Disclaimer

To all radiology residents….

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2. The outline for the database is roughly based on the ACVR 2003 written examination objectives.

3. This database will only grow through additional resident submissions. If you use this information please submit a posting in return. If you feel that a posting is inaccurate, or incomplete you are encouraged to post your own answer to that objective. If you would like to make a posting simply e-mail the completed objective to Matt Wright at wrighm3@vetmed.auburn.edu

4. If you have any questions or concerns, do not hesitate to contact me.

Good luck on boards and thanks to everyone that has already contributed.

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ANATOMY

1. General
2. General Musculoskeletal System (canine, feline, and equine)
    1. GENERAL_BONE_GROWTH_AND_FORMATION
    2. STRUCTURE_AND_NUTRITION_OF_CARTILAGE
3. Axial Skeletal System (canine, feline and equine)
4. Appendicular Skeleton (canine, feline, and equine)
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    2. COMMON_CALCANEAL_ULTRASOUND
5. Arthrology (canine, feline and equine)
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    1. AORTIC_ARCH_ANOMALIES
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7. Nervous System (canine, feline and equine)
    1. ANATOMICAL_RELATIONSHIPS_OF_THE_SPINAL_CORD
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    3. NERVE_SUPPLY_TO_HORSE_DISTAL_LIMB_NERVE_BLOCKS
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8. Digestive System (canine, feline and equine)
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9. Respiratory System (canine, feline, equine)
    1. MICROSTRUCTURAL_ANATOMY_OF_THE_LUNG
10. Urogenital (canine, feline, equine)
    1. ANATOMY_OF_THE_KIDNEY_DOG_HORSE_LLAMA_COW
    2. EMBRYOLOOGY_OF_THE_UROGENITAL_TRACT
11. Miscellaneous (canine, feline, equine)
    1. AVIAN_AIR_SAC_ANATOMY_AND_PULMONARY_STRUCTURES
    2. RECUMBENCY_&_POSITION_OF_THE_THORACIC_RADIOGRAPH

General Bone growth and formation:

Submitted by: Matt Wright

- All bone is derived from mesoderm
    - Initial formation results from mesenchymal cells that align with one another and cause local humeral interaction leading to cellular differentiation
- Osteoprogenitor cells are pluripotential mesenchymal cells that have the ability to differentiate into osteoblasts, chondroblasts, fibroblasts, etc.
- Cell types:
    - Osteoblast:
        - Synthesizes matrix and osteoid
            - Type 1 collagen is 90% of the organic component of bone matrix
                - As bone matures, osteoid becomes mineralized
                - Osteoid composes 35% of the bone mass with inorganic matrix making up the rest
            - Calcium hydroxyapetite makes up the majority of the inorganic matrix
        - There is a 12-15 day lag between osteoid production and mineralization of the matrix
    - Osteocyte:
        - As the osteoid lays down osteoid, the osteoblast loses it's ability to produce osteoid and becomes an osteocyte
        - Osteoblasts on the bone surface that are surrounded by mineralized matrix. It rests in a **lacunae** and has branching communications or **canaliculi** with its neighbor.
        - Osteocytes are the most numerous cells in bone
        - Osteocyts are connected by a system of tunnels called canaliculi
    - Osteoclasts:
        - Multinucleated cells formed by circulating mononuclear precursors
        - These function to resorb bone
        - Calcium and phosphorus are released with bone breakdown and absorbed by the osteoclast which uses them to maintain homeostasis of these nutrients
        - Osteoclasts and osteoblasts work as a unit (the basic multicellular unit)
    - Osteoprogenitor cells are also present in the inner cambium layers of the periosteum and endosteum
        - With injury of disease these cells can be stimulated to form bone.

Apophysis: other secondary ossification center that contribute to the shape of the bone but not bone growth. For example, the greater trochanter of the femur.

- Located in areas of ligamentous or tenidnous attachment
- These areas will ultimately fuse to the associated bone

Endochondral ossification:

- Mesenchymal progenitor first differentiate into a catilagenous model the forms the framework for bone formation
- Chondrocytes do their business and ultimately die within the matrix
    - This area of cell death forms the scaffolding for ingrowth of blood vessels and osteoperogenitor cells that differentiate into osteoblasts.
- A perichondral cuff of undifferentiated mesenchyme surrounds it

- The perichondral cuff ossifies (intramembranous ossification) to become the periosteum
- In a long bone
  - the model of endochondral ossification is located in the physis and metaphysis where active bone formation takes place.
  - The metaphysis is known as the primary center of ossification
  - The physis is the cartilaginous model that leads to the development of metaphyseal bone
    - The physis an metaphysis contribute to increased length and width toward the diaphysis
  - Within each epiphysis is a secondary center of ossification that develops from a cartilaginous model
    - The epiphysis contributes to increased length and width of the bone toward the end of the bone.
- Areas of active bone remodeling (metaphysis) is the predominant location for primary bone tumors.
- There are four zones to a physis
  - Reserve zone: on the epiphyseal side
  - Zone of proliferation
  - Zone of hypertrophy
    - Physeal fractures happen through this zone
  - Zone of mineralization
- With maturity, hormonal changes cause the plate to turn to bone.
  - Factors involved
    - GH
    - Thyroid hormone
    - Sex hormones
  - Large dogs have longer physeal closure times than small dogs

Intramembranous ossification:

- Mesenchymal progenitors differentiate into al layer of fibrous tissue which then undergoes further differentiation into osteoblasts.
  - These bones develop in sheets of connective tissue – not cartilage
- Remember…ossification is a process by which the organic matrix is produced with subsequent mineralization of the inorganic matrix.
- Mesenchymal cells mature to osteoblasts which secrete osteoid at multiple centers of ossification
  - Also, as osteoid is laid down, the cells are trapped in lacunae
  - Initially bone is woven but later turns to lamellar bone.
- Most physeal growth is longitudinal but in the zone of ranvier and subperisteal locations, appositional growth contributes to circumferential expansion.

Types of bone:

1. Lamellar: collagen forms in parallel arrangements
   a. Requires a cartilage precursor
   b. As the primary spongiosa (metaphysis) is replaced by new bone during remodeling there is an organized pattern that is seen histologically as layers.
   c. IN young animals, trabecular lamella is present in the epiphysis and metaphysis and extends toward the central diaphysis
      i. Subchondral bone is a thickend areas of trabecular bone at the end of the epiphysis.

    d. With age, the trabecular pattern recedes from the diaphysis and metaphysis
    e. There is a lack of cancellous bone n the diaphysis of and adult long bone.
2. Woven Bone: random collagen organization and can be deposited de novo and laid rapidly. Normally it is removed to lamellar bone

References: this is basically a summary of the bone formation section in the 4th edition of thrall with a few other tidbits thrown in.

