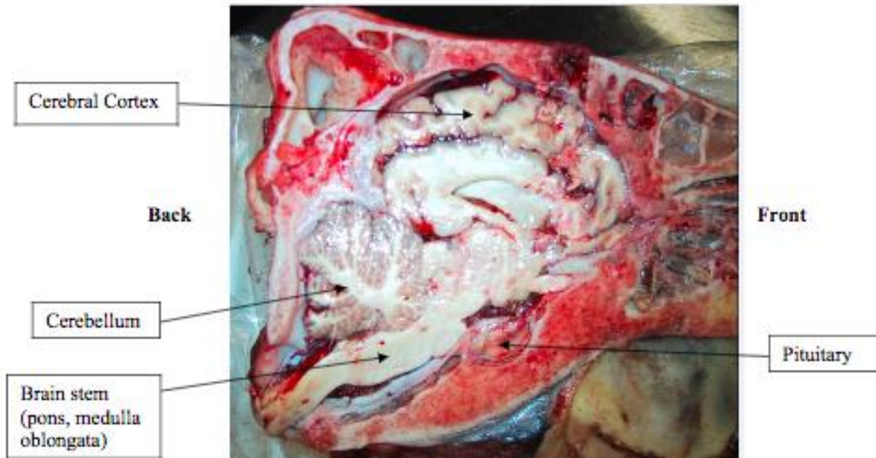
Lab Quizzes Overview

LAB #1: Nervous System

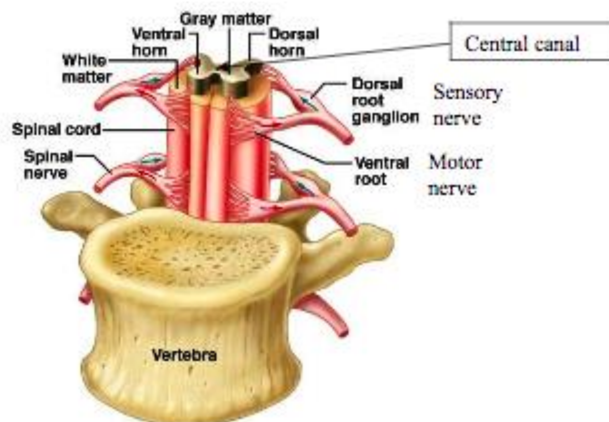
Question 1: Annotate the picture shown below



Question 2: What composes the grey matter and white matter?

The grey matter has a greyish color and corresponds to nerve cell bodies. The white matter is white and corresponds to nerve cell processes mainly myelinated axons.

Question 3: Draw a cross section of the spinal cord in place in a vertebra. Indicate where the spinal nerves enter and exit, and what type of fibers are present (motor/sensory). Can you indicate where the central canal is located? What is present in that canal?

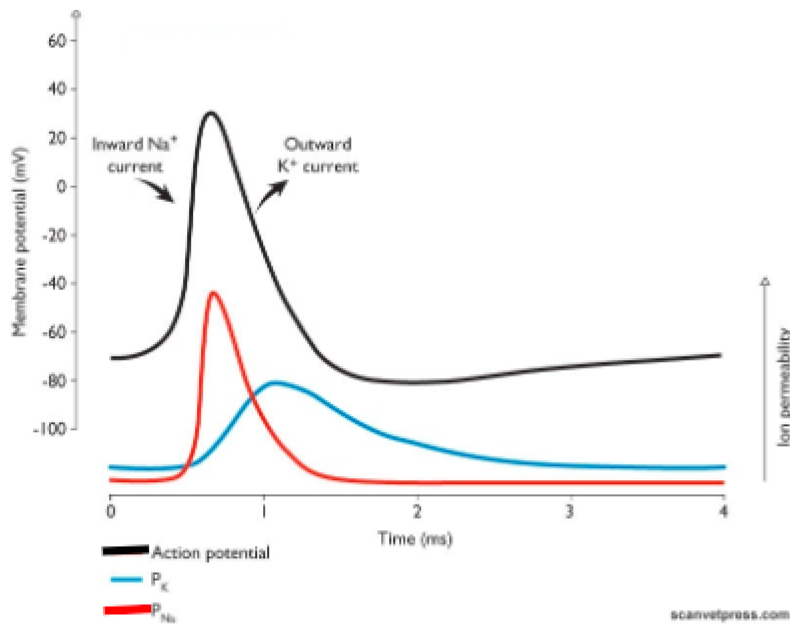


Cerebrospinal fluid is present in the central canal. In the spinal nerve both sensory and motor fibers are present. However, at the level of the spine the spinal nerve splits into 2 roots: The dorsal roots enter the dorsal horn of the spinal cord and corresponds to sensory fibers. Note that the cell bodies from these fibers are located outside the spinal cord in what is called a ganglion. The ventral root enters the ventral

horn of the spinal cord and corresponds to motor fibers. The cell bodies from these fibers are located inside the CNS (brain or spinal cord).

The butterfly shape grey matter corresponds to the cell bodies of motor neurons and association neurons.

<u>Question 4</u>: On the scale below, draw an action potential. Annotate the scale (volt on the Y axis, time on the X axis). For each phase on this action potential indicate what major events occur regarding ion channels. What would happen if Na+ channels would remain constantly open?



The black curve represents the membrane potential, the red curve corresponds to the membrane permeability to Na (channels open or closed) and the bleu curve corresponds to the membrane permeability to K (channels open or closed)

***Depolarization** following the opening of gated Na channels; **repolarization** following the closure of gated Na channels and the opening of K gated channels; **hyperpolarization** due to an excess in K outflow (these channels are slower to close); return to resting membrane potential.*

If Na channels remain open the repolarization will not occur, that could lead to a tetanic phase followed by a total lack of stimulation. This inability to be restimulated is due to the fact that the membrane does not return to its resting membrane potential and thus stuck in refractory mode.

<u>Question 5</u>: What is the main transmitter involved in the neuromuscular synapse?

Acetylcholine (Ach)

<u>Question 6</u>: Once released, how does the neurotransmitter transmit the nerve impulse to the muscle cell?

Ach will bind to specific receptors associated to Na channels = Ligand gated channels. Once binding occurs the gated channel opens, and the influx of Na will trigger the depolarization of the muscle fiber which in turn provokes the muscle contraction.

<u>Summary Functions CNS</u>

Brain stem

Midbrain: *relays motor impulses from the cerebral cortex to the pons and sensory impulses from the spinal cord to the thalamus. Superior colliculi coordinate movements of the eyeballs in response to visual and other stimuli, and the inferior colliculi coordinate movements of the head and trunk in response to auditory stimuli. Contributes to control of muscular movements.*

Pons: *relays impulses from one side of the cerebellum to the other and between the medulla and midbrain. Pneumotaxic area and apneustic area, together with the medulla oblongata, help control breathing.*

Medulla Oblongata*: Relays motor and sensory impulses between other parts of the brain and the spinal cord. Vital centers regulate heartbeat, breathing (together with pneumotaxic and apneustic area of pons), and blood vessel diameter. Other centers coordinate swallowing, sneezing, vomiting, coughing, and hiccupping.*

Reticular Formation*: Helps maintain consciousness, causes awakening from sleep, filters repetitive sensory input, and contributes to regulation of muscle tone.*

