhage or acute exudation[1,2]

Cutaneous Loss
- Sweating
- Exudative skin lesions

Edematous Disorders
- **Congestive heart failure causing ascites**
- Hepatic disease / hepatic cirrhosis causing ascites
- Nephrotic syndrome causing effusion
- Advanced renal failure (primarily oliguric or anuric)[2]

Expanded Extracellular Fluid Volume (Without Edema)
- Excess sodium-poor fluid administration (parenteral)
- Inappropriate fluid therapy with 5% dextrose, 0.45% saline solution, or hypotonic fluids[2]
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)[1,2]
- Antidiuretic drugs (e.g. heparin solutions containing chlorbutol, vincristine, cyclophosphamide, nonsteroidal anti-inflammatory drugs)[2]
- Myxedema coma of hypothyroidism (rare)[2]
- Psychogenic polydipsia[2]

Extracellular Translocation of Water
- **Hyperglycemia**
- Mannitol infusion (intravenously)[1]

Intracellular Translocation of Sodium
- Hypokalemia
- Acute muscle injury[1]

Extravascular Fluid Translocation of Sodium
- **Uroperitoneum (ruptured bladder or abdominal urethra)**[1]

NB. Common differential diagnoses are in bold.

Ref.

SODIUM (Na)

ARTIFACT

- Lipemia may cause pseudo-hyponatremia (ion-exclusion effect)[2]
- Marked hyperproteinemia may cause a false decrease in measured sodium[2]
- Hemolysis may cause a decreased sodium concentration
- Sample dehydration may cause artefactually increased sodium[1]
- Anticoagulant: Na_2EDTA will increase the Na plasma concentration
  - Use of Na-Heparin will not cause clinically relevant changes in heparinized plasma Na[1]

INTERPRET THE SODIUM WITH:

- Electrolytes (K^+, Cl^-)
- Total protein
- Blood Urea Nitrogen
- Creatinine
- Osmolality
- Urinalysis
- Hematocrit

NB. Common differential diagnoses are in bold.

Ref.

THYROXINE (Total T4 / TT4)

INCREASED

Hyperthyroidism

- **Thyroid adenoma/ adenomatous (common in cats, rare in dogs)** [1, 2, 3]
- Thyroid carcinoma / adenocarcinoma (cats, rare in dogs) [1, 2, 3]
- Hyperplasia [1]
- Multiple endocrine neoplasia (Type II) [1]

Drugs (Dogs) [2]

- Amiodarone
- Ipodate
- Overdose of levothyroxine supplementation [1]

DECREASED

Primary Hypothyroidism (Dogs)

- **Lymphocytic thyroiditis**
- **Idiopathic thyroid atrophy**
- **Congenital thyroid gland dysgenesis**
- **Destruction of thyroid gland (neoplasia, surgery, radioactive iodide treatments, etc** [1, 2, 3]

Secondary Hypothyroidism

- Hyperadrenocorticism [1]
- Pituitary failure
- May be normal for some breeds

Defective Thyroxine Production

- Iodine organification defect [1]
- Congenital thyroid peroxidase deficiency in Toy Fox Terriers [1]
- Iodine deficiency [1]

Non-Thyroidal Illnesses: "Euthyroid Sick Syndrome" [2]

- **Acute diseases**
  - **Bacterial bronchopneumonia**
  - **Sepsis**
  - **Distemper**
  - **Autoimmune hemolytic anemia**
  - **Systemic lupus erythematosus**
  - **Intervertebral disk disease**
  - **Polyradiculoneuritis**
  - **Acute renal failure**
  - **Acute hepatitis**
  - **Acute pancreatitis**
- **Chronic diseases**
  - **Generalized demodicosis**
  - **Generalized bacterial furunculosis**
  - **Systemic mycoses**
  - **Lymphoma**
  - **Chronic renal failure**
  - **Diabetes mellitus**
  - **Congestive heart failure**
  - **Cardiomyopathy**
  - **Chronic hepatitis, cirrhosis**
  - **Gastrointestinal disturbances**
  - **Megaesophagus**

Drugs

- Dogs [2]
  - Aspirin
  - Carprofen
  - Glucocorticoids
  - Clomipramine
  - Furosemide
  - Methimazole
  - Phenobarbital
  - Phenylbutazone
  - Progestagens
  - Propylthiouracil
  - Sulfonamides
- Cats
  - Following radioactive iodine or methimazole therapy for hyperthyroidism [1]

VARIABLES THAT MAY AFFECT BASELINE T4 IN DOGS

Age	Inversely proportional effect [2]
Neonate (< 3 mo)	Increased T4
Aged (> 6 yr)	Decreased T4
Body size	Inversely proportional effect [2]
Small (< 10 kg)	Increased T4
Large (> 30 kg)	Decreased T4
Breed	
Sight Hounds (e.g. Greyhound)	T4 may be lower than normal range established for dogs [2]
Nordic breeds (e.g. Huskies)	

NB. Common differential diagnoses are in bold.