Structure and nutrition of articular cartilage:

A complex structure in which fine fibers within the matrix are arranged in arches. Splitting in disease follows the fiber course. So superficial lesions lead to tangential flaking where those that extend deeper lead to cracks.

It is insensitive and avascular:
Oxygen and nnutritive requirements are met by diffusion from three sources:

1. joint fluid
2. vessels in the periphery of the cartilage
3. vessels in sujadjacent amrrow spaces

Diffusion is assisted by the porosity of the cartilage matrix which soaks up and releases the fluid as it is loaded and unloaded.

General composition of articular cartilage:

- sparse chondrocytes embedded in a hydrated gel of proteoglycans and entrapped in a collagen framework
- The proteoglycan matrix are sugars that attract water
    - Chondroitin sulfate
    - Keratin sulfate
    - Aggrecan
    - Etc

Synovial fluid has hyaluronic acid glycoproteins secreted by type B synoviocytes

- Synovial fluid is really like an extracellular matrix as there is no epithelial layer over the synovium so that there is a continual exchange of oxygen, carbon dioxide and nutrients between the blood and synovium.

collagen is parallel to the surface superficially which resists shearing

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Ultrasound imaging and anatomy of the bicipital tendon (canine and equine)

Submitted by: Matt Wright

Canine:

1. Anatomy:
    - Origin: supraglenoid tuburcle
    - Insertion: Radial tuberosity
    - Additional:
        - An extension of the shoulder joint invaginates the tendon and forms the bicipetal bursa
        - It is held in place in the bicipetal groove by a small transverse humeral ligament
        - Distal to the groove the tendon blends into the muscle belly
2. Ultrasound
    - Technique and normal appearance:
        - Brachicephalic and pectoral MM are a natural standoff
        - Tendon in homogenously hyperechoic
        - Flex the shoulder with some supination
        - Proximally it is circular and gets flatter distally but the cross sectional area is constant throughout.
        - The biceps bursa is seen a as a small accumulation of fluid medial to the tendon near the insertion on the muscle
        - A small amount of joint fluid is seen under the tendon at the origin on the surprglenoid tuburcle.
        - The surface of the intertuburcular groove is smooth.
    - Abnormal:
        - Lucent line around the tendon due to fluid accumulation in the tendon sheath
        - Enlarged hypoechoic tendon with fiber disruption
        - Irregular or Proliferative synovial lining.
        - Irregular surface of the groove
        - **Fluid within the bursa is a nonspecific finding and may be due to inflammation of the shoulder joint.

Supraspinatous tendon (canine):

3. Anatomy:
    - curves around the neck of the scapula and inserts on the greater tuburcle.
    - The tendon is short with a broad region of insertion.
        - Part of the insertion is on the medial aspect of the tuburcle near the biceps bursa. Calcification in this region is reported as a cause of lameness.
    - It lies immediately beneath the skin.
4. Clinical correlates:
    - Calcifying tendinopathy
        - More likely associated with lameness if is in the region near the biceps bursa.
        - Ultrasound is useful in differentiating
            - Supraspinatous calcification
            - Sclerosis of the bicipital groove
            - Calcifying biceps tendinopathy.

Bicipital Tendon – Horse

- Anatomy
    - Origin: supraglenoid Tuburcle
    - Insertion: radial tuberosity
        - Medial collateral ligament of the elbow
        - ECR tendon
    - Additional
        - The tendon conforms to the shape of the intertuberal groove
        - The tendon has two lobes. The lateral is bigger than the medial
            - An isthmus attaches the two lobes
            - The isthmus lies directly cranial to the intermediate tuburcle of the humerus.
            - The lateral lobe rides over the greater tuburcle of the humerus
            - The medial lobe rides over the lesser tuburcle
            - A tendinous band between the greater and lesser tuburcle anchors the tendon in the intertubural groove.
        - The biceptial bursa runs between the tendon and the intertuburlar groove.
- Ultrasound and abnormals
    - Bicepital bursitis: may just have an increased amount of fluid in the bursa
        - Thickening of the bursa and the synovial lining are present
        - Adhesions between the tendon and bursa
        - The tendon may be normal

- Biceps tendonitis:
  - Standard tendonitis stuff
  - Always evaluate the underlying bone. Chips and Osteomyelitis have been reported.

Common Calcaneal Ultrasound in the dog and horse

Submitted by: Matt Wright

Canine:

- Anatomy:
  - everything that attaches to the calcaneal tuberosity
    - SDFT*: This tendon lies cranial to the gastroc proximally but crosses medially to lie caudally at the insertion on the calcaneus.
    - Gastroc*:
    - Biceps Femoris
    - Gracilis
    - Semitendinosis
  - Calcaneal Bursa: Between the SDFT and the Gastroc proximally and distally between the SDFT and the plantar ligament.

- Technique:
  - IN the mid tibia region the SDFT and the Gastroc are equal size.
  - Near the calacaneus, the SDFT flattens and becomes broader. At this point the Gastroc is more rounded and is twice the size of the SDFT.
  - The fluid within the bursa is not usually visible.

- Uses:
  - Will differentiate tendon edema from those with tendon tears which is important because surgery is recommended for the tears but conservative therapy for tenosynovitis.
  - One study with 6 dogs says U/S is good to localize tears but cannot differentiate partial from complete tears. In humans this can be done.

Equine:

- Anatomy:
  - The calcaneal bursa is large and lies under the SDFT between the SDFT and Gastroc. It runs from the distal 4th of the tibia to the midmetatarsal region.
  - The gastrocnemius bursa is on the lateral side of the leg and usually communicates with the calcaneal bursa. It lies between the gastrocnemius tendon and the tuber calcis.
  - A subcutaneous bursa may also be present(capped hock).
  - Just like in the dog, the SDFT will wind medially to assume a caudal position over the point of the hock
- SDFT:
  - Injury is usually traumatic in this area. It may be sole or in conjunction with the gastroc tendon.