Spinal Cord

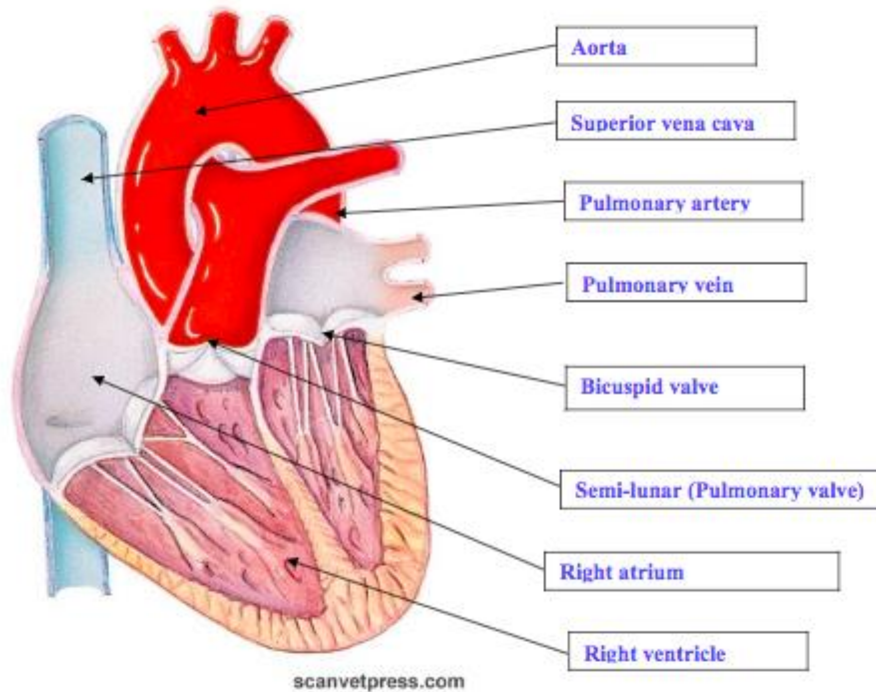
Conducts sensory nerve impulses toward the brain and motor nerve impulses from the brain toward the skeletal muscles and other effector tissues. Integrates spinal reflexes.

Cerebellum

Compares intended movements with what is happening to smooth and coordinate complex, skilled movements. Regulates a posture and balance. May have a role in cognition and language processing.

<u>LAB #2: Cardiovascular System</u>

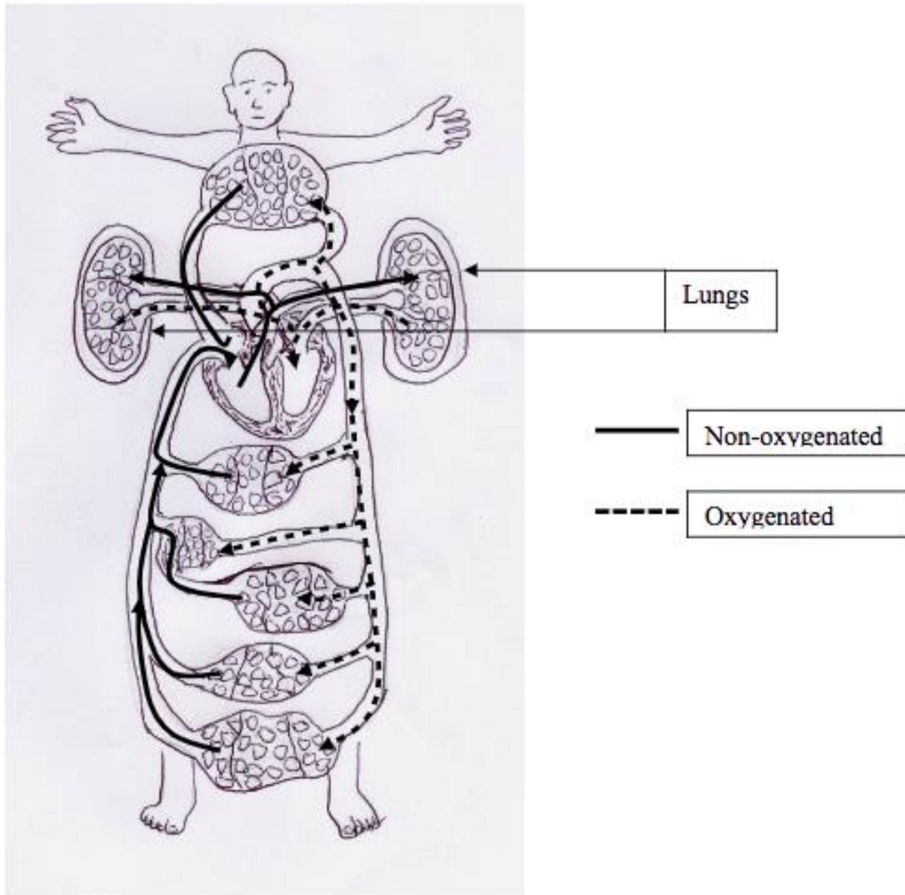
<u>Question 1</u>: On this diagram, identify the structures pointed by arrows



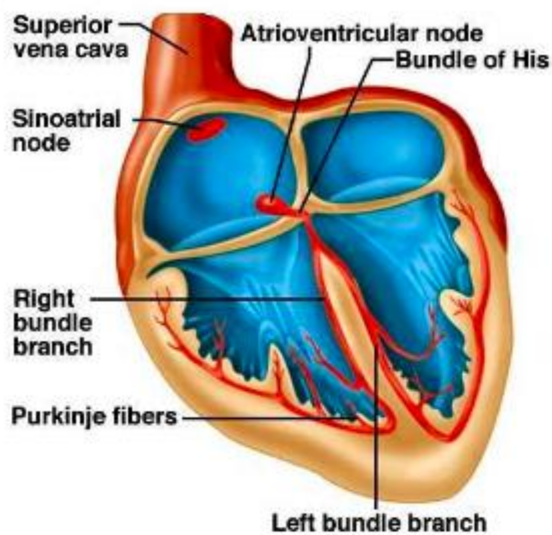
Question 2: Why is the aorta elastic?

The elasticity allows the aorta to absorb the pressure coming from the left ventricle after ejection. This large pressure (force) is then stored by the elastic fibers and progressively given back to the arterial system = maintains a higher pressure on the arterial side at all time = maintains blood flow

Question 3: Using two colors (use your own legend) draw the path taken by oxygenated and non-oxygenated blood in the pulmonary and systemic circulation. Identify the parts pointed by arrows

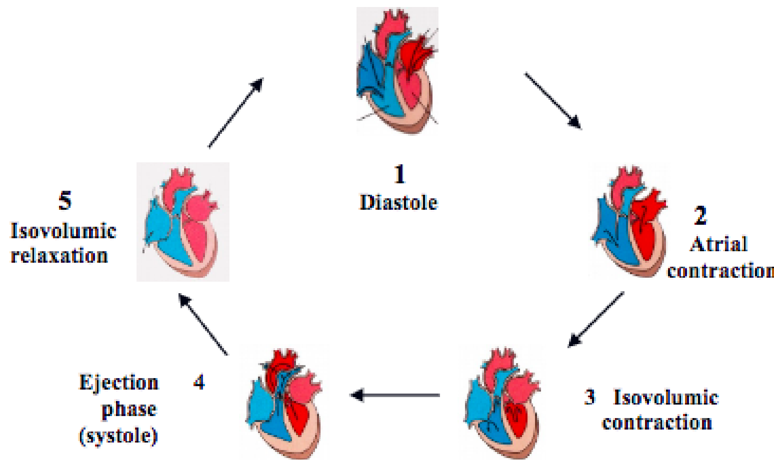


Question 4: Using a simple diagram of the heart, locate and name the autorhythmic structures. How does the electrical activity propagate?



SA node self-excites (fires an action potential) - Depolarization propagates through the atria, reaches the AV node with a little delay. AP then propagates to the bundle of hiss and the purkinje fibers very quickly. In turn, contractile cells depolarize from the apex through the ventricular wall = ventricular depolarization

Question 5: Using a diagram, detail the sequences of the cardiac cycle. For each step, describe the status of the different valves (open/closed)



Phase 1, Diastole: This is the "refilling" phase. Venous blood floods the atrium pushing the AV valves open. The heart is in a relaxed state.