Ref.

THYROXINE (TT4)

Strenuous exercise [2] ..... Increased T4

Pregnancy (progesterone) [2] ..... Increased T4

Surgery/anesthesia [2] ..... Decreased T4

Concurrent illness (Euthyroid Syndrome) [2] ..... Decreased T4

ARTIFACT

- Presence of anti-T4 autoantibodies can cause an artefactual increase in T4 (radioimmunoassay) (dogs) [2]
- Administration of compound containing iodine

INTERPRET THYROXINE WITH:

- Cholesterol
- Free T4
- Thyroid Stimulating Hormone (canine)
- Alanine Aminotransferase (feline)

NB. Common differential diagnoses are in bold.

TOTAL BILIRUBIN (TBIL)

INCREASED

HEMOLYSIS
(not an extensive list)

Immunological Alterations
- **Immune Mediated Hemolytic Anemia (IMHA)** [1, 2]
- **Blood transfusion reaction**
- Drug induced hemolytic anemia
- Vaccine associated [1, 2]

Infectious
- ***Mycoplasma haemofelis***
- ***Mycoplasma haemocanis***
- ***Babesia canis***
- ***Cytauxzoon felis***
- ***Anaplasma spp.***
- **Feline leukemia virus (FeLV)**
- **Bacteremia** [1]

Toxins
- Lead poisoning
- Zinc / copper toxicity [1, 2]

Oxidative Damage
- **Heinz body anemia**
- Methemoglobulinemia
- Eccentrocytic hemolysis (acquired or inherited) [1]

Defect in Adenosine Triphosphate (ATP) Generation
- Pyruvate kinase (PK) deficiency [1, 2]
- Phosphofructokinase deficiency [1, 2]
- Hypophosphatemic hemolyisis [2]

Erythrocyte Fragmentation in Blood
- **Disseminated intravascular coagulation**
- Vasculitis
- Microangiopathic disease
- **Hemangiosarcoma**
- Rheological processes
  - Caval syndrome of dirofilariasis
  - Cardiac valvular disease [1]

Other Unknown Pathogeneses
- Envenomation [1]
- Hemophagocytic histiocytic sarcoma [1]
- Increased osmotic fragility [1]
- Hereditary non-spherocytic hemolytic anemia of Beagles [1]
- Idiopathic hemolytic anemia of Abyssinian and Somali cats [1]

HEPATOBILIARY DISEASE

Infectious
- Viral
- Bacterial
- Systemic fungal infection

Cirrhosis
- **Chronic active hepatitis**

Metabolic or Endocrine
- **Hepatic lipidosis**
- **Diabetes mellitus**
- **Hyperadrenocorticism**
- **Hyperthyroidism**

Immunological
- **Chronic active hepatitis**
- **Feline lymphocytic or suppurative cholangiohepatitis**
- **Cirrhosis**

Neoplasia
- **Lymphoma**
- **Hepatocellular carcinoma**
- **Hepatoma**

Toxins
- Mushrooms
- Chemicals - insecticides, carbon tetrachloride
- Numerous pharmaceuticals
- Plants - aflatoxins, pyrrolizidine alkaloids, glycosides (sago palm)

Biliary Disorders
- **Cholangitis – (bile sludge)**
- **Gall bladder stones**
- **Pancreatic disease (extrahepatic biliary obstruction)**
- **Neoplasia (gallbladder, bile duct adenocarcinoma)**
- **Biliary mucocele**

Sepsis Associated Cholestasis
- *Escherichia coli, Staphylococcus intermedius* infection
  - Pneumonia
  - Peritonitis
  - Endocarditis
  - Urinary tract infection
  - Soft tissue infection

DECREASED

Not clinically significant but may be seen in bone marrow suppressive anemias

ARTIFACT

- Hemolysis or lipemia may artifactually increase values [3]
- Exposure to ultraviolet light (including sunlight) will decrease bilirubin levels [3]

INTERPRET BILIRUBIN WITH:

- Hepatic enzyme activity
- Creatine kinase
- Lactate dehydrogenase if available
- Complete blood count
- Hematocrit

NB. Common differential diagnoses are in bold.