- Gastrocnemius Tendon:
    - Diffuse injury is more common the focal injury
    - Usually occurs without concurrent SDFT tendon injury
    - Calcaneal bursitis may accompany

Congenital abnormalities of the aortic arch

Submitted by: Matt Wright

- Abnormalities may be more common than reported because many are asymptomatic. Only a small percentage are significant

Persistent Aortic Arch: aka dextroaorta with a left sided ligamentum arteriosum

- 6 branchial arteries connect the primitive paired ventral and dorsal aortas
  - ventral aorta becomes the heart
  - dorsal aortas become the descending aorta
- L4: normally becomes the aorta
- R4: normally becomes the right subsclavian and brachiocephalic
- PR4AA: the right fourth aortic arch persists to become the permanent aorta
- The vascular ring is formed by the
  - Right ascending aorta: which still courses on the left side of the thorax but it must cross midline and forms the ring as it crosses the esophagus
  - Ligamentum arteriosum
  - Main pulmonary arterial segment
  - Heart base
- No vascular ring may be formed if there is a mirror image transformation of the aortic arches where everything is reversed if the DA also originates from the right aortic arch.
- On radiographs, the normal slight rightward curvature of the trachea is not present in the cranial thorax. Instead, the trachea is on midline or on the left
- May be associated with a persistent left cranial vena cava which is usually not of clinical significance

Aberrant Subclavian Arteries: both of these constrict the esophagus

- **Aberrant Right Subclavian:** aka dysphagia lusoria
  - Arises directly from the aortic arch or with the left subclavian in a bisubclavian trunk
  - The esophagus is constricted dorsally but no complete ring is formed
  - As the right subclavian crosses retroesophageally from the left to right it causes an indentation or half ring stricture of the esophagus
- **Anomalous left subclavian**
  - The right aortic arch develops instead of the left and the left subclavian crosses retroesophageally on the left side
  - This is part of the persistent right fourth aortic arch malformation
  - The constriction caused by the left subclavian on the esophagus will be more cranial than the heart base!!

Double Aortic Arch

- A vascular ring anomaly due to the complete persistence of both sides of the fourth arch
    - The ascending aorta divides into a right branch passing right and caudal to the esophagus and a left branch that passes left and ventral to the esophagus.
    - They fuse distally to form the descending aorta
- In one report the right one as functional and the left was atretic. The dog also had respiratory distress due to tracheal ring malformations.
    - This is the only vascular ring anomaly where tracheal compression will cause clinical signs.
- Can be treated by ligating the smaller of the two aortas.

Aortic Coarctation

- A severe narrowing of the aorta caudal to the level of the ductus arteriosis
- Results in hypertrophy and left heart failure due to increased afterload
- Notching of the caudal margins of the ribs due to high pressure collateral circulation through the tortuous intercostals arteries may be seen
- Pathophysiology:
    - Spread of specialized ductal tissue into the aorta. After birth the ductus closes and the aorta constricts
    - There are large ostia for the intercostals arteries in the descending aorta that offer collateral circulation
    - In humans, it may cause left heart failure in the young or found incidentally in the workup for hypertension in an adult
    - Two lesions are possible
        - Juxtaductal: a localized constriction in the region of the ductus
        - Tubular: diffuse narrowing of the isthmus proximal to the ductus
- In three dogs, one was found incidentally and two died in infancy
- Radiographically
    - Indentation of the aorta with dilation on either side
    - Approach from the common carotid artery for angiography

Persistent Left Cranial Vena Cava: (Buchannon JAVRS 1963)

- The abnormal left cranial vena cava will pass along the left border of the heart and cross over to the right side caudal to the left atrium where it joins the right atrium
- Usually considered incidental. This is a remnant of a normal fetal structure.
- Most common in German Shepherds (as is PR4AA)
- Can cause surgical exposure problems when correcting a vascular ring anomaly
- Can ligate it as long as the right cranial vena cava is normal and present

- Usually drains into the caudal part of the right atrium into the coronary sinus. Not a problem unless associated with entrance into the left atrium
- Normal in rabbits
- Seen associated with persistent right fourth aortic arch

References:

- The best single reference which has excellent diagrams of these abnormalities is the old Ettinger book simply called Canine Cardiology
- Poteet's review of thoracic imaging (which is posted in the residents section) from a previous ACVR review

Aortic Stenosis

Submitted by: Matt Wright

Anatomy:

- Usually subvalvular due to a large muscular ridge in the area
    - Two types:
        - Caused by a fibrous ring below the valve – static
        - Dynamic obstruction caused by hypertrophy of muscular ridge. (the same problem as with HCM). These dogs may only be obstructed if excited.
- Supravalvular is extremely rare but may be seen in cats
- Valvular is also rare but can be seen in association with subvalvular
- Also associated with valve dysplasias
    - Mitral dysplasia
    - Immovable, stiff aortic leaflets +/- fusion

Sig: Neuf, golden, GSD

Pathophysiology:

- Increased afterload cause post-stenotic dilation of AA, aortic arch, and brachiocephalic trunk
- Left ventricular hypertrophy/IVS hypertrophy: ischemia leads to ventricular extrasystoles
    - Thickening usually correlates to degree of obstruction
- May have a to and fro murmur of AS/AR possibly due to involvement of the valves in the fibrous ring or jet lesions causing valve abnormalities or endocarditis
- Leads to mitral regurgitation
    - Due to mitral dysplasia
    - Due to venturi effect and systolic anterior mitral valve motion
- Left atrial enlargement
    - Mitral insufficiency
    - Diastolic dysfunction and backup
- Eventually leads to LHF
    - Usually exertional syncope or sudden death.

Diagnostics:

1. RADS:
    a. +/- pulmonary venous engorgement
    b. Left atrial enlargement
    c. Seen as a widening of the caudal mediastinum in the VD view
    d. Elongation of the LV due to hypertrophy
    e. Loss of cranial cardiac waist
    f. Loss of caudal cardiac waist.

2. CATH
    a. Pressure gradient across the valve: 40-50% less than an echo reading due to anesthesia
    b. Small LV
    c. Subvalvular obstruction
    d. Mitral Regurg
    e. Must do a supravavular injection to RO aortic insufficiency
    f. Left coronary AA and major branches are prominent due to increased oxygen demand.
    g. Nonselective angio: in normal dogs the widest part of the ascending aorta should be the sinus of valsalva just distal to the aortic valve. Therefore, if the ascending aorta is wider there is AS.
3. ECHO
    a. Early systolic closure of the AV valve because of the obstruction
    b. Myocardial fibrosis an increased echogenicity
    c. LV systolic function is preserved
    d. Doppler:
        - Altered flow profile as peak velocity is reached closer to systole- dagger shape
        - Do not mistake mitral regurg for aortic flow
        - A deviation of 15-20o will underestimate severity

        - <50mmHg= mild, no clinical signs
        - 50-80mmHg=moderate
        - >80mmHg poor prognosis

        - >2.50m/s=As
        - <200=normal
        - 2.0-2.50m/s is the gray zone where these velocities can be seen in athletic animals or minimally affected animals with aortic stenosis

        **An accurate gradient cannot be measured if there is an associated PDA, insufficiency etc.
        **With mild to moderate gradients the main complication is endocarditis.