Phase 2, Atrial Contraction. The action potential generated in the by the SA node has propagated throughout the atria wall and triggered atrial contraction. Blood is pushed into the ventricles (AV valves open).

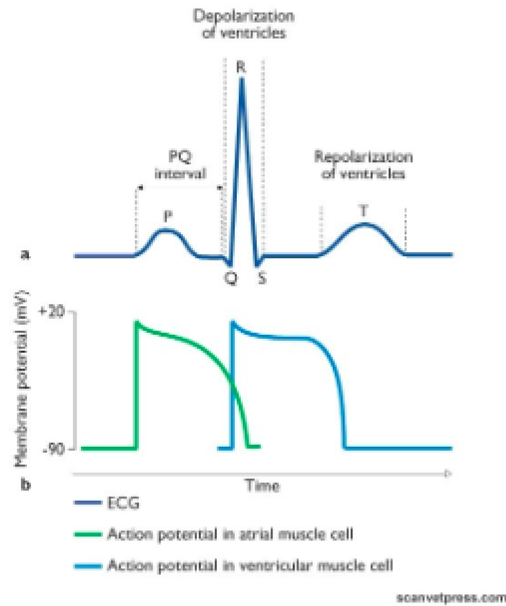
Phase 3, Isovolumic Contraction: This is the period between contraction of the atria and contraction of the ventricle. This corresponds to the lag time in electric conduction between atria & ventricles. At that stage the ventricles are full packed with blood and the pressure is higher than in the atria (AV valves closed). However, the pressure is not high enough to push the semilunar valves open.

Phase 4, Ejection: Finally, the action potential wave propagated across the ventricles and triggered the contraction. This significantly increases the pressure within the ventricles and pushes the semilunar valves open. Blood then flows out of the heart into the arteries.

Phase 5, Isovolumic Relaxation: Now blood pressure has been transferred to the arteries thus closing the semilunar valves shut. Pressure in both the atria and ventricle is low and AV valves are also closed. As venous blood starts to refill the atria the cycle moves back to phase 1 for another turn.

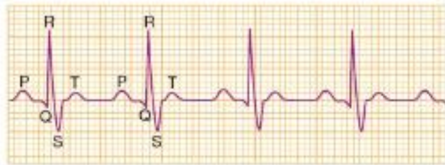
Valve	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5
Tricuspid	Open	Open	Closed	Closed	Closed
Bicuspid	Open	Open	Closed	Closed	Closed
Aortic	Closed	Closed	Closed	Open	Closed
Pulmonary	Closed	Closed	Closed	Open	Closed

Question 6: Draw the different waves (episodes) recorded during a "normal" electrocardiogram (ECG). What electrical event do they represent?

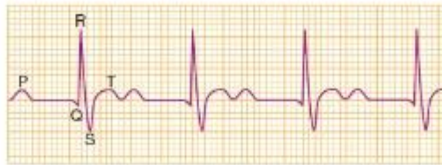


P = atrial depolarization, QRS = ventricle depolarization, T = ventricle repolarization, PQ interval = time taken by the action potential to pass through the AV node (from the atria to the ventricles).

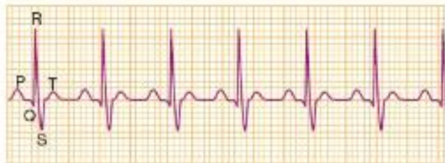
Examples of ECG



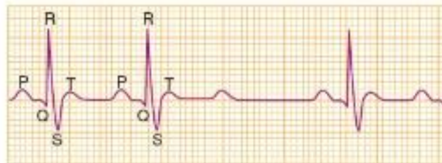
a Normal ECG.



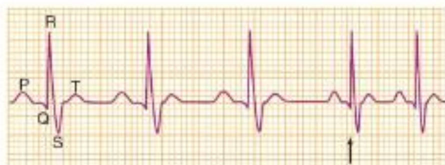
e First degree AV block. The propagation of the atrial depolarizations to the ventricles is slow, and the PQ interval is abnormally long.



b Sinus tachycardia. The ECG-recordings demonstrate increased heart rate (decreased distance between P waves).

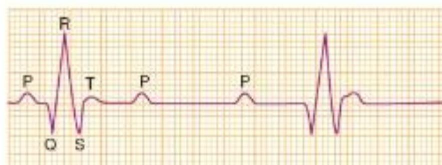


f Second degree AV block. Some of the atrial depolarizations are not conducted to the ventricles.



Start of tachycardi

c Paroxysmal tachycardia. The heart rate increases abruptly (indicated by arrow).



g Third degree AV block. There is a complete failure of impulse conduction from the atria to the ventricles. The QRS complexes originate in tissues below the AV node. The ventricular rhythms are slow, but usually regular.

LAB #3: Case Study I

Heartworm Case

History:

"Violet" a female hound ~ 5 years of age, recently adopted. When rescued she was found living outside a swamp. Vaccinated for rabies in order to enter Canada however, no additional medical history is known. "Violet's" new owners present her to your clinic for a general wellness exam and to discuss an occasional cough & a low tolerance for exercise.

Physical Exam:

- → Following are found to be within normal limits on exam: temperature (normal = 37.5-39.2C), ocular (eye) exam, aural (ear) exam, mucous membranes, integument (skin), lymph nodes, mentation and joints
- → Following are **abnormal** on exam:
  - Heart murmur is present
    - Rate appears WNL (normal HR = 80-120 bpm)
  - Increased lung sounds (wheezing) & tachypnea (increase respiratory rate)
    - RR = 45 (normal = 15-30 breaths per minute)
  - Distended abdomen with a palpable fluid wave (ballottement) = ascites

Differential Diagnosis:

- → Due to cough consider pneumonia, collapsing trachea or infectious tracheobronchitis
  - Ruled out because: murmur and x-rays changes not associated with these diseases

- → Pulmonary hypertension due to congenital defects (patent ductus arteriosus or ventricular septal defect)
    - o Ruled out because unlikely in aged dog (present at birth)
- → Heartworm disease

Diagnostic work-up:

- → Chest x-rays show:
    - o Right ventricular enlargement
    - o Pulmonary trunk bulge
    - o Centrally enlarged & tortuous lobar pulmonary arteries
        - ▪ Usually no larger than the 9th rib on dorsoventral (DV) view
    - o Lung opacity noted – suggest edema or pneumonia
- → ECG – WNL
- → Blood work – WNL

Next Step: *Additional diagnostics tests*

- → Adult heartworm antigen (Ag) test
    - o Primary screen with a commercially available kit
    - o Detect circulating Ag from the adult female
    - o ELISA test
- → Microfilaria concentration test
    - o Confirmatory test to identify microfilariae
- → Cardiac ultrasound

Diagnosis: Heartworm (dirofilarial immitis) disease

Treatment:

- → Adulticide therapy (eg. Melarsomine)
- → Microfilaricide therapy (eg. Milbemycin)
- → Surgical removal of adult heart worms possible.
- → Treatment for heart failure

Prognosis: Poor due to progression of the disease

<u>Discussion Questions:</u>

1. **Why were the differential diagnosis considered?**

They share clinical signs that were noted on Violet's physical exam

2. **What is a heart murmur?**

An abnormal heart sound cause by turbulent flow. When listening to the heart you can hear the valves closing. A murmur can result from an obstruction or valve failure

3. **What could be causing a heart murmur in this case?**

In this case, a murmur may be heard as a result of the presence of adult worms in the pulmonary arteries & right ventricle, which, produces an obstruction or the worm physically obstruct to the tricuspid and pulmonary valves causing insufficiency

4. **What is preload?**

The volume of blood present in the ventricles at the end of diastole. Diastole is the filling of the heart with blood. Another name for preload is **EDV (End of diastolic volume)**

5. **What is afterload?**

The volume of blood contained in the ventricles after systole. Systole is contraction of the heart. Afterload should be a small amount as it indicates most of the blood was ejected from the heart. Another name for afterload is **ESV (End of systolic volume).** Cardiac output is the difference between Preload and Afterload

6. **What will be the impact on preload and afterload in "Violet's" case?**

The afterload and preload will increase. Because of the obstruction and valve sufficiently that the worms cause, the blood is not properly (or completely) ejected from the heart. This results in a greater amount of blood remaining in the ventricle resulting in an increase afterload & subsequently an increased preload.

