Clinical Pathology Biochemistry Abnormalities

	Glu	Chol	Trig	BUN	Crea	P	Ca	Mg	Alb	Glob
Increase	Postprandial Stress Excitement Drugs Pancreatitis Sepsis DM/EMS Cushing's HyperT4	Postprandial Cholestasis Pancreatitis DM Cushing's HypoT4 PLN	Postprandial Pancreatitis DM/EMS Cushing's HypoT4 PLN	Dec GFR -Dehydration -Drugs -Shock -ARF/CRF -Uroabd/Obs GI protein -Hemorrhage -Diet -Urea tox	Dec GFR -Dehydration -Drugs -Shock -ARF/CRF -Uroabd/Obs	Dec GFR -Dehydration -Drugs -Shock -ARF/CRF -Uroabd/Obs HypoPTH Grass tetany Vit D tox Rhabdomyo HyperT4	Neoplasia -LSA -Apocrine -Myeloma -OSA -HyperPTH Vit D tox Addison's ARF*/CRF Gran inflam	Dec GFR -Dehydration -Drugs -Shock -ARF/CRF -Uroabd/Obs Milk fever Mg Admin In vitro lysis	Dehydration	Dehydration Positive APP Neoplasia -Lymphoma -Leukemia -Myeloma
Decrease	Delayed spin Sepsis Pregnancy Lactation Neonatal Exertion Xylitol Liver failure PSS Addison's Insulinoma	Liver failure PSS PLE Addison's	N/A	Liver failure PSS	Low muscle	CRF Diuresis -Osmotic -Other Insulin OD Low P diet Maternal -Milk fever -Eclampsia -Preg tox	HypoPTH Pancreatitis ARF/CRF* Low Alb GI disease Low Ca diet Rhabdomyo Eth Glycol Maternal -Milk fever -Eclampsia -Preg tox	CRF Diuresis -Osmotic -Other Low Alb GI disease Low Mg diet Grass tetany	Hemorrhage Negative APP Liver failure PSS PLN*/PLE	Hemorrhage PLE FPT
	ALT, SDH	**AST, LDH**	**Amyl, Lipase**	**ALP**	**GGT**	**Bilirubin**	**Na, Cl**	**K**	**TCO2**	**AG**
Increase	Liver injury	Liver injury Muscle injury	Panc injury Liver injury GI injury Dec GFR -Dehydration -Drugs -Shock -ARF/CRF -Uroabd/Obs	Cholestasis Drugs -Cortisol -Phenobarb -T4 Colostrum Bone activity -OSA -Myeloma -Fracture/Sx	Cholestasis Drugs -Cortisol -Phenobarb -T4 Colostrum	Hemolysis Hemorrhage (internal) Fasting (LA) Cholestasis	Dehydration	ARF Addison's Uroabd/Obs Rhabdomyo Acidosis	Met alkalosis -Vomiting -Gastric obs -GDV/DA -GI stasis	Ketones -DM/DKA -Ketosis Lactic acid -Hypoxia Uremic acids -See dec GFR Eth glycol
Decrease	N/A	N/A	N/A	N/A	N/A	N/A	GI disease ARF/CRF Diuresis -Osmotic -Other Addison's Uroabd/Obs Rhabdomyo	GI disease CRF Diuresis -Osmotic -Other Anorexia Alkalosis	Excess acid -See AG Bicarb loss -GI disease -RTA	Low Alb

- **ALT = small animals only**
  - Largely liver specific
  - Marked muscle injury can cause mild increase
- **AST = all animals; large animals**
  - Liver and muscle (more muscle than ALT)
  - Response to injury is less than ALT
- **SDH = cattle and horses**
  - Liver specific
  - No advantage over ALT in dogs and cats
- **GDH = large animals and birds**
  - Not used much
  - Liver specific
- **ALP**
  - Cholestatic indicator
  - Elevated before cholestasis in the dog, rat, monkey
  - NOT great for cats and horses (no elevation until AFTER cholestasis)
  - Elevated in dogs with cushing's
  - Can be elevated in young animals
- **GGT = all species except horses and alpacas**
  - Biliary disorders indicator
- **Bilirubinuria**
  - Normal if in urine of dog
  - Abnormal if in urine of cat; indicates liver disease
  - Cats are very sensitive; usually elevated after ALP is increased = cholestatic disorder

- ALT < AST = pyometra in dogs
- ALP > GGT = hepatic lipidosis
- Increase in total bilirubin and direct bilirubin in ruminants = hemolysis
- Increase GGT and ALP in ruminants = think Pyrrolizidine alkaloid toxicity