Ref.

TOTAL CARBON DIOXIDE (tCO_2)

tCO_2 provides an indirect estimate of bicarbonate

INCREASED

Metabolic Alkalosis

- **Gastrointestinal loss / sequestration H^+ and Cl^-**
- **Vomiting, gastric atony / torsion / pyloric obstruction (functional or mechanical)**
- Contraction alkalosis secondary to vomiting[1]

Renal Loss of H^+

- **Hypokalemia**[1]
- Loop or thiazide diuretics (e.g. furosemide)[1]
- Renal compensation for respiratory acidosis (chronic)[1]

Iatrogenic

- **Administration of bicarbonate containing solutions**

DECREASED

Bicarbonate Consumption

(Titration Acidosis)

- **Lactic acidosis**
- **Ketoacidosis (diabetes mellitus)**
- **Decreased excretion of non-carbonic acid**
  - **Sulfates**
  - **Phosphates**
  - **Renal failure**
- **Toxicity (ethylene glycol, paraldehyde, salicylate, methanol)**[1]

Decreased Renal Excretion of H^+

- Proximal renal tubular acidosis
- Distal renal tubular acidosis (Type I)[1]
- **Hypoadrenocorticism**[1]
- Uroperitoneum and/or urinary tract obstruction[1]

Bicarbonate Loss

(Hyperchloremic Metabolic Acidosis)

- **Vomiting (biliary and/or pancreatic fluids)**
- **Diarrhea**
- **Sequestration or GI fluids**[1]

Compensation for Primary Respiratory Alkalosis

- Hyperventilation – hypoxemia
- Primary pulmonary disease
- Pain

Dilutional Acidosis

- Rapid infusion of saline

Iatrogenic

- Ammonium chloride administration

ARTIFACT

- Increased contact with room air may decrease tCO_2[2]
  - Underfilling the sample tube (heparin or plain tube)
  - Failure to properly cap the sample or repeatedly opening the tube
- Prolonged venous stasis will decrease tCO_2[2]
- Use of EDTA, oxalate, or fluoride anticoagulants will decrease tCO_2[2]
- Prolonged contact with the clot can cause decrease in tCO_2[2]

INTERPRET TCO_2 WITH:

- Acid-Base analysis
- Anion gap
- Electrolytes
- Glucose
- Blood Urea Nitrogen
- Creatinine
- Urinalysis

NB. Common differential diagnoses are in bold.

Ref.

TOTAL PROTEIN (TP)

Serum total protein concentration is a direct reflection of cumulative serum albumin and globulin values. Therefore, the value only gives an overview of the general state of protein homeostasis.[1, 2]

INCREASED

Decreased Blood Volume

- **Hemoconcentration – dehydration (most common cause)**[1, 2]

Increased Protein Production

- Inflammatory Disease
  - **Infectious**
    - **Bacterial**
    - **Viral**
    - **Fungal**
    - **Protozoal**[1]
  - **Noninfectious**
    - **Necrosis**
    - **Neoplasia**
    - **Immune-mediated disease**[1]

B-Lymphocyte Neoplasia

- Plasma cell (monoclonal gammopathy): multiple myeloma, plasmacytoma[1]
- Lymphocyte: lymphoma, lymphocytic leukemia[1]

DECREASED

Protein Loss

- **Blood loss**
- **Protein-losing nephropathy**
  - **Glomerulonephritis**
  - **Amyloidosis**
- Protein-losing enteropathy
  - **Small intestinal mucosal disease**
  - **Lymphangiectasia**
  - **Intestinal blood loss**
- Plasma loss (sequestration / third space losses)
  - **Peritonitis / pleuritis**
  - Vasculitis
  - Exudative dermatopathies[1, 2]

Decreased Protein Synthesis / Increased Protein Catabolism

- **Hepatic insufficiency**
- Malabsorption or maldigestion
  - **Intestinal mucosal disease**
  - **Exocrine pancreatic insufficiency (EPI)**[1, 2]
- Cachectic state
  - **Chronic disease**
  - **Neoplasia**
  - **Malnutrition**
  - **Starvation**
  - Lymphoid hypoplasia[1, 2]