7. **Why is the right ventricle enlarged in this case?**

Pulmonary artery obstruction > pulmonary hypertension > increased afterload in the right ventricle > right ventricular hypertrophy. It is the myocardium (wall) of the R ventricle that causes the R heart enlargement. The wall thickens because the heart muscle must work harder (increase force of contractions) to be able to pump blood out against the high pressure in the pulmonary arteries.

8. **Why would a dog with heart failure have abdominal ascites (abdominal fluid)?**

Because of the backlog of blood in the venous system. The backing of blood from the right side of the heart increases the pressure in the venous system, all the way down to the capillary network in the abdominal cavity (liver etc.). This increase in hydrostatic pressure increases filtration pressure and loss of fluid. C. is correct statement however it does not cause abdominal fluid. The lymphatic system is overwhelmed in this case and cannot handle the increase fluid present.

Myasthenia Gravis Case

History:

"Jake", 9-year-old neutered male Golden Retriever was presented for progressive weakness in the hind end, which worsens with exercise but seems to improve slightly with rest. The owners have also noticed that just after eating, he will vomit food in formed, cylinder-shaped boluses. No previous history of medical problems.

Physical Exam:

→ Majority of exam is within normal limits:
  - Heart rate = 90 (normal HR = 80 –120 bpm)

        - Respiratory Rate = 17 (normal RR = 15-30 breaths per min)
        - Temp = 38.2 (normal = 37.5-39.2C)
        - Ocular (eye) exam, aural (ear) exam, mucous membranes, integuement (skin), lymph nodes, abdominal palpation
        - Mentation, reflexes, prioception (awareness of limbs & their movement), and pain withdrawal responses. [Note: these signs are associated with the sensory nervous system]
    - → Appears to be able to ambulate (walk) however in a hunched posture with short strides and his hind limbs tucked underneath him

Diagnostic Work-up:

- → General blood work is within normal limits
    - o Biochemical profile looks at organ function
    - o Complete blood count (CBC) looks at white and red blood cells
- → Thyroid hormone levels within normal limits
- → X-rays reveal:
    - o No spinal abnormalities
    - o An enlarged esophagus

Differential diagnosis:

- → <u>Polymyositis</u> (type of chronic inflammation of the muscles) - no fever, no muscle swelling, serum creatine kinase (CK) not elevated (which is a muscle enzyme)
- → <u>Tick paralysis</u> – no history of tick expose, preventative "Advantix" used
- → <u>Metabolic disorders:</u> hypokalemia, hypoglycemia – blood work normal
- → <u>Polyarthritis</u> – joints not swollen or painful with normal range of motion

Suspected Illness: Myasthenia Gravis (MG) - autoimmune neuromuscular disorder in which circulating antibodies block **nicotinic acetylcholine** receptors. The acquired form of the disease is suspecyed as it spontaneously develops in old dogs. (There is a congenital form however symptoms develop in young puppies.)

Next Step: Additional diagnostics tests

- → Acetylcholine receptor antibody test – takes time to complete blood test
- → Anticholinesterase edrophonium chloride (Tensilon) test: a drug that inhibits enzymatic hydrolysis of acetylcholine **at the neuromuscular junction.**

Diagnosis: Acquired myasthenia gravis

Treatment:

- → Supportive care
    - o Upright feeding – gravity can help draw food down the enlarged esophagus in to the stomach
    - o Minimize exercise
- → Immunosuppressive therapy (e.g. Prednisone which is a steroid)

    - Steroids are given with caution as they suppress the entire immune system, not just the production of antibodies against Ach receptors
- → Monitor anti-acetylcholinesterase antibody titer every 4-8 weeks
    - Important as acquired myasthenia gravis can spontaneously resolve and drug therapy is no longer needed when remission occurs
- → Anticholinesterase drugs (e.g. Pyridostigmine)

Prognosis:

- → Good (in this case)
- → Drug treatment effect on esophageal function is variable
- → Spontaneous remission occurs in 90% of acquired cases
    - Average 6.4 months after diagnosis (range 1-18 months)
    - Regardless of treatment used
- → Relapse rare

<u>Discussion Questions:</u>

1. **What is the common theme between the differential diagnoses?**

They all can cause changes in a dog's gait & skeletal muscle movement. A differential diagnosis is considered by considering the clinical symptoms present with a disease. All of the listed DD can cause changes in a dog's gait and skeletal muscle movements.

2. **Why perform follow up tests?**

To confirm your diagnosis.

3. **Where is Ach found?**

The somatic nervous system at the neuromuscular junction in skeletal muscles & the ganglionic synapses and the target organ synapses of the parasympathetic system. Acetylcholine (Ach) is the transmitter in neuromuscular synapses that converts the electrical signal from an action potential (AP) between a motor nerve and a skeletal muscle cell. The receptor on skeletal muscle is the nicotinic receptor. Ach is found in secretory vesicles on the presynaptic membrane. Ach is released into the synaptic cleft when the AP opens voltage gated Ca^{2+} channels. The influx of Ca^{2+} triggers Ach exocytosis with diffusion across the cleft to the postsynaptic membrane. Ach binds to specific nicotinic receptors on post-synaptic membranes, which control ligand-gated ion channels. Ligand-gated ion channels are opened and Na^+ ions diffuse into the muscle cell, causing depolarization of the postsynaptic muscle cell membrane. Depolarization opens voltage-gated Na^+ channels and lead to the generation of an action potential on the muscle cell membrane eliciting a response. The neurotransmitter in C. is Epinephrine & nor-epinephrine which are Catecholamines (a.k.a. adrenaline & nor-adrenaline).

4. **What type of receptors does Ach use?**

Nicotinic & Muscarinic. Nicotinic receptors are found at the ganglionic synapses of the autonomic nervous system and at the neuromuscular junction of skeletal muscles. Muscarinic receptors are found on the effector tissue of the autonomic nervous system (e.g. targets of the parasympathetic system like

smooth muscle, glands). The receptors for epinephrine & nor-epinephrine are alpha and beta-adrenergic receptors.

5. **How can "Jake's" symptoms be explained?**

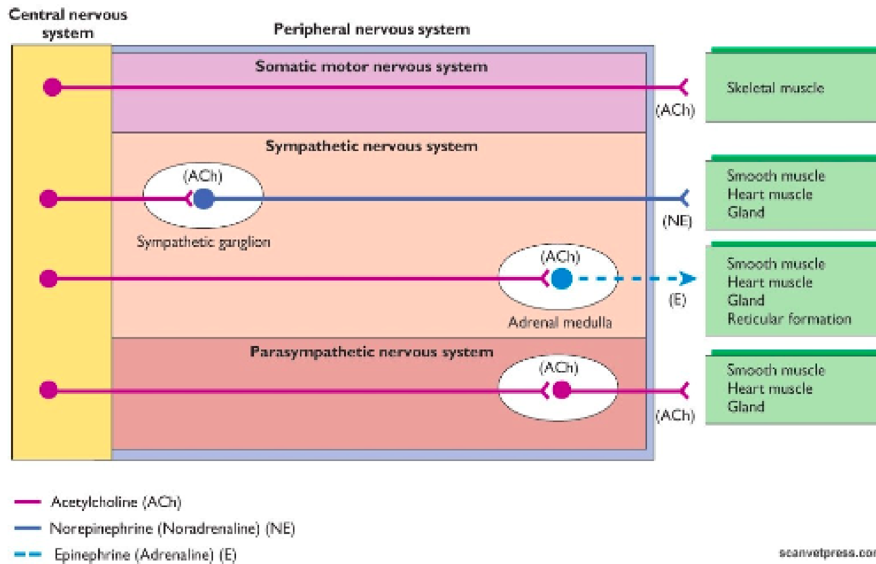
The symptoms all relate to lack of smooth muscle contractions leading to weakness, and a lack of skeletal muscle contractions leading to weakness. Normal proprioception, reflexes and no ataxia indicate that sensory nerves are not affected and the spinal cord did not appear to be effected on x-ray. "Jake" still knows what his limbs should be doing he just has difficulty ambulating due to muscle weakness. Overall, the CNS is not impacted in this disease. The upper part of the esophagus contains striated (skeletal) muscles that help push the food down. The lack of muscle tone/activity can lead to enlargement of the esophagus and regurgitation.

