# **Blood Urea Nitrogen (BUN)**

# Interpretive Summary

**Description:** Blood Urea Nitrogen (BUN) is produced in the liver and excreted by the kidneys. It is a marker for both liver and kidney dysfunction.

## **Decreased BUN**

#### **Common Causes**

- Chronic, severe liver disease
  - o Portosystemic shunts (PSS)
  - o Cirrhosis
- Low protein diets
- Severe polyuria/polydipsia (common causes)
  - o Diabetes mellitus
  - o Cushing's disease
- Drugs
  - o Corticosteroids
  - o Other drugs causing diuresis

#### **Uncommon Causes**

- Urea cycle enzyme deficiencies
- Protein malnutrition
- Late pregnancy
- Addison's disease
- Severe polyuria/polydipsia (uncommon causes)
  - Other causes of glucose in urine (renal tubular disorders)
  - Diabetes insipidus
  - o Psychogenic polydipsia
- Overhydration
- Drugs
  - o Growth hormone
- Artifacts (depending on method of analysis)
  - o High concentrations of sodium fluoride and sodium citrate
  - o Chloramphenicol
  - o Severe lipemia

# **Related Findings**

- Chronic, severe liver disease
  - o Decreased albumin, cholesterol, glucose
  - o Increased bilirubin and/or increased liver enzymes (ALT, AST, ALP, GGT)
  - o Abnormal liver function tests: fasting and post prandial bile acids, fasting ammonia
  - Microcytic, hypochromic anemia (especially in PSS), acanthocytes
- Severe polyuria/polydipsia
  - o Decreased urine specific gravity
  - o Diabetes mellitus
    - Increased serum glucose
    - Glucose +/- ketones in urine
  - o Cushing's disease
    - Increased ALP



Abnormal adrenal function tests

### **Increased BUN**

#### **Common Causes**

- Pre renal
  - Hypovolemia/blood loss
  - Dehydration
  - Poor cardiac output
  - o Addison's disease
  - Recent high protein meal
  - Gastrointestinal hemorrhage
- Renal
  - Infectious
  - Inflammatory
  - o Toxic
  - o Ischemic
- Post renal
  - o Bladder, urethral or ureteral rupture
  - o Ureteral or urethral obstruction

#### **Uncommon Causes**

- Pre renal
  - o Catabolism of body tissues
    - Fever
    - Trauma
- Renal
  - Congenital
  - Amyloidosis
  - Neoplasia

# **Related Findings**

- Pre-renal disease
  - o Normal or increased creatinine
  - o Increased phosphorus only with severe pre renal disease
  - o Increased urine specific gravity (hypersthenuria; >1.030 dogs, >1.035 cats, >1.025 horses)
  - o Dehydration
    - Increased serum proteins
    - Increased hematocrit
  - Addison's disease
    - Decreased resting serum cortisol, abnormal ACTH stimulation test
    - May have decreased sodium and chloride, increased potassium
  - Shock/decreased cardiac output
    - Decreased blood pressure
    - Cardiac/vascular abnormalities on thoracic radiographs/echocardiography
  - Gastrointestinal hemorrhage
    - Positive fecal occult blood
- Renal disease
  - Increased creatinine and phosphorus
  - Isosthenuria (urine specific gravity 1.008-1.012)
  - o Mild to moderate nonregenerative anemia with chronic disease (decreased erythropoietin)
  - Positive PCR or serology for leptospirosis, Lyme or other infections
  - o Urinary casts, pyuria, hematuria, proteinuria, glucosuria, and bacteria
  - Positive urine culture with pyelonephritis
  - o Increased blood pressure
  - Abdominal ultrasound shows abnormal renal size and structure



- o Protein losing nephropathy (glomerulonephritis or amyloidosis)
  - Decreased albumin
  - Increased urine protein:creatinine ratio
- Post-renal
  - o Increased creatinine
  - o Increased potassium with severe cases
  - o Urine sediment can show crystals, blood, white blood cells with obstructive disease or blood with rupture
  - Uroabdomen
    - Abdominal fluid contains higher creatinine concentrations than serum
    - Abnormal ultrasound suggests rupture
    - Contrast radiographs for urinary tract rupture/leakage
  - o Abdominal ultrasound for masses, stones, other causes of obstruction in the urinary tract

### **Additional Information**

### **Physiology**

- Urea is produced exclusively by the liver from ammonia.
- It is predominantly excreted by the kidneys and is the main form in which nitrogen is removed from the body. Some urea is passively reabsorbed from the renal tubules back into the blood, but most is excreted.
- While urea is predominantly a waste product of protein metabolism, it is an important factor in establishing the medullary osmotic gradient in the kidneys. This allows the renal tubules to modify the water content of urine. In medullary washout syndrome there is insufficient urea in the renal medulla to allow the urine to be appropriately concentrated.
- Renal urea reabsorption is enhanced by antidiuretic hormone (ADH).
- There is an inverse relationship between BUN and glomerular filtration, and between BUN and renal tubular flow rate.
- Increased BUN is not specific for primary renal disease, but is a major contributor to azotemia, along with creatinine
- Urea concentrations are affected by other extra renal factors (more so than creatinine concentrations). These extra renal factors include other diseases, some chemicals and drugs, diet, and condition of serum (e.g. lipemia).
- BUN readily diffuses into blood and all body water in similar concentrations.

### References

- Stockham SL, Scott MA. Fundamentals of Veterinary Clinical Pathology, 2nd ed. Ames, IA: Blackwell; 2008.
- Willard MD, Tvedten H, eds. *Small Animal Clinical Diagnosis by Laboratory Methods*, 4th ed. St. Louis, MO: Saunders; 2004.

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