Prognosis

1. Depends on the Doppler
    a. 75mmHg may be normal
    b. >100-125 sudden death
    c. prone to endocarditis
    d. A 50% decrease in gradient with a vavluloplasty is common
    e. Poteet says “treatment very limited – poor prognosis”

Atrial Septal Defect:

Submitted by: Matt Wright

Developmental Anatomy:

- A failure of closure of the
    - Ostium primum: common in cats as part of an endocardial cushion defect (seen with a concurrent VSD and AV vale malformation), seen low in the septum. These defects are usually very large. These result in CHF.
        - You can remember which is which in that this one is located at the junction of the AV valves and the septum.
    - Ostium secundum: a true patent foramen ovale. This is present high in the septum. Common in dogs. These defects are well tolerated.
- A persistent foramen ovale is not really a septal defect but is it doesn't seal the foramen may be pushed open (elevated RHP)
- In the normal animal, the lower and upper septum are connected by the growth and differentiation of the endocardial cushion

Pathophysiology

- A left to right shunt unless there is another reason for a high right hear pressure such as PH, pulmonic or tricuspid malformation
    - Any increase in RH will keep the foramen ovale open as in the fetus as the pressure will prevent it from closing
    - Elevated LA pressure with mitral stenosis will also keep it open.
- Usually the ASD causes few clinical signs
- A murmur is due to relative stenosis across the pulmonic.
- Only see right sided cardiac enlargement.
- Dilated MPA with pulmonary over circulation.
- Overall, volume overload of the right heart with enlargement.
- Commonly asymptomatic
- Oxygen step up in the right atrium.

Echo:

- Seen in a right parasternal long axis view
- Ostium Secundum shows septal dropout in the middle of the septum
- Varying degrees of volume overload of the MPA, RV, RA and LA
- Paradoxical septal motion is seen with elevated RH pressures
- Jet lesion and turbulent flow on color doppler
- Increased pulmonary artery velocity
- Ostium Primum Defect: AV valves will be abnormal because the annulus of these valves is located at what would be the septum that is not present. Both valves may be dysplastic and insufficient.

- With reversal there is severe RV hypertrophy and prominent MPA with elevated aortic flow velocity.

RADS:
- Enlarged MPA and RH
- Increased pulmonary vascularity unless there is increased PVR
- LA is normal to slightly enlarged. If severely enlarged, suspect and endocardial cushion defect with an anomalous MV.

Treatment is limited:
1. Pulmonary artery banding to prevent L-r flow
2. patch graft required bypass..

ECG: May be normal or show evidence of RA or RV enlargement. P pulmonale or an increased P-R interval are common

Angiocardiography: demonstration of an ASD may be difficult as selective LA injection is tough to accomplish. A PA injection is often times diagnostic. Agiocardiography may also reveal concurrent defects and can be used to quantitate shunt fraction.

Oximetry: Can be used to assess the degree of shunting. Blood oxygen in RA is increased compared to CrVC indicating shunting at the level of the atrium

Pressure measurements:
1. with CHF there is increases CV, RA, and RV diastolic pressure increases
2. RV systolic pressure is normal or elevated when there is pulmonary hypertension or a large L-R shunt.
3. High flow across the pulmonic valve creates a relative pulmonic stenosis and of 5-20mmHg are common.

Blood supply to the femoral head:

Submitted by: Matt Wright

Immature Animal:

- The vessels:
  - Lateral and medial circumflex femoral contribute 70%
    - These arise off of the femoral artery
  - Minor supply from the caudal gluteal and cranial gluteal

- The anatomy
  - These three vessels form an extracapsular arterial ring at the base of the femoral neck the supplies the whole hip joint
  - The arteries pierce the joint capsule and give rise to a series of ascending cervical arteries that ascend the femoral neck within the synovial reflections of the joint capsule
  - They cross over the periphery of the capital growth plate and enter as the epiphyseal artery
  - There is an artery of the ligament of the head of the femur but it does not sufficiently supply the head of the femur

- The problem
  - A physeal fracture will compromise the physeal barrier and healing must occur through metaphyseal vessels
  - An increase in joint fluid, subluxation of the femoral head, trauma to the femoral neck or Salter Harris of the capital growth plate can limit the blood supply
  - Therefore, capital femoral physeal fractures do not fit the general Salter Harris classification

References: Compendium Nov 1996 (I do not have the full reference just a picture of the vasculature from this article)

Development of the cardiovascular system: a process of growth and degeneration

Submitted by: Matt Wright

Early formation of the heart

Stage 1. Formation of the heart tube.

- Mesodermal cardiogenic tissue migrates from the head to the thorax with growth
- Heart begins as two tubes located laterally. With fetal folding the two tubes are brought together for form a single tube. This is the endocardial tube. It fuses cranially to caudally and us ultimately covered with myocardium and epicardium.
- The truncus arteriosis differentiates and starts to beat. It is the common outflow at this point.
- Ventricles and atria form with fusing of the tube.
- The sinus venosus remain paired until much later. They receive venous blood.

*With appearance of each new chamber there is an increase in heart rate. This is known as the pacemeker phenomenon.

Stage 2: Formation of the cardiac loop

- The tube is anchored cranially and caudally and it bulges ventrally and caudally as it grows. Thus the ventricle moves caudal to the adult position.

Stage 3: partitioning of the heart

- Partitioning of the common atrioventricular pathway: Atriventricular endocardial cushions grow toward each other.

- Partitioning of the common atrium:
    - Septum 1: grows down toward endocardial cushion leaving foramen 1. Before it gets to the bottom Foramen 2 forms cranially.
    - Septum 2: Grows down parallel to Septum 1 and covers Foramen 2 leaving and the Foramen Ovale develops in the mid portion of septum 2. Septum 1 acts as a flutter valve permitting fetal circulation across the foramen ovale and foramen 2.

- Partitioning of the Ventricle and outflow tract: The interventricular septum grows upward toward the endocardial cushion leaving and opening – the interventricular foramen.

- Partitioning of the Truncus Arteriosis:
    - Truncal ridges form and grow toward each other and spiral around one another forming the spiral septum. These two parallel, spiraling outflow channels represent the pulmonary and arterial vasculature.

- Closure of the interventricular foramen: as partitioning of the truncus arteriosis continues, the two edges of the truncal cushions grow toward the endocardial cushions closing off the IVS and finishing the two outflows. The membranous part of the septum is the last to close.