6. **How does the tensilon test work?**

Increases the half-life (duration) of Ach in the synaptic cleft. The tensilon test inhibits enzymatic hydrolysis of Ach at the neuromuscular junction, increasing the effective concentration of Ach and the duration of its effect in the synaptic cleft, this optimize the opportunities for successful Ach and Ach receptor interaction. The point of increasing the duration of Ach at the NMJ is to allow more opportunity for Ach to compete with the antibodies. This increases the chance of Ach binding to the receptors and facilitating transmission of an action potential. In C. increased membrane sensitivity implies that there are more receptors therefore less than Ach needed.

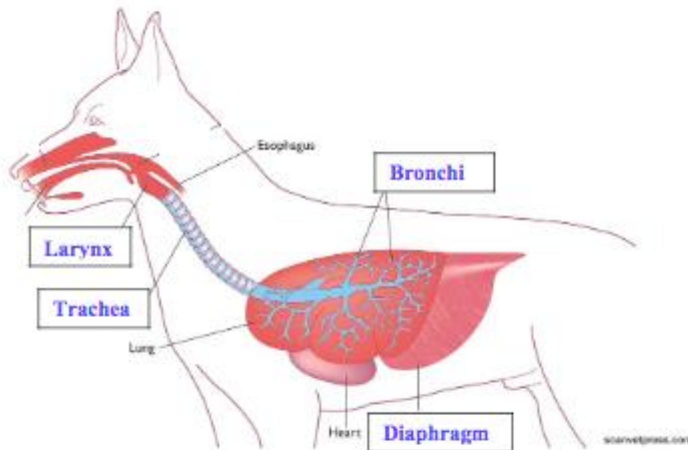
7. **What would a drug need to do to help "Jake"?**

Slow Ach destruction allowing longer action at the NMJ. In immune-medicated myasthenia gravis, antibodies are produced which bind to Ach receptors on the postsynaptic membrane of skeletal muscles resulting in impaired neuromuscular transmission. Antibodies bound to receptors reduce the sensitivity of the post synaptic membrane to Ach. Antibodies also decrease the # of receptors available to bind to Ach. Acetylcholinesterase inhibitors allow acetylcholine to work longer at the synapse, facilitating normal transmission. Slowing Ach destruction at NMJ allows more a local effect. If a drug were to increase Ach production the action would not specifically target the NMJ.



LAB #4: Respiratory System

Question 1: Complete the legend (boxes) on the diagram shown below



Question 2: What are the major functions of the:

A) - Upper air ways

Gas humidification, filtration and warming

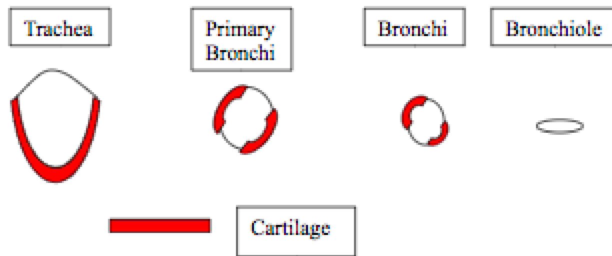
Gas distribution to the respiratory zone (NO gas exchange)

B) - Lungs

Gas transport and exchange for metabolism (bring O2 remove CO2)

Filter blood, chemical processing (lung receives 95% of cardiac output)

<u>Question 3</u>: Looking at the structure of the organs on display. Some parts of the airways (trachea) are sturdy whereas other parts (bronchiole) collapsed. Draw a CROSS-SECTION of the trachea, bronchi and bronchiole (if you can see them) AS YOU SEE THEM ON THE TISSUES DISPLAYED. What is the main anatomical reason for the difference in shape?



The main difference is the presence of the cartilage. An almost complete ring in the trachea = maintain it open. Progressively disappear from the primary bronchi to the bronchiole. As a result, the opening progressively collapses. In vivo, bronchiole stay open due to the elasticity (recoil) of the lungs.

<u>Question 4</u>: How do airways get cleared?

Ciliated and mucus producing cells (Goblet cells) in the trachea help move the "trash upward".

<u>Question 5</u>: What controls (dictates) the movement of air between the airways and the respiratory zone

The difference in pressure and its relation to lung volume (P1V1 = P2V2, changes in volume result in changes in pressure allowing air flow).

<u>Question 6</u>: When you placed the piece of lung in water, why did it float?

Due to the structure of the alveoli, some air always remains in the lung, It is called the RESIDUAL VOLUME.

<u>LAB #5: Endocrinology</u>

Question 1: For each of the glands listed below, describe where it is in the body, what hormone(s) does it produce, and its main physiology impact

a. **Thyroid Gland:**

The thyroid gland is located on both side of the trachea, below the larynx. It is composed of 2 lobes bridged by the isthmus.

The two main hormones produced are thyroxine (T4 or tetraiodothyronine) and triiodothyronine (T3).

The main tole of thyroid hormones is to increase basal metabolic rate (resting rate of calorie expenditure) of all cells of the body (except brain) thus increasing heat production (calorigenic) and O2 consumption.

b. Adrenal Glands (focus on the cortex):

The adrenals are paired organs capping the kidneys.

They consist of 2 layers: an outer cortex and an inner medulla. The medulla is part of the sympathetic nervous system and secretes catecholamines (epinephrine, norepinephrine). The cortex is composed of 3 zones:

Zona glomerulosa: secretes mineralocorticoids responsible for the metabolism of important inorganic ions (stimulate the reabsorption of Na+ and the secretion of K+ in the kidney)

Zona fasciculata: secretes glucocorticoids involved in the response to stress by enhancing the effect of norepinephrine on blood pressure, stimulating gluconeogenesis and inhibiting tissue glucose utilization. At high level, they stimulate the degradation of fat and protein. They also possess an anti-inflammatory role.

Zona reticularis, secretes androgens (functions not known – source of sex hormone during menopause?)

c. Endocrine pancreas:

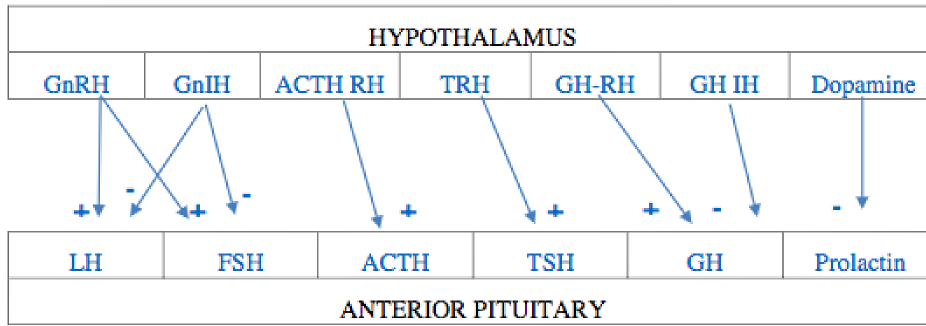
Located in the abdominal cavity alongside the duodenum (blood supply via the celiac artery – connected to the aorta).