Hemodilution

- Excess administration of intravenous fluid
- Edematous disorders
  - **Congestive heart failure**
  - Cirrhosis
  - Nephrotic syndrome
  - Excess ADH secretion (syndrome of inappropriate antidiuretic hormone secretion or SIADH)[1, 2]

ARTIFACT

- Hemolysis or gross lipemia may cause an increase in measured TP[1, 5]
- Icterus may falsely decrease the measured total protein value[1, 2, 3, 4, 5]
- Plasma > Serum (very slight difference)
  - Fibrinogen may slightly increase the plasma TP concentration[5]

INTERPRET TOTAL PROTEIN WITH:

- Albumin
- Globulin
- Blood Urea Nitrogen
- Liver enzymes
- Creatinine
- Hematology
- Urinalysis
- Urine Protein: Creatinine Ratio

NB. Common differential diagnoses are in bold.

Ref.

REFERENCES

Alanine Aminotransferase (ALT)

1. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 191-225. Saunders Elsevier.
3. Vaden SL, Knoll JS, Smith FWK, and Tilley, LP. (2009) Alanine Aminotransferase. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 32-33. Wiley-Blackwell.

Albumin (ALB)

1. Stockham SL, Scott MA. (2008) Proteins. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 369-413. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Immunologic and Plasma Protein Disorder. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 278-293. Saunders Elsevier.
3. Cooper ES, Wellman ML, Carsillo ME. Hyperalbuminemia associated with hepatocellular carcinoma in a dog. Vet Clin Pathol. 2009 Dec;38(4):516-20.
4. Torrente C, Manzanilla EG, Bosch L, Fresno L, Rivera Del Alamo M, Andaluz A, Saco Y, Ruiz de Gopegui R. Plasma iron, C-reactive protein, albumin, and plasma fibrinogen concentrations in dogs with systemic inflammatory response syndrome. J Vet Emerg Crit Care (San Antonio). 2015 Sep-Oct;25(5):611-9.
5. Stokol T, Tarrant JM, Scarlett JM. Overestimation of canine albumin concentration with the bromcresol green method in heparinized plasma samples. Vet Clin Pathol. 2001;30(4):170-176.
6. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Albumin. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 34-35. Wiley-Blackwell.

Alkaline Phosphatase (ALP)

1. Lawler DF, Keltner DG, Hoffman WE, Nachreiner RF, Hegstad RL, Herndon PA, Fischer BJ. Benign familial hyperphosphatasemia in Siberian Huskies. Am J Vet Res. 1996 May;57(5):612-7.
2. Gallagher AE, Panciera DL, Panciera RJ. Hyperphosphatasemia in Scottish Terriers: 7 cases. J Vet Intern Med. 2006 Mar-Apr;20(2):418-21.
3. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.
4. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 191-225. Saunders Elsevier.
5. Karayannopoulou M, Polizopoulou ZS, Koutinas AF, Fytianou A, Roubies N, Kaldrymidou E, Tsioli V, Patsikas MN, Constantinidis TC, Koutinas CK. Serum alkaline phosphatase isoenzyme activities in canine malignant mammary neoplasms with and without osseous transformation. Vet Clin Pathol. 2006 Sep;35(3):287-90.
6. Thomas JS. Introduction to Serum Chemistries: Artifacts in Biochemical Determinations. Small Animal Clinical Diagnosis by Laboratory Methods, Fourth Edition. 2004, 113-116.

Amylase (AMY)

1. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 191-225. Saunders Elsevier.
3. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Lipase. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 399-400. Wiley-Blackwell.

Aspartate Aminotransferase (AST)

1. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. Pp 191-225. Saunders Elsevier.
3. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Aspartate Aminotransferase. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 72-73. Wiley-Blackwell.

Bile Acids (BA)

1. Stockham SL, Scott MA. (2008) Liver Function. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 675-706. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 191-225. Saunders Elsevier.
3. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Bile Acids. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 91-93. Wiley-Blackwell.

Blood Urea Nitrogen (BUN)

1. Stockham SL, Scott MA. (2008) Urinary System. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 415-494. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Urinary Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 126-155. Saunders Elsevier.
3. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Urea Nitrogen. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 660-661. Wiley-Blackwell.