Aortic Arches: <u>only the 3rd,4th,and 6th arches are retained</u>

- Simultaneously two ventral aortae fuse to form the aortic sac (heart) and the dorsal aortic arches form vessels
    - Some arches degenerate and some persist
        - Right 6th: pulmonary trunk and pulmonic circulation
        - Left 6th: distal portion retains its connection with the dorsal aorta as the ductus arteriosus
        - Left 4th: aortic root (and joins with the left dorsal aorta)
        - Right 4th: root of the subclavian
        - Two 3rd: internal carotid carotid AA

References:

- Fox: Canine and feline Cardiology (good pictures)
- Pasquini: Anatomy of Domestic Animals

Differences in fetal and post fetal circulation (all species)

Submitted by: Matt Wright

- Blood is shunted to fetal brain and heart by three shunts
  - Ductus Venosus
  - Foramen Ovale
  - Ductus Arteriosis

- Umbilical Vein: From mom to portal vein via the ductus venosus to CVC
  - CauVC goes to RA and 3/5 of the blood is shunted across the Foramen Ovale
  - CrVC returns oxygen poor blood from cranial parts of the body to RA and this mixture (of unshunted umbilical vein blood) gets shunted away from the lungs via the ductus arteriosis.
    - It enters caudal to the brachiocephalic trunk so it does not rob the oxygen rich blood going to the brain.
  - The umbilical arteries are the terminal branches of the aorta (internal iliac) return blood to the umbilical cord to placenta for oxygenation.

- At birth:
  - As umbilical vein is disrupted, the ductus venosis become part of the portal circulation
  - As lungs expand, vessels open and resistance to blood flow drops allowing more blood from the pulmonary arteries to reach the lungs. Since the DV is a muscular artery it begins to close due to the change in oxygen content of the blood.
  - Increasing blood flow to the left atrium increases pressure against the FO causing functional closure. With time it becomes a functional closure…the fossa ovale.

Patent foramen ovale is normal in calves

In foals, the ductus arteriosis is functionally closed at 16 hours

Endocardial Fibroelastosis
Submitted by: Matt Wright

- Diffuse thickening of the endocardium but collagenous and elastic tissue
- Pathogenesis:
    - Subendothelial fibroblastic proliferation secondary to localized, chronic, cardiac lymphedema (I believe this is from a Fossum article but don't quote me on it!!☺

- Appearance
    - Diffuse thickening of the endocardium
    - Dilation and hypertrophy of the ventricles and dilation of the left ventricle
    - Narrowing of the aortic outflow tract
    - Mitral and tricuspid are thickening and malformed
    - **This is a left heart disease. Enormous heart.**

- Radiographs:
    - Signs of severe general heart failure
    - A globular, large cardiac silhouette with or without a visible enlarged left atrium in kittens with signs of heart failure or thromboembolism is suspicious.
- Angio:
    - Moderately to severely dilated left ventricle
    - Atrial dilation
    - AV regurgitation
    - Ventricular borders are smooth
    - Ventricle barely changes in size between systole and diastole and has a rigid appearance
    - IVS may bulge toward the right
    - Poor contractility causes failure of mixing of the blood and may stream(this may mimic an uneven endocardial surface)

Siamese predisposed
This resembles a restrictive Cardiomyopathy. However, the chambers are huge (at least as described in Suter's Book).

Hepatic AV malformations

Submitted by: Matt Wright

Hepatic AV fistulas:

- Communications between the hepatic artery and portal veins.
  - Blood bypasses the hepatic sinusoids and flows retrograde into the portal system
  - Resulting in portal hypertension, ascites, multiple acquired PSS
    - Appear as large, thin walled, tortuous pulsating vascular channels that distort the involved liver lobes and elevate the overlying hepatic capsule
  - Right medial is most commonly affected.
    - Hepatic parenchymal changes are similar to that seen with PSS
- Secondary systemic changes are the same as for any AV malformation
  - Decreased peripheral vascular resistance and pooling of blood in the venous circulation
  - Increased blood volume, heart rate, and eventually high output heart failure (no dogs have failed and it is presumed that the sinusoids attenuate the increased portal venous pressure so secondary cardiac defects are minimized.
- Etiology:
  - Congenital: *AKA hamartoma or hemangioma*
    - Abnormal development of vessels before differentiation into arteries and veins
  - Acquired
    - Trauma, surgery, tumor, rupture of an aneurysm
    - No veterinary reports of acquired
- Clinical Signs
  - Less than 1.5 years old
    - **Acute** ascites, depression, vomit, diarrhea
      - Unknown why it is acute but it is
    - Also, HE, wt loss, poor doer
  - Due to sever portal hypertension
  - BRUIT over the abdomen
  - DDX Ascites in young animals
    - Remember, it looks the same as PSS but ascites is uncommon with PSS
    - Congenital heart
    - P-losing eneteropathy
    - P-losing nephropathy
    - **AV fistula should be considered in all cases of multiple PSS in young animals.**
- Diagnosis
  - Ultrasound: *Bailey Q: Ultrasonographic findings associated with congenital AV fistula in three dogs. JAVMA 192: 1099, 1988*

        - The fistula appears as a tortuous, anechoic, tubular structure in the area of the liver
        - Secondary PSS is multiple vessels.
        - Celiac Arteriography or nonselective aortography
            - Contrast outlines hepatic arteries that appear as multiple, dilated, tortuous vessels that communicate directly with the portal vein.
            - Normal arterial phase is absent because of hepatofugal flow into the PV.
            - Multiple PSS as the dilated portal vein fills.

- Treatment:
    - Surgical resection of affected lobes or de-arterilaization
    - Poor outcomes because of continued shunting through the acquired PSS>

Pulmonic Stenosis

Submitted by: Matt Wright

Signalment:

- Beagles, schnauzer, Chihuahua, bulldog, terrier
    - Anomalous coronary artery development in the bulldog and boxer
        - Left main CA originates from a single right coronary AA that encircles the stenotic RVOT

Developmental Anatomy:

- Valvular Dysplasia is the most common defect
    - Fusion or hypoplasia of valve leaflets
- Other possibilities in clude
    - Infundibular
    - Subvalvular
    - Supravalvular
- Also commonly associated with tricuspid valve malformation in larger breeds. However, a distinction between dysplasia and a secondary change is not always clear.

Pathophysiology:

- Increased afterload causes RV hypertrophy with bowing toward the left (or flattening)
- Stiff RV is responsible for vigorous atrial contraction (A-wave) in jugular venous furrow
- Diminished RV coronary blood flow
- Clinical signs are most severe with exercise, syncope, tiring, usually >1 year.
    - May see hypoxemia from shunting across a d PFO or ASD

Diagnostics:

1. ECG: RH enlargement
2. Cath:
    a. Pressure gradient across the PV
    b. Elevated RVEDP
    c. Elevated RA a wave
    d. Narrow orifice, dysplastic valves, RVH, secondary dynamic muscular obstruction in the RV infundibulum, enlargement of RCA, poststenostic dilation of the MPA
    e. A nonselective angiogram shows poststenotic dilation but the lesion may not be seen if a jugular or cephalic injection as there is overlap with contrast in the CVC and RA. May consider a caudal injection.
3. RADS:
    a. RV enlargement
    b. Poststenotic Dilation of MPA (1oclock)
    c. Pulmonary Underperfusion (small PA) or normal vessels.

d. Some may just have rounding of the right heart and no gross cardiomegally.