Hormones produced are Insulin (β- cells) and Glucagon (α-cells) in the Islet of Langerhans.

Main role is Glucose uptake and metabolism in liver adipose and muscle cells. Insulin leads to the up regulation of membrane glucose transporters and thus stimulates the cellular uptake of glucose in muscle and adipose tissue. Insulin stimulates incorporation of glucose in energy storage molecules: glycogen in liver and muscle, and triglycerides in adipose tissue. Insulin also stimulates the uptake of amino acids. Glucagon has an opposite effect to insulin as it activates enzyme responsible for glycogenolysis leading to the release of glucose from glycogen by the liver. It also stimulates gluconeogenesis in the liver (synthesis of glucose) and stimulates the release of fatty acids from triglycerides in adipose tissue.

<u>Question 2</u>: List the hormones produced by the hypothalamus and the anterior pituitary gland in the table below

Using arrows indicate which hypothalamic hormone stimulates (+) or inhibits (-) the corresponding pituitary hormone



Question 3: Using the concept of action and negative feedback control complete the following table listing the hormonal effects on different organs

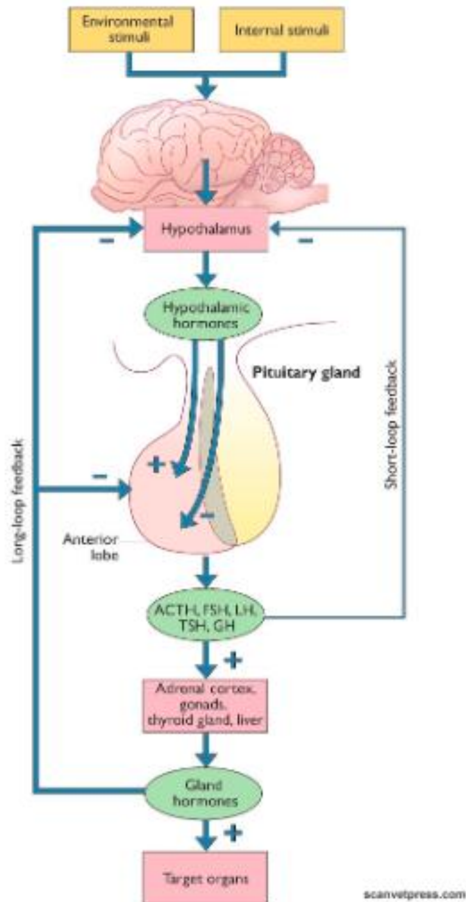
A (+) denotes a stimulation. A (-) denotes an inhibition. NC denotes no change.

	TRH	TSH	ACTH	Cortisol	Testosterone		LH	
					Intact	Castrated	Intact	Castrated
Pituitary Gland	+	-	-	-	-	-	-	-(almost NC)
Thyroid Gland	+	+	NC	NC	NC	NC	NC	NC
Adrenal Gland	NC	NC	+	-	NC	NC	NC	NC
Thymus Gland	NC	NC	-	-	NC	NC	NC	NC
Testes	NC	NC	NC	NC	-	X	+	NC
Prostate	NC	NC	NC	NC	+	+	+	NC
Seminal Vesicles	NC	NC	NC	NC	+	+	+	NC
Body Weight	-	-	-	-	+	+	+	NC

Question 4: Using a hypothalamo-pituitary-target gland axis (several choice possible) draw a typical negative feedback system

Negative feedback from the target gland hormone (eg. T3/T4) to hypothalamus, to pituitary = Long-loop feedback

Negative feedback from pituitary hormone (eg. TSH) to hypothalamus = Short-term feedback



LAB #6: Case Study II

Cushing Case

History:

Physical Exam:

- Following are found to be within normal limits (WNL) on physical exam:
  - Temperature = 38C (normal = 37.5 - 39.2C)
  - Heart rate = 95 bpm (normal HR = 80 – 120 bpm)
  - Respiratory rate = 23 breath per minute (normal = 15 – 30 breaths per minute)
  - Ocular (eye) exam
  - Aural (ear) exam
  - Mucous membranes – pink and moist
  - Lymph nodes
  - Mentation – bright, alert and responsive (BAR)
  - Joints and gait
- Following are abnormal on physical exam:
  - Integument (skin)
    - Bilateral symmetrical alopecia (loss of hair) on her body

        - Thin skin
        - Hyperpigmentation (black coloration on her white skin)
        - Seborrhea – abnormal secretions from the sebaceous glands associated with abnormalities of keratinization
            - Examples: dandruff, greasy scales, crusts
        - Pyoderma – bacterial skin infection
    - Abdominal enlargement (pot belly)

Diagnostic Work-up:

- → Blood work:
    - Biochemical profile, increased parameters:
        - Alkaline phosphatase (ALP)
            - Non-specific (can come from many sources within the body) but normally low in circulation and a good indicator of malfunction. High levels often associated with liver damage, intestinal damage, hyperthyroidism, hyperadrenocorticism...
        - Cholesterol
            - Can be elevated with fatty meals, liver disease, hyperadrenocorticism, diabetes mellitus, hypothyroid
        - Glucose (mild increase)
            - Can be elevated with stress, high carbohydrate meals, hyperadrenocorticism and diabetes mellitus
- → Urinalysis:
    - Proteinuria (protein in the urine)
    - Increased bacteria & white blood cells – suggest infection
    - Specific gravity <1.020 = the urine is dilute (range or urine specific gravity can be 1.006-1.040)
- → Abdominal radiographs
    - Hepatomegaly (enlarged liver)
    - Cystic calculi in the kidney

Next Step:

- → Endocrine work-up (measure hormone levels in blood)
    - Cortisol level abnormally high (>30mcg/dl) - diagnostic of hyperadrenocorticism
- → ACTH (adrenocorticotropic hormone) stimulation test
    - Cortisol levels measured before and 1 to 2 hours after the administration of exogenous ACTH.
- → Abdominal ultrasound
    - Bilateral enlargement (pituitary dependent) or atrophy (adrenal dependent) of the adrenal glands
- → Low dose dexamethasone suppression test (LDDST) test if often preferred.
    - Dexamethasone should lower cortisol by negative feedback in a normal individual
    - If it does not, then Cushing is most likely. The LDDST also has the advantavge in that it can pick up clues as to whether your dog has an adrenal tumor

Diagnosis: Hyperadrenocorticism (aka. Cushing's)

- → Chronic slowly progressing disease
- → Syndrome caused by an excess of one or more adrenal steroids (most commonly cortisol)
- → Pituitary dependent = 80 – 85% of cases
- → Adrenal tumor = 15 – 20% of cases
- → Iatrogenic can occur due to glucocorticoid administration

Refined diagnosis: Pituitary Dependent Hyperadrenocorticism (PDH)

- → Commonly due to microadenoma (a tumor)
- → Excessive ACTH secretion from a pituitary gland stimulates the adrenal glands to produce excessive amounts of cortisol resulting in adrenal hypertrophy. The abnormal pituitary cells become less sensitive to negative feedback inhibition by cortisol, continuing to secrete ACTH despite hypercortisolemia.

Treatment:

- → Trilostane
  - A competitive inhibitor of 3-beta hydroxysteroid dehydrogenase thereby reducing synthesis of cortisol, aldosterone and adrenal androgens
    - Remember: All steroids are produced from a stepwise conversion of Cholesterol. The drug trilostane acts as a false substrate which interacts with the enzyme usually responsible to produce cortisol.
  - Given orally with peak levels 1.5-2 hours after administration. Effect on cortisol production lasts for no more than 20 hours
  - It is metabolized in the liver
  - Cost ~ 6 dollars per day for a 65 lb. dog (~weight of a large standard poodle)

Prognosis: Survival with therapy averages ~ 30 months

Shipping Fever Case

History:

Mr. Mcdonald calls you to his farm to look at some yearling dairy heifers. They purchased the heifers at a sales yard 10 days earlier. All of them are originally from different farms. At first the heifers were doing well and now he notices they are not eating as much feed, they seem dull and one heifer has a cough.