Calcium (Ca)

1. Stockham SL, Scott MA. (2008) Calcium, Phosphorous, Magnesium and their Regulatory Hormones. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 593-638. Wiley-Blackwell.
2. Willard MD, Tvedten H. (2012) Endocrine, Metabolic, and Lipid Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 156-190. Saunders Elsevier.
3. Bergman PJ. Paraneoplastic hypercalcemia. Topics Companion Anim Med. 2012 Nov;27(4):156-8.
4. Robat CS, Cesario L, Gaeta R, Miller M, Schrempp D, Chun R. Clinical features, treatment options, and outcome in dogs with thymoma: 116 cases (1999-2010). J Am Vet Med Assoc. 2013 Nov 15;243(10):1448-54.
5. Hirt RA, Kneissl S, Teinfalt M. Severe hypercalcemia in a dog with a retained

fetus and endometritis. J Am Vet Med Assoc. 2000 May 1;216(9):1423-5

6. Midkiff AM, Chew DJ, Randolph JF, Center SA, DiBartola SP. Idiopathic hypercalcemia in cats. J Vet Intern Med. 2000 Nov-Dec;14(6):619-26.

7. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Calcium. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 148-150. Wiley-Blackwell.

Chloride (Cl^-)

1. Stockham SL, Scott MA. (2008) Monovalent Electrolytes and Osmolality. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp495-558. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Electrolyte and Acid-Base Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 112-125. Saunders Elsevier.

3. DiBartola SP, Willard MD (2006) Disorders of Chloride: Hypochloremia and Hyperchloremia. In: Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice. pp 82-91. Ed. S DiBartola, Saunders Elsevier.

Cholesterol (CHOL)

1. Stockham SL, Scott MA. (2008) Lipids. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 763-782. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Endocrine, Metabolic, and Lipid Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 156-190. Saunders Elsevier.

3. Moore PF, Affolter VK, Vernau W. Canine hemophagocytic histiocytic sarcoma: a proliferative disorder of CD11d+ macrophages. Vet Pathol. 2006 Sep;43(5):632-45.

4. Patel RT, Caceres A, French AF, McManus PM. Multiple myeloma in 16 cats: a retrospective study. Vet Clin Pathol. 2005 Dec;34(4):341-52.

5. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Cholesterol. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 174-175. Wiley-Blackwell.

Creatine Kinase (CK)

1. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Neurologic Disorder. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 304-314. Saunders Elsevier.

3. Fascetti AJ, Mauldin GE, Mauldin GN. Correlation between serum creatine kinase activities and anorexia in cats. J Vet Intern Med. 1997 Jan-Feb;11(1):9-13.

4. Lippi G, Avanzini P, Cosmai M, Aloe R, Ernst D. Incomplete filling of lithium heparin tubes affects the activity of creatine kinase and gamma-glutamyltransferase. Br J Biomed Sci. 2012;69(2):67-70.

Creatinine (CRE)

1. Stockham SL, Scott MA. (2008) Urinary System. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 415-494. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Urinary Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 126-155. Saunders Elsevier.

3. Rørtveit R, Saevik BK, Eggertsdóttir AV, Skancke E, Lingaas F, Thoresen SI, Jansen JH. Age-related changes in hematologic and serum biochemical variables in dogs aged 16-60 days. Vet Clin Pathol. 2015 Mar;44(1):47-57.

4. Misbach C, Chetboul V, Concordet D, Médaille C, Gruet P, Speranza C, Hoffmann AC, Rocha A, Balouka D, Petit AM, Trehiou-Sechi E, Pouchelon JL, Lefebvre HP. Basal plasma concentrations of routine variables and packed cell volume in clinically healthy adult small-sized dogs: effect of breed, body weight, age, and gender, and establishment of reference intervals. Vet Clin Pathol. 2014 Sep;43(3):371-80.

5. Deppe TA, Center SA, Simpson KW, Erb HN, Randolph JF, Dykes NL, Yeager AE, Reynolds AJ. Glomerular filtration rate and renal volume in dogs with congenital portosystemic vascular anomalies before and after surgical ligation. J Vet Intern Med. 1999 Sep-Oct;13(5):465-71.

6. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Creatinine. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 198-199. Wiley-Blackwell.

Gamma-Glutamyltransferase (GGT)

1. Stockham SL, Scott MA. (2008) Enzymes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 639-674. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Gastrointestinal, Pancreatic, and Hepatic Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 191-225. Saunders Elsevier.

3. Ji, J. Z., & Meng, Q. H. (2011). Evaluation of the interference of hemoglobin, bilirubin, and lipids on Roche Cobas 6000 assays. Clinica Chimica Acta, 412(17–18), 1550–1553.

4. Lippi G, Avanzini P, Cosmai M, Aloe R, Ernst D. Incomplete filling of lithium heparin tubes affects the activity of creatine kinase and gamma-glutamyltransferase. Br J Biomed Sci. 2012;69(2):67-70.