4. ECHO:
    a. Evaluate valve morphology. There are tree types of stenosis lesions:
        i. Thick immovable dysplastic cusps
        ii. Fusion of peripheral edges of the valves with a domed valve. In this type the severity is proportional to the degree of fusion
        iii. Infundibular hypertrophy
            1. may be primary or secondary. With time, 96% will have infundibular obstruction
            2. **Valvular or Subvalvular are most common**
        iv. Increased echogenicity of the valve.
    b. Post stenotic dilation of MPA:
        i. NORMAL at the valve and gets wider distally.
        ii. This differentiates from PH or PDA
        iii. The degree of dilation does not correlate to the severity of the obstruction.
    c. Septal hypertrophy with a **small chamber**.
        i. A large chamber indicates TS, PI, or shunt in addition
    d. RA dilation because 50% have associated tricuspid dysplasia
    e. Pseudohypertrophy of the LV which is really just decreased preload.
    f. Doppler:
        i. <50mmHg=mild
        ii. 50-75mmHg=moderate
        iii. >75mmHg=severe
        iv. Velocity may not really indicate the degree of obstruction if there is a shunt or PI as these two cases will increase volume and increase the derived pressure gradient.

Treatment:

- Cannot precisely say when to treat these but
    - if >100 do consider treatment
- Balloon valvuloplasty is only applicable if the valves are thin and the annulus is not hyperplasic.
    - A single right coronary artery is a contraindication
    - May develop a right BBB from increased intraluminal pressure during dilation
    - May fail as a result of postsurgical scarring
- Valvulotomy with a patch graft over the outflow tract is best but will not correct a severe subvalvular muscular hypertrophy.

Tetrology of Fallot

Submitted by: Matt Wright

SIG: keeshonds, English bulldogs

Anatomy:

- 2 lesions
  - Pulmonic Stenosis
  - VSD
- 2 consequences
  - overriding aorta
  - RV hypertrophy

Pathophysiology:

- Pulmonic stenosis leads to increased right heart pressure and hypertrophy
- Increased RH pressure leads to R-L shunting or deoxygenated blood through the VSD to the LV which is undercirculated
- Hear an ejection murmur of the PS
- This results in **hypoxemia, polycythemia, hyperviscosity, and cyanosis**

Diagnostics:

1. RADS:
   a. Right heart enlargement predominated without left heart enlargement. Overall the size is not huge
   b. Keep in mind that the MPA is not enlarged like a PS
   c. Small LA due to poor venous return.
   d. Filling of the cranial waist with an overriding aorta
   e. Hypoperfusion fo the lungs
   f. Some say normal heart size with rounding of the RV border
   g. “Couer Sabot boot shape heart
2. CATH:
   a. Nonselective angio: simultaneous filling of the PA and AO without contrast in LV
   b. Equilibration of R and Left systolic pressures
   c. Oxygen step down in the LVOT with desaturation of aortic blood
   d. RV hypertrophy, PS with minimal poststenotic dilation, VSD
   e. Anticoagulants shoul dbe considered to prevent cerebral thromobembolism. Also be warned o the air emboli.
3. ECG: right axis deviation
4. ECHO:
   a. RV hypertrophy
   b. Small LA and LV
   c. Subaortic VSD

d. Pulmonic Stenosis but the post-stenotic dilation of the MPA is not present due to unloading of the RV through the VSD.
e. The VSD is usually large and obvious
f. Bubble study can demonstrate R-L shunt (bubbles in LV lumen)

Prognosis
CHF is uncommon and animals may survive years if hyperviscosity is controlled

Treatment: requires bypass and creation of a L-R shunt

Anatomical Relationship of the spinal cord, spinal nerves, and meninges

Submitted by: Matt Wright

Spinal Cord: anatomy:

- The cord is divided into segments corresponding to the somites by the serial origins of the roots of the paired spinal nerves.
  - In addition to the dorsal and ventral roots (see below) the first seven (sometimes eight) cervical segments have rootlets that emerge mediolaterally from the spinal cord and join to form the **spinal root of the accessory nerve** which runs cranially through the foramen magnum
  - Remember: there is an extra spinal cord segment (C8) in the cervical region and there are only 5 caudal cord segments
- Central gray matter
  - Central canal is the residual embryonic neural tube
  - Dorsal and ventral horns:
    - Afferent neurons run (incoming) dorsally
    - Efferent neurons come in the ventral horn
    - Dorsal root ganglion
- White matter:
  - Divided into three funiculi on each side
  - The funiculi are composed of ascending and descending nerve fibers many of which are grouped into bundles (fasciculi or tracts)
  - These tracts connect the brain and interconnected the various spinal segments of the cord.
    - Examples include: rubrospinal tract, ventral spinocerebellar tract etc.
- Dorsal and ventral median fissure

Peripheral Nerves:

- 12 pairs of cranial nerves
- pairs of spinal nerves whose total varies with the vertebral formula
- The origin of the spinal nerves reveals the segmentation of the spinal cord:
  - Dorsal root: afferent (sensory) fibers whose cell bodies are clumped together to form a visible swelling – the dorsal root ganglion
  - Ventral Root: Efferent fibers emanating form the ventral horn of the gray matter
  - <u>These two roots join peripherally to the dorsal root ganglion to form the mixed spinal nerve that leaves the canal through the IVF.</u>
  - The mixed trunk divides almost immediately to the
    - **Dorsal branch**: smaller branch to dorsal structures (epaxial) only sensory and motor fibers
    - **Ventral Branch**: to the ventral structures (hypaxial)
  - Brachial and lumbosacral plexus:

        - Brachial Plexus: C6-T2
        - Lumbosacral Plexus: last few lumbar and first two sacral
    - Note: these nerves do not innervate blood vessels, glands, or internal organs. That is taken care of by the autonomic nervous system which is not present at every spinal nerve but the pattern is similar.
- Cauda Equina: The structure formed by the nerve roots leaving the caudal part of the spinal cord

Segmental relationships to vertebrae:

- The cord grows slower than the spinal column so it ends in the lumbar region. Therefore, the last spinal nerves must pass progressively caudally to exit form the IVF
    - The positional relationship between the spinal cord segments and the vertebrae is regionally variable
    - Spinal cord segments alternatively lengthen, shorten and lengthen again
    - Remember, spinal roots always travel to IVF formed by corresponding vertebrae so spinal root length reflects the location of the spinal cord segment relative to its numerically corresponding vertebrae
        - Spinal roots are short in the initial cervical area and TL region
        - The first spinal root exits through a lateral vertebral foramen in C1
        - C2-C7 exit through IVF formed by cranial margins of corresponding C2-C7 vertebrae
        - C8 exits through the IVF formed by the cranial margin of T1
        - T1-Ca5 exit through the IVF formed but the caudal margins of their corresponding vertebrae
            - The three sacral segments are in vertebral body L5
            - The 5 caudal segments are in vertebral body L6
- Initial Cervical Region: the first cervical segment lies within the corresponding vertebra
- Caudal Cervical through cranial thoracic region: segments are positioned cranial to their respective vertebrae
- Thoracolumbar region: segments lie within their corresponding vertebrae
- Caudal lumbar, sacral, caudal region where segments lie progressively cranial to their respective vertebra each nerve emerges cranial to the vertebra of the same number except the eighth that emerges caudally.