Physical Exam:

- → Following are found within normal limits (WNL) on physical exam:
  - Heart rate 65 beats per minute 5 beats per minute (normal HR = 55 – 80 bpm)
  - Ocular (eye) exam
  - Mucous membrane – pink & moist, capillary refill time <2 sec
  - Integument (skin)

- Lymph nodes
- Joints, limbs & gait

→ Following are abnormal on physical exam:
  - Dull/depressed attitude
  - Temperature is increase to 40C (normal = 37.8 - 39.2C)
  - Respiratory rate elevated to 40 (normal = 10 – 30 breaths per minute)
  - Increased lung sound/crackles in the ventral lung field
  - Rattling of the trachea on auscultation
  - Grunt heard on expiration
  - Bilateral mucopurulent nasal discharge
  - Gaunt abdomen
  - Decreased rumen contractions – 1 heard over 3 minutes (normal = 1 – 2 per minute)

Diagnostic work-up:

→ Blood work
  - Complete blood count
    - Leukopenia (reduction in the number of white blood cells)
      - Mainly due to neutrophils moving to the site of infection
  - Fibrinogen can be increase (a non-specific indicator of inflammation)
  - Biochemical profile is within normal limits

Next Step:

→ Transtracheal wash and nasopharyngeal swab cultures were submitted and identified m*annheimia hemolytic* bacteria
    - Not practical on-farm
    - Post-mortem bacterial culture of the lungs is another possible form of diagnosis in the cases where animals have died

→ Radiographs of the lungs could estimate degree of lung consolidation
  - Not usually practical on-farm

Diagnosis: Shipping fever pneumonia

→ Lab results from a culture performed on the swab identified mannheimia hemolytic bacteria

→ When taking into consideration the history and the physical exam findings shipping fever is most likely cause of pneumonia in the calves

Treatment:

→ Given to all 4 heifers

→ Given before test results are completed due to the severity of the clinical signs

→ Broad spectrum antibiotic is used:
  - Ex. Tulathromycin (Draxin)
    - All heifers are given one dose and the 2 most severely affected are given a second dose after 5 days. Restrictions: not for use past 20-month-old (not for lactating animals). Other alternatives exist like Ceftiofur.

→ Anti-inflammatory drug was given for 3 days
  - Ex. Flunixin Meglumine
    - Non-steroidal anti-inflammatory drug

Prognosis:

→ Good in this case as the farmer identified a problem early in the disease process and the treatment was successful (heifers responded within 24-72 hours)
  - In cases where cattle take longer than 72 hours to respond there is a greater risk of chronic lung damage

→ Shipping fever morbidity can be 35% with mortality at 5-10%

Discussion Questions:

Question #1: The physical exam findings suggest a sceptic process is under way due to the pyrexia (fever) noted. Septic fever is commonly due to bacterial infections.

True

Question #2: Physical exam findings also suggest involvement of the respiratory system. What is an increase respiratory rate indicative of?

Decreased ventilation and Decreased Diffusion – cause lower PPO2 in blood therefore triggering chemoreceptors therefore increasing respiratory rate

Question #3: What are the main determinants of gas exchange in the lungs?

Driving partial pressure gradient, surface area available for diffusion, thickness of the air-blood barrier, and physical properties of the gas

Question #4: What could be causing decreased ventilation in this case?

Poor air quality (dust particulate) in the barn, mucus in the airways and inflammation causing a loss of lung compliance. Ventilation = transport of air to and from the lungs. Lung diffusion = gas exchange between respiratory zone and blood. "Lung sounds" and a grunt heard on expiration are indicative of airways obstruction possibly due to accumulation of mucus. Mucopurulent nasal discharge suggests a severe inflammatory process likely occurring posterior (behind) the nasal system confirming the presence of obstructing mucus in the airways causing poor ventilation (transport of air into the lungs). Inflammation and mucous causes a thickening at the alveoli-capillary membrane resulting in poor diffusion of oxygen into the lungs. However, it can impact lung compliance which result in poor distension and recoil affecting ventilation.

Question #5: The chemoreceptors are located ONLY in the brain (central chemoreceptors) & carotid artery (peripheral chemoreceptors)

False. Central chemoreceptors in the brain indirectly detect changes in pCO2 in the blood to the brain by H+ concentration. Peripheral chemoreceptors in the carotid artery and the aorta directly detect changes in pCO2 and pO2.

Question #6: What would be the advantage of performing a transtracheal wash?

Identification of specific bacteria involved allowing for targeted treatment.

Question #7: The reason to give an antibiotic is to help combat the virus that is causing pneumonia

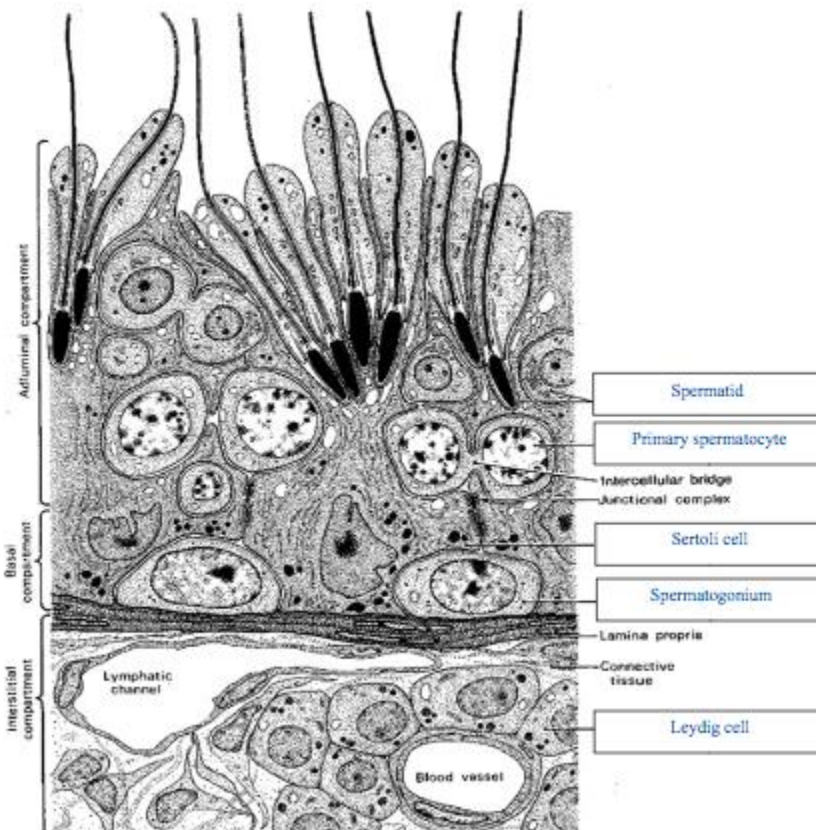
False. Antibiotics do not have action against viruses. A broad-spectrum antibiotic is used to combat the bacterial overload in the respiratory system, which the heifer's immune system is unable to combat successfully. Tulathromycin (product name = Draxxin) is approved for the use is non-lactating dairy cattle against bovine respiratory disease (shipping fever)

Question #8: Flunixin meglumine (Banamine) is a non-steroidal anti-inflammatory drug that will help decrease the fever, make the heifer feel generally better and reduce the inflammation in the airways

True. Flunixin meglumine (Banamine) is a non-steroidal anti-inflammatory drug that has analgesic, anti-inflammatory and antipyretic activity. With the administration of Flunixin the fever will be decreased which will allow the heifer's to generally feel less dull, their appetite will increase, and their rumen function will return. It will also reduce the inflammatory process occurring in the airways and lungs. This is important because histamine, (from mast cells which are part of the inflammatory process) causes bronchoconstriction, which further decreases the respiratory capacity of the heifers.