Globulin (GLOB)

1. Stockham SL, Scott MA. (2008) Proteins. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 369-413. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Immunologic and Plasma Protein Disorder. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 278-293. Saunders Elsevier.

3. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Globulins. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 341-343. Wiley-Blackwell.

Glucose (GLU)

1. Stockham SL, Scott MA. (2008) Glucose, Ketoamines, and Related Regulatory Hormones. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 707-737. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Endocrine, Metabolic, and Lipid Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 156-190. Saunders Elsevier.

3. Collicutt NB, Garner B, Berghaus RD, Camus MS, Hart K. Effect of delayed serum separation and storage temperature on serum glucose concentration in horse, dog, alpaca, and sturgeon. Vet Clin Pathol. 2015 Mar;44(1):120-7.

4. Burkhard MJ, Garry F. Artifactual hypoglycemia associated with hemotrophic mycoplasma infection in a lamb. Vet Clin Pathol. 2004;33(4):244-8.

5. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Glucose. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 347-349. Wiley-Blackwell.

Phosphorus (PHOS)

1. Stockham SL, Scott MA. (2008) Calcium, Phosphorous, Magnesium and their regulatory hormones. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 593-638. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Endocrine, Metabolic, and Lipid Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 156-190. Saunders Elsevier.

3. DiBartola SP, Willard MD (2006) Disorders of Phosphorus: Hypophosphataemia and

Hyperphosphataemia. In: Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice. pp 195-209. Ed. S DiBartola, Saunders Elsevier.

4. Kristensen AT, Klausner JS, Weiss DJ, Schultz RD, Bell FW. Spurious hyperphosphatemia in a dog with chronic lymphocytic leukemia and an IgM monoclonal gammopathy. Vet Clin Pathol. 1991;20(2):45-48.

5. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Phosphorous. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 479-481. Wiley-Blackwell.

Potassium (K^+)

1. Stockham SL, Scott MA. (2008) Monovalent Electrolytes and Osmolality. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 495-558. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Electrolyte and Acid-Base Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 112-125. Saunders Elsevier.

3. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Potassium. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 492-494. Wiley-Blackwell.

Sodium (Na^+)

1. Stockham SL, Scott MA. (2008) Monovalent Electrolytes and Osmolality. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp495-558. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Electrolyte and Acid-Base Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 112-125. Saunders Elsevier.

Thyroxine (Total T4 / TT4)

1. Stockham SL, Scott MA. (2008) Thyroid Function. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 783-803. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Endocrine, Metabolic and Lipid Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 156-190. Saunders Elsevier.

3. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Thyroxine Total. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 624-626. Wiley-Blackwell.

Total Bilirubin (TBIL)

1. Stockham SL, Scott MA. (2008) Erythrocytes. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 110-221. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Erythrocyte Disorders. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. Pp 38-62. Saunders Elsevier.

3. Vaden SL, Knoll JS, Smith FWK, Tilley, LP. (2009) Bilirubin. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 94-95. Wiley-Blackwell.

Total Carbon Dioxide (TCO_2)

1. Stockham SL, Scott MA. (2008) Monovalent Electrolytes and Osmolality. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 495-558. Wiley-Blackwell.

2. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Bicarbonate. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 88-90. Wiley-Blackwell.

Total Protein (TP)

1. Stockham SL, Scott MA. (2008) Proteins. In: Fundamentals of Veterinary Clinical Pathology, 2nd Edition. pp 369-413. Wiley-Blackwell.

2. Willard MD, Tvedten H. (2012) Immunologic and Plasma Protein Disorder. In: Small Animal Clinical Diagnosis by Laboratory Methods, 5th Edition. pp 278-293. Saunders Elsevier.

3. Garner BC, Priest H, Smith J. Pseudo-hypoproteinemia in a hyperbilirubinemic dog with immune-mediated hemolytic anemia. Vet Clin Pathol. 2014 Jun;43(2):266-9

4. Gupta A, Stockham SL. Negative interference of icteric serum on a bichromatic biuret total protein assay. Vet Clin Pathol. 2014 Sep;43(3):422-7

5. Vaden SL, Knoll JS, Smith FWK, Tilley LP. (2009) Total Protein. In: Blackwell's Five-Minute Veterinary Consult: Laboratory Tests and Diagnostic Procedures: Canine & Feline. pp 634-635. Wiley-Blackwell.

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