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 analyser. 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 optimise 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
Pre-Anaesthetic + Critical Care	Prep Profile II	ALP, ALT, BUN, CRE, GLU, TP
	Electrolyte Plus	Cl^-, 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 anaemia/ 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
- Haemochromatosis [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
- Haemolysis can cause mild increase [3]
- Lipaemia can cause artifactual increase [3]

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

NB. Common differential diagnoses are in bold.

Ref.

ALBUMIN (ALB)

INCREASED

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

DECREASED

Blood Loss (Haemorrhage) [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]
- Hypergammaglobulinaemia [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]
- Haemolysis or hemoglobinaemia can cause false increases
- Marked lipaemia or hypertriglyceridaemia can cause false decreases
- Severe hypoalbuminaemia 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.

ALKALINE PHOSPHATASE (ALP)

INCREASED

Physiologic
- Age (young dogs with rapid bone growth)
- Breed (Siberian Huskies – benign familial hyperphosphatasaemia; 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
- Haemangiosarcoma
- 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
- Oestrogens (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 haemolysis 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)
- Macroamylasaemia [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.

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

- Haemolysis will increase AST serum activity [3]
- Icterus may affect results
- Marked lipaemia 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.

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

- Haemolysis [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

- Lipaemia (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

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

Renal Conditions

- Inflammatory
  - Glomerulonephritis
  - Pyelonephritis (e.g. leptospirosis)
  - Tubular-interstitial nephritis [1, 2]
- Amyloidosis
- Toxic nephrosis
  - Hypercalcaemia
  - Ethylene glycol intoxication
  - Myoglobin
  - Gentamicin
  - NSAID intoxication [1]
- Renal ischaemia 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

- Haemorrhage 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 haemolysis will increase BUN concentration [1, 3]
- Severe icterus may increase BUN concentrations [1]
- Severe lipaemia 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
- Haematology
- 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]

Hypercalcaemia of Malignancy / Humoral Hypercalcaemia (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 Hypercalcaemia 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 hypercalcaemia of malignancy
- Thiazide diuretics [1]

Increased Protein-Bound Calcium

- Hyperglobulinaemia in multiple myeloma
- Hyperalbuminaemia [1]

Iatrogenic Disorders

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

Other or Unknown Mechanisms

- Haemoconcentration
- Hypothyroidism (juvenile onset) [1]
- Retained foetus and endometritis in dog (rare) [5]
- Idiopathic hypercalcaemia in cats [6]

DECREASED

Hypoalbuminaemia

- Hypoproteinaemia [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 Hypocalcaemia

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

Hypercalcitonism

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

Excess Urinary Excretion 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

Other or Unknown Mechanisms continued

- (dogs) [1,2]
- Vitamin D-receptor defect rickets [1]
- Nutritional hypocalcaemia (rare) [1]
- Oxalate toxicity [2]
- Tetracycline administration [1]
- Calcium deposition during fracture healing
- Acute pancreatitis in dogs and cats
- Urinary tract obstruction
- Acute and chronic renal failure
- Phosphate enaema
- Sepsis
- Acute tumour lysis syndrome
- Nutritional secondary hyperparathyroidism [1,2]

NB. Common differential diagnoses are in bold.

CALCIUM (CA)

ARTIFACT

- Total Ca is falsely increased by lipaemia and haemolysis[7]
- Total Ca can be decreased by marked bilirubinaemia[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
- Ionised Calcium
- Phosphate
- Blood Urea Nitrogen
- Creatinine
- Urinalysis

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

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 diarrhoea

Gastrointestinal (GI) Loss/ Sequestration of Cl^-/HCl
- Vomiting (biliary, pancreatic fluids)
  - Upper GI obstruction
- Secretory (osmotic)
  - diarrhoea
- 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 diarrhoea
- 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 hypocapnoea
  - Hypoxaemia
  - 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/diarrhoea
  - 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 haemorrhage
- Acute exudation
- Cutaneous (sweating)

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

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

Expanded Extracellular Fluid Volume (without oedema)
- 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

- Myxoedema coma of hypothyroidism (rare) [2]
- Psychogenic polydipsia [2]

Extracellular Translocation of Water

- Hyperglycaemia
- Mannitol infusion (intravenously) [1]

Intracellular Translocation of Na (Cl follows)

- Hypokalaemia (to maintain the intracellular electronegativity)
- Acute muscle injury [1]

Extravascular Fluid Translocation of Na (Cl follows)

- Uroperitoneum (ruptured bladder, or abdominal urethra) [1]

ARTEFACT

- Lipaemia causing pseudo-hypochloraemia (ion-exclusion effect when using the titrimetric methods) or causing pseudo-hyperchloraemia (using the colorimetric method) [2]
- Potassium bromide therapy will falsely increase the reported chloride concentration (common and important) [2]
- Hyperviscosity may cause problems in analysers 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.