LAB #7: Male Reproduction

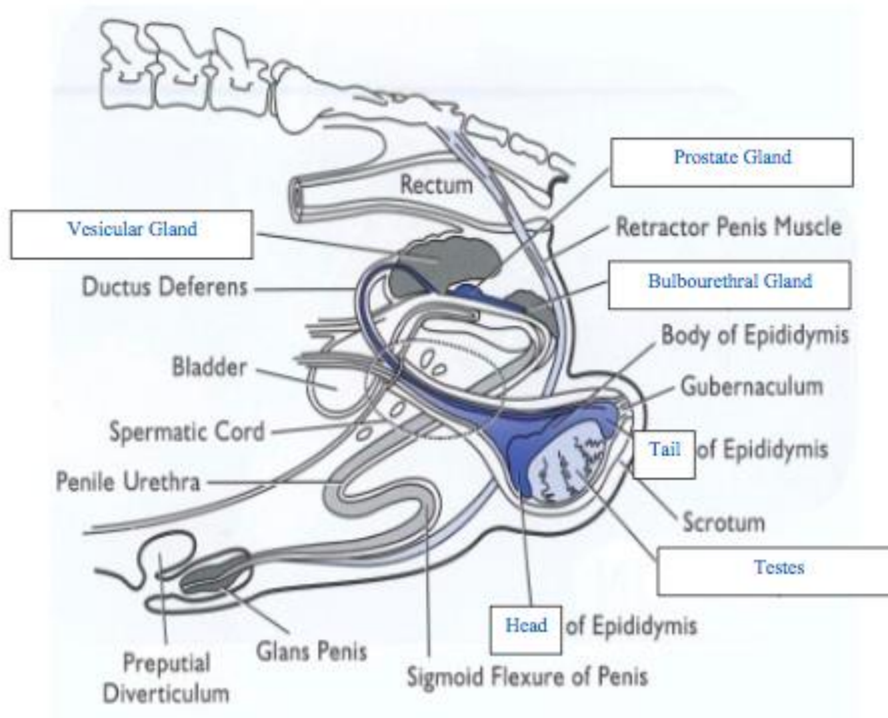
Question 1: Relationship of the Germ Cells to the Adjacent Sertoli Cells.



Question 2: The table below highlights the major events occurring during the journey of a sperm.

	Event/Process		
	Spermatogenesis	Maturation	Storage
Location	Seminiferous Tubule	Head and body of the epididymis	Tail of the epididymis
Fertile (YES/NO)	NO	NO	YES

Question 3: Boar Reproductive Tract



Question 4: What controls the ejaculatory process?

Arc Reflex:

Sensory stimulation of the glans penis (receptor), Sensory nerve fiber to spinal cord

Integration in spinal cord

Motor nerve fiber to effectors, contraction of the ejaculatory muscles (urethralis; bulbosponguis and isochiocavernosus)

Question 5: Write a brief description of semen collection and the changes in composition of the ejaculate during semen collection from a boar

1. *Wash hands well*
2. *All the equipment should be PREWARMED*
3. *Use a DUMMY or a real sow in heat to STIMULATE the boar*

4. *Grasp the penis with WARM/GLOVED hand and squeeze hard*
5. *Separate the ejaculate as follow*
    a. *Clear seminal fluid = waste*
    b. *Creamy sperm rich fraction = collect into a prewarmed thermos through a filter*
    c. *Gel fraction = waste*
6. *After ejaculate collected, evaluate sperm density and color*
    a. *Motility – under microscope*
    b. *Color – Yellow (urine contamination) Pink (Blood contamination)*

Question 6: Seminal Plasma comes from

Seminal Vesicular Gland

Question 7: Gel fraction from boar semen comes from

Bulbo-urethral Gland

LAB #8 Female Reproduction

Question #1: Where does ovulation occur on the follicle?

At the stigma.

Question #2: Describe the rupture of the follicle, the release of follicular fluid, cumulus mass of cells and egg from the follicle. How is the cumulus cell mass detached from the follicle?

The stigma will begin to hemorrhage 15-30 minutes before the rupture occurs. The follicle wall thins and bursts under hydraulic pressure.

The follicular fluid escapes first, very quickly.

The release of cumulus mass of cells (containing the egg) is next and occurs more slowly.

The egg is released enclosed in a tight mass of epithelial cells called the corona radiata.

The follicle is not completely emptied. Some of the sticky material surrounding the egg remains inside the follicle and keeps the mass of cells attached to the follicle until the ciliated cells of the fimbria of the oviduct remove it.

Question #3: How long does the process of ovulation take (from the point of follicle rupture)?

<2 minutes

Question #4: How is the cumulus mass transported by the fimbria into the ostium of the oviduct?

Via the action of ciliated cells

Question #5: How is the cumulus mass transported through the ampulla to the ampullary isthmic junction?

Via peristaltic contraction of the oviductal musculature.

Question #6: Where does fertilization occur? How long will the egg reside there?

Fertilization occurs at the ampullary-isthmic junction, and it will reside there for 15-18 hours.

Question #7: What is the difference between the ovaries of a horse and say, those of a pig or cow?

Horse ovaries are inside out

Question #8: The lining of the uterus is called the:

Endometrium

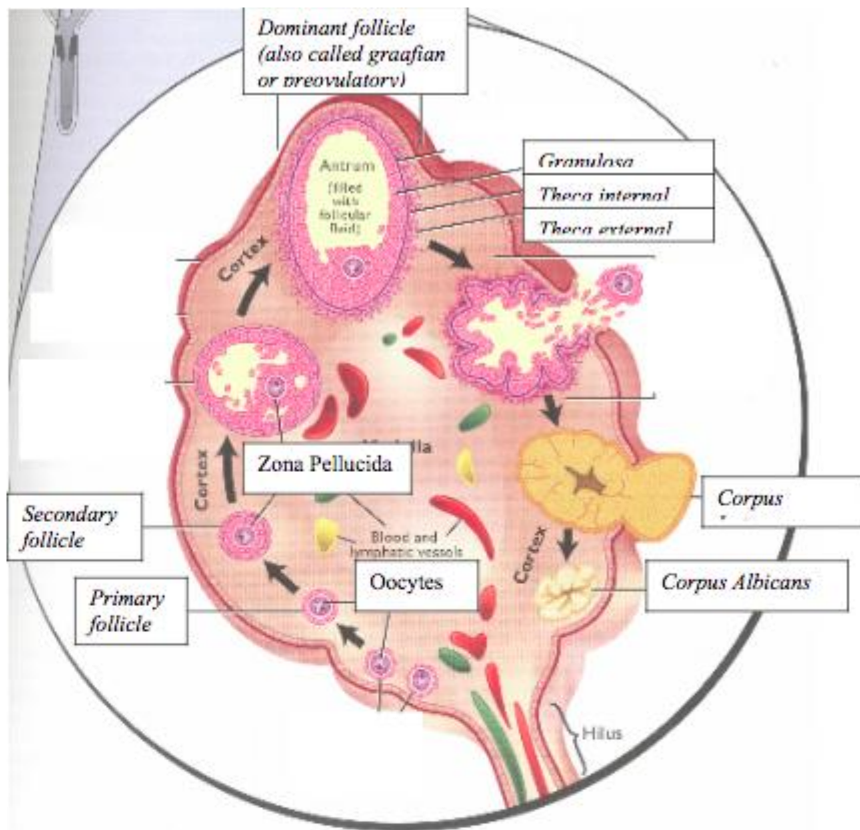
Question #9: The muscular layer in the wall of the uterus is called the:

Myometrium

Question #10: What is a caruncle?

A button-like area of the uterine endometrium that will form the maternal side of the placenta.

Question #11: Label the following diagram of an ovary:



Question #12: Label the following diagram of a cow reproductive tract:

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