Meninges

Three Layers:

1. Dura Mater
    - Fused with the inner periosteum of the skull bones
    - The cranial venous sinuses are enclosed within the dura mater.
        1. there is no epidural space within the skull
    - Reflections of the dura:
        i. Falx cerebri:
        ii. Tentorium Cerebelli: it is ossified in it's median part
        iii. Hypophyseal fossa
    - Splits at the foramen magnum so it is separated from the vertebra by the epidural space in the spinal cord.
        i. Epidural space has fat and the internal vertebral venous plexus

2. Arachnoid membrane:
    - Separated from the dura mater by the subdural space that normally only has a minute amount of fluid.
    - The spinal part of the subdural space is crossed by s series of **bilateral denticulate ligaments** (extensions of pia) that attach the inner meninges to the dural tube and thus indirectly sling the cord. They anchor laterally.
    - Pia is connected to arachnoid by arachnoid trabeculae

3. Pia Mater:
    - Attached to the brain and the cord
    - Leptomeninges: Pia+Arachnoid

The caudal aspect of the cord

- **Conus Medullaris:** caudal to the lumbar intumescence the cord tapers into an elongate cone consisting of segments S2,3,Ca1-5. These segments become successively smaller and are surrounded by caudally directed spinal roots
- **Terminal Filament:** Approximately 1cm caudal to the spinal cord termination (last segment) the cord is reduced to a uniform strand of glial and ependymal cells called the terminal filament. A caudal extension of dura mater that envelopes the filum terminale is the **spinal dura mater filament**.
- **Lumbar Cistern:** A dural sac enclosing the subarachnoid space extends about 2cm beyond the end of the spinal cord before the dura mater constricts around the filum terminale
- **Cauda Equina:** Within the canal the sacral and caudal roots stream caudally beyond the conus medullaris to exit at their respective IVF. Collectively these roots are called the cauda equina. In the dog most of the cauda equina lies caudal to the lumbar cistern

References: Millers anatomy of the dog and Dyce, Sack, and Wensing.

The lumbosacral Plexus and innnervation to the hind limb

Submitted by: Matt Wright

The lumbosacral plexus consists of nerve roots of L4-S2

- **Femoral Nerve**
  - Exits through the psoas muscles to reach a gap between the dorsocaudal corner of the flank and the psoas muscles (femoral canal). It is accompanied by the external iliac artery and vein and runs between the sartorius and pectineus.
  - Innervates the extensors of the limb
  - Damage is severe as the stifle joint cannot be fixed.
    - Saphenous nerve: supplies the skin over the medial limb from stifle to metatarsus. And motor to sartorius

- **Obturator Nerve**
  - Follows the medial shaft of the ileum to pass through the obturator foramen
  - Adductor muscles of the thigh
  - Injury allows the limb to slip sideways as there are no adductor muscles

- **Common Lumbosacral Trunk:** gives off the other branches of the limb as it leaves over the ischiatic notch then gives off three branches. After that it continues as the sciatic
  - **Short cranial gluteal nerve**: flexors and extensors of the hip (gluteals)
  - **Caudal Gluteal Nerve**
  - **Caudal Cutaneous Femoral**
    - **Sciatic:** Supplies the caudal thigh muscles as well as the muscles of the crus and pes. It passes between the middle and deep gluteals then turns toward the thigh caudal to the hip joint where it is protected by the greater trochanter of the femur. Then it runs between the biceps femoris and the semitendinosis before dividing into terminal branches.
      - **Common Peroneal**: Courses over the lateral aspect of the leg and divides at the fibula and goes dorsal. It supplies the craniolateral muscles of the crus and superficial stuff distally. Paralysis produces overextension of the hock and flexion of the digits that will rest on their dorsal surface (knuckling)
        - Superficial peroneal
        - Deep Peroneal
      - **Tibial**: dives between the heads of the gastroc and supplies the caudal leg after it divides over the point of the hock. Damage will be overflexion of the hock and everextension of the digits.
        - Medial Plantar nerve
          - Lateral plantar nerve

Know the nerve supply to the horse distal limb as it pertains to common nerve blocks

Submitted by: Matt Wright

The thoracic limb:

- Radial nerve does not extend past the carpus in the horse
- **Median**: divides to medial and lateral palmar proximal to Carpus
    - Median palmar: solely median
    - Lateral palmar: in combo with ulnar
        - Give off dorsal branch at fetlock
        - Continue as palmar digital nerves
- **Ulnar:** divides to dorsal and palmar branches proximal to Carpus
    - Dorsal br: Descends on lateral dorsal limb
    - Palmar br: joins with lateral br of median
- **Communicating branch:**
    - Crosses at mid metacarpus from median to ulnar
- **Deep branch of lateral palmar nerve**
    - Continues as the medial and lateral **palmar metacarpal nerves**
    - Travel under buttons of the splint bones
- **Medial Cuntanous antebrachial**
    - Travels straight down the medial aspect of the dorsal limb right to the fetlock. An extension of the musculocutaneous.

The nerve blocks

- Palmar Digital Block:
    - Palmar digital nerves to the heel of the foot
- Abaxial Sesmoidean Nerve Block:
    - Medial and lateral palmar nerves
        - Palmar digital nerves
    - Dorsal Branch of digital nerve
- Low Four Point
    - Medial and lateral palmar
    - Medial and lateral palmar metacarpal
    - Cutaneous
        - Dorsal branch of ulnar
        - Medial cutaneous antebrachial
- High Four point
    - Medial and lateral palmar
    - Medial and lateral palmar metacarpal
- Proximal to carpus
    - Median and ulnar are blocked

The pelvic limb

- Tibial: divides just proximal to the tibiotarsal joint
    - Medial Plantar
    - Lateral Plantar
        - Deep branch:
            - Medial Plantar Metatarsal

- Lateral Plantar metatarsal

***Communicating branch connects the lateral and medial plantar nerves in the mid metatarsus
***The plantar nerves give off dorsal branches at the fetlock then continue in the foot as the medial and plantar digital nerves.