CHOLESTEROL (CHOL)

INCREASED

Postprandial Hypercholesterolaemia [1, 2, 5]

Secondary Hypercholesterolaemia

- 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 Hypercholesterolaemia

- Idiopathic hyperlipoproteinaemia (Miniature Schnauzers and other breeds) [1, 2, 5]
- Hypercholesterolaemia in Briards (dog) [5]
- Idiopathic hyper-chylomicronaemia (cat) [2]
- Lipoprotein lipase deficiency (cat) [1, 2, 5]
- Idiopathic hypercholesterolaemia [1, 2]

Drug-Induced Hypercholesterolaemia

- 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]

ARTEFACT

- Haemolysis and hyperproteinaemia artefactually 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.

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)
- Hyperkalaemic periodic paralysis
- Malignant hyperthermia [1]

Other pathologies with uncertain pathogeneses

- Critically-ill anorectic cat [3]

DECREASED

Not clinically significant

ARTEFACT

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

NB. Common differential diagnoses are in bold.

CREATININE (CRE)

INCREASED

Pre-Renal Conditions

- Hypovolaemia
  - Dehydration
  - Hypoadrenocorticism
  - Shock
  - Blood loss
- Decreased cardiac output
  - Cardiac insufficiency
  - Shock
  - Hypoadrenocorticism
- Shock
  - Hypovolaemic
  - Cardiogenic
  - Anaphylactic
  - Septic
  - Neurogenic [1]

Renal Conditions

- Inflammatory
  - Glomerulonephritis
  - Pyelonephritis
  - Tubular-interstitial nephritis
  - Amyloidosis
- Toxic nephrosis
  - Hypercalcaemia
  - Ethylene glycol intoxication
  - Myoglobin
  - Aminoglycosides
  - NSAID intoxication
- Renal ischaemia 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]

ARTEFACT

- 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

- Haemolysis 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.

Ref.

GLOBULIN (GLOB)

Calculation using measured parameters (Total Protein and Albumin)

INCREASED

HAEMOCONCENTRATION

- 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 leukaemia 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 haemolytic anaemia (IMHA), thrombocytopaenia (IMT), and polyarthritis [2]
- Immune-mediated haemolytic anaemia (IMHA) [2] and Immune-mediated thrombocytopaenia (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
  - Macroglobulinaemia
  - Lymphoma
  - Extramedullary plasmacytoma (rare)
  - Chronic lymphocytic leukaemia
  - Waldenström's macroglobulinaemia [2]

Miscellaneous

- Cutaneous amyloidosis [3]
- Lymphocytic-plasmacytic gastroenterocolitis

DECREASED

- Blood Loss (haemorrhage) [2]
- Protein Losing Enteropathy (PLE) [2]
- Acquired
  - Chemotherapy, radiation therapy, or other compounds (e.g. toxins, drugs)
- Inherited
  - IgM deficiency (Dobermans)
  - 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 hypogammaglobulinaemia (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.

GLUCOSE (GLU)

INCREASED

Physiologic
- Post-prandial
- Excitement, fright
- Steroid-associated
- Dioestrus/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 Hyperglycaemia (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 hypoglycaemia ("hunting dog hypoglycaemia") [1, 2]

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

Uncertain Pathogeneses
- Sepsis (especially with endotoxaemia) [i]
- Non- β-cell neoplasia neoplasms
  - Hepatocellular carcinoma, hepatoma, leiomyosarcoma, leiomyoma, hemangiosarcoma, etc. [2]
- Pregnancy hypoglycaemia
- Malonic aciduria (Maltese dogs) [1]
- Idiopathic hypoglycaemia
  - Neonatal hypoglycaemia
  - Juvenile hypoglycaemia (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 / bacteraemia (parasitaemia) 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.