NEUTROPHILS – NEUTROPHILIA

MAIN CAUSES OF NEUTROPHILIA

1. Stress (cortisol-mediated)
2. Excitement or physiologic (epinephrine-mediated)
3. Inflammation

(1) STRESS NEUTROPHILIA (cortisol-mediated)

Background

A stress neutrophilia occurs in response to endogenous (made within the body) or exogenous (administered by a veterinarian or owner) corticosteroids. This type of neutrophilia is seen in stressed animals (e.g. illness) & usually occurs **within hours to days** (not minutes seen with a physiologic/epinephrine-mediated neutrophilia). Animals with hyperadrenocorticism (Cushing's disease) can also have a stress neutrophilia due to excess corticosteroids. Conversely, animals with hypoadrenocorticism (Addison's disease) lack a stress leukogram, despite having clinical signs and a clinical history suggesting illness.

CBC findings that support interpretation of a stress neutrophilia:

- Mild neutrophilia (neutrophilia < 2x URL)
- No left shift
- Lymphopenia* (best support)
- ± Monocytosis
- ± Eosinopenia

Chemistry findings (may or may not be present):

- Hyperglycemia (related to cortisol effects on insulin, glycogen, lipolysis, etc)
- Increased ALP activity (dogs only)

Pathophysiology

Why is there a neutrophilia?

- Corticosteroids: Increase rate of release of mature neutrophils from bone marrow storage pool
- Corticosteroids: Down-regulate adhesion molecules on neutrophils (decreased 'stickiness' to vessel wall) so that marginal neutrophils → circulating neutrophils

Neutrophil appearance (may or may not be present)

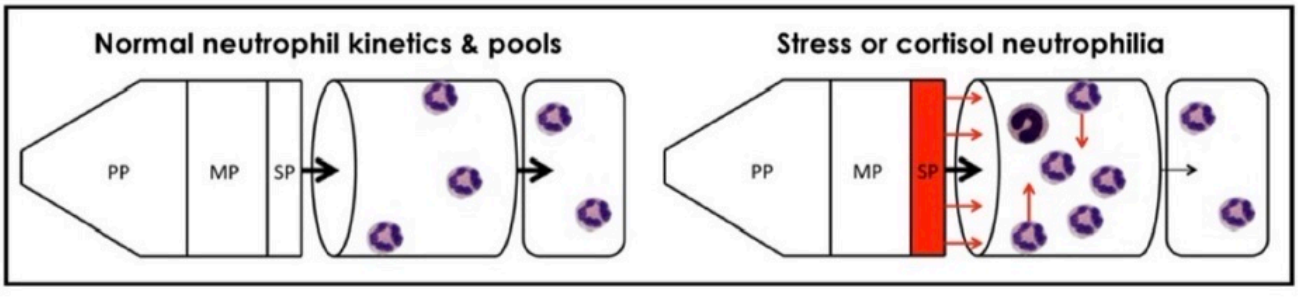
- Neutrophils continue to age, regardless of their location - increased time in circulation (due to diminished margination into tissue) can result in 'excess' (>5 segments) segmentation of neutrophil nuclei (hypersegmented neutrophils). Hypersegmented neutrophils are considered normal in horses.

Predisposition to infections

- Decreased margination into tissues and sites of inflammation may predispose an animal to infection

Why is there a lymphopenia?

- Lymphopenia is the key to a stress leukogram. Corticosteroids 'trap' lymphocytes in lymphoid organs.



- **Mean Corpuscular Volume (MCV)**
  - Average volume of RBCs
    - Macrocytic = increased MCV
      - Ex: increased reticulocytes, poodles, FeLV infection, folate/cobalamin deficiency
    - Normocytic = normal MCV
    - Microcytic = decreased MCV
      - Ex: iron deficiency, young animals, liver disease
- **Mean Corpuscular Hemoglobin Concentration (MCHC)**
  - Average concentration of hemoglobin within circulating RBCs
    - Hyperchromic = increased MCHC
      - Ex: artifacts such as lipemia and hemolysis
    - Normochromic = normal MCHC
    - Hypochromic = decreased MCHC
      - Ex: iron deficiency, increased reticulocytes, liver disease

- Calcium binds to albumin
  - So if there is low albumin, then there will be low calcium too because calcium needs to bind to albumin
- Calcium x phosphorous >80-100 = tissue mineralization
- Chloride should mirror sodium