- Dorsal Limb
  - Saphenous Passes dorsomedially
  - Common Peroneal: branched to the superficial and deep peroneal at the stifle
    - Superficial Peroneal:
    - Deep Peroneal: At the tarsus it divides to become the medial and lateral dorsal metatarsal nerves (great metatarsal nerves) that travel between the cannon and the splint bones. They travel all the way to the foot.

**Don't forget that the dorsal digital extension from the plantar side of the limb come across at the fetlock.

References: Pasquini has an excellent review with cartoon pictures in the book Anatomy of Domestic Animals.

Origin of cranial nerves and their function

Submitted by: Matt Wright

I. **Olfactory**:
   a. Enters through the cerebrum (all others enter in the brainstem)
   b. Entirely sensory

II. Optic Nerve:
   a. Cell bodies are located in the retina
   b. Enters cranium through the optic foramen
      i. Crosses at the chiasm
      ii. Called optic tracts after they cross at the optic chiasm
      iii. Continues along the brainstem to the lateral geniculate nucleus as it arches around the diencephalon
   c. Goes to the thalamus then to the occipital lobe
   d. Entirely sensory
   e. Assessed by
      i. Visual placement reactions
      ii. Menace response
      iii. PLR

III. Oculomotor
   a. Arises in the midbrain
      i. Passes through the orbital fissure
      ii. Travels in the cavernous venous sinus on the way to the orbital fissure
   b. Mixed nerve
      i. Motor to the extraocular muscles and eyelid
         1. dorsal rectus
         2. medial rectus
         3. ventral rectus
         4. ventral oblique
         - ----the sensory comes from the CN 2-----
      ii. ANS motor to the ciliary body (pupil and accommodation)
   c. Assessed by
      i. PLR
   d. Damage:
      i. Strabismus

IV. Trochlear
   a. Arises from the dorsal surface of the brainstem (the only one to leave dorsally)
   b. Exits via the orbital fissure after passage in the cavernous venous sinus
   c. Motor to the dorsal oblique of the eye

d. The smallest cranial nerve.
e. Damage:
    i. Strabismus

V. Trigeminal:

a. Arises from the pons (the only one to arise from pons) (caudal extent)
b. A mixed nerve with three divisions
    i. Ophthalmic: sensory to skin of forehead, cornea, nasal cavity
        1. exits via the orbital fissure
    ii. Maxillary Division: sensory upper teeth, skin of face and oral cavity.
        1. exits the skull via the round foramen
        2. becomes the Infraorbital nerve traveling in the infraorbital canal
    iii. Mandibular Division: mixed
        1. exits the skull via the oval foramen
        2. motor to the muscles of mastication (masseter, temporalis, pterygoid)
        3. Sensory to lower cheek and jaw (mental nerve)
c. To assess
    i. Atrophy of muscles of mastication
    ii. Dropped jaw (idiopathic trigeminal neuropathy. Can live but hydration is a problem. Self limit for 21d)
    iii. Palpebral reflex (also 7)
    iv. Corneal reflex (also 7)

VI. Abducens:

a. Motor to the lateral rectus and retractor bulbi
    i. *Abduct is to move laterally –abducens—need lateral rectus*
b. Arises from the medulla (<u>thin and on midline</u>)
c. Exits the skull via the orbital fissure
d. Assess
    i. Cannot move eye laterally
    ii. Strabismus

VII. Facial

a. Originates from the pons
b. Exits the skull via the internal acoustic meatus to the stylomastoid foramen
c. Mixed nerve
    i. Innervates the muscles of facial expression
        1. Auriculopalpebral br: orbicularis oculi: wide eye

2. Ventral and dorsal buccal branch: crosses masseter" facial deviation
   ii. Sensory to the rostral 2/3 of the tongue (taste)
   iii. Autonomic to the lacrimal gland, sublingual and mandibular salivary

d. Assess
   i. Schirmers : lacrimation
   ii. Palpebral reflex (also 5)
   iii. Facial reflex (also 5)
   iv. Facial symmetry

e. The course of the nerves
   i. 7 and 8 run together through the external acoustic meatus then CN 7 crosses the middle ear cavity.
   ii. 7 leaves the skull through the stylomastoid foramen

f. Damage due to inner ear infection
   1. Peripheral: facial asymmetry with no effect on lacrimation
      a. Auriculopalpebral br: wide eye and ptosis
      b. Buccal br: deviation of face
   2. Middle ear infections:
      a. All branches are affected
      b. Open eyes, facial deviation, loss of lacrimation
      c. Horners
   3. Cranial cavity between brain and internal acoustic meatus
      a. All branches
      b. Also vestibular signs
      c. No horners

VIII. Vestibulocochlear

a. Two branches
   i. Vestibular: vestibular proprioception
   ii. Damage:
      1. head tilt
      2. leaning and circling toward affected side
      3. ataxia
      4. nystagmus
         a. vertical is central
         b. horizontal or rotary is peripheral or central
      5. Unilateral otitis
         a. Vestibular signs and horners and facial paralysis
   iii. Cochlear: hearing. Damage is deafness. Runs the organ of corti

b. Exits the skull via the internal acoustic meatus to the inner ear
c. Damage:
   i. Head tilt and circling toward the lesions.
   ii. Central Nystagmus

1. vertical
2. changes from one form to another

IX. Glossopharyngeal:

a. A mixed nerve
    i. Motor to the pharynx for swallowing
    ii. Sensory to the caudal tongue and pharyngeal region
    iii. Taste to caudal 1/3 of tongue
b. Exits the skull via the jugular foramen
c. Assess
    i. Gag reflex test
d. Damage:
    i. Pharyngeal paralysis

X. Vagus:

a. A mixed nerve
    i. Motor to the muscles of the pharynx
    ii. ANS to the pharynx, larynx, trachea, esophagus, and thoracic, and abdominal organs.
        1. Recurrent laryngeal is a vagal component.
    iii. Sensory to the baroreceptors and chemoreceptors in aortic body
b. Course
    i. Exit through the jugular foramen then out tympanooccipital fissure
    ii. 9 and 10 do not diverge till late so it is unlikely to damage one without the other.
c. Damage
    i. Megaesophagus
    ii. Left recurrent laryngeal – roarers
    iii. GI signs do NOT Pont to vagus except in ruminants.

XI. Accessory:

a. The last nerve in the chain in the medulla
b. Exits the skull via the foramen magnum
c