- 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 hyperglycaemia [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 azotaemia
- 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
- Ischaemic intestinal lesions (shift intracellular fluid to extracellular fluid)
- Granulomatous disease
  - Fungal, parasitic
- Humoral hypercalcaemia 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 tumour 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 hypercalcaemia 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 / steatorrhoea [1, 2]

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

Defective Mobilisation 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)
  - Haemoglobin (haemolysis)
  - Lipaemia
  - 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)
  - Hyporeninaemic 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 haemolysis with potassium-rich erythrocytes
- Massive tissue damage
  - Acute tumour lysis syndrome
  - Reperfusion of extremities after aortic thromboembolism in cats with cardiomyopathy
  - Crush injuries [2]
  - Hyperkalaemic 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
- Diarrhoea [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 alkalaemia [2]
- Catecholamine release [2]
- Endotoxaemia [1]
- Third space loss (body cavity effusion) [1]
- Hypokalaemic renal failure in cats
- Hypokalaemic myopathy of Burmese kittens

NB. Common differential diagnoses are in bold.

POTASSIUM (K)

ARTIFACT

- Lipaemia 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 bilirubinaemia: slight increase (ion-selective electrodes) [2]
  - Marked thrombocytosis [1, 2]
  - Marked leukocytosis (physiologic or neoplastic) [3]
  - In vitro haemolysis of K-rich erythrocytes in the Akita and Shiba Inu breeds [2]
  - In vivo haemolysis 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 diarrhoea
- 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/diarrhoea
- 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 haemorrhage or acute exudation [1, 2]

Cutaneous Loss
- Sweating
- Exudative skin lesions

Oedematous 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 Oedema)
- 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]
- Myxedaema coma of hypothyroidism (rare) [2]
- Psychogenic polydipsia [2]

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

Intracellular Translocation of Sodium
- Hypokalaemia
- 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)

ARTEFACT

- Lipaemia may cause pseudo-hyponatraemia (ion-exclusion effect) [2]
- Marked hyperproteinaemia may cause a false decrease in measured sodium [2]
- Haemolysis 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 heparinised plasma Na [1]

INTERPRET THE SODIUM WITH:

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

NB. Common differential diagnoses are in bold.

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 haemolytic anaemia
  - Systemic lupus erythematosus
  - Intervertebral disk disease
  - Polyradiculoneuritis
  - Acute renal failure
  - Acute hepatitis
  - Acute pancreatitis
- Chronic diseases
  - Generalised demodicosis
  - Generalised bacterial furunculosis
  - Systemic mycoses
  - Lymphoma
  - Chronic renal failure
  - Diabetes mellitus
  - Congestive heart failure
  - Cardiomyopathy
  - Chronic hepatitis, cirrhosis
  - Gastrointestinal disturbances
  - Megaoesophagus

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.

THYROXINE (TT4)

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

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

Surgery/anaesthesia [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

HAEMOLYSIS
(not an extensive list)

Immunological Alterations
- Immune Mediated Haemolytic Anaemia (IMHA) [1,2]
- Blood transfusion reaction
- Drug induced haemolytic anaemia
- Vaccine associated [1,2]

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

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

Oxidative Damage
- Heinz body anaemia
- Methaemoglobulinaemia
- Eccentrocytic haemolysis (acquired or inherited) [1]

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

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

Other Unknown Pathogeneses
- Envenomation [1]
- Haemophagocytic histiocytic sarcoma [1]
- Increased osmotic fragility [1]
- Hereditary non-spherocytic haemolytic anaemia of Beagles [1]
- Idiopathic haemolytic anaemia 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 anaemias

ARTEFACT

- Haemolysis or lipaemia 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
- Haematocrit

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^+

- Hypokalaemia [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 (Hyperchloraemic Metabolic Acidosis)

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

Compensation for Primary Respiratory Alkalosis

- Hyperventilation – hypoxaemia
- Primary pulmonary disease
- Pain

Dilutional Acidosis

- Rapid infusion of saline

Iatrogenic

- Ammonium chloride administration

ARTEFACT

- 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

- Haemoconcentration – 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 leukaemia [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]

Haemodilution

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

ARTIFACT

- Haemolysis or gross lipaemia 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
- Haematology
- Urinalysis
- Urine Protein: Creatinine Ratio

NB. Common differential diagnoses are in bold.

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Produced December 2022 • MM-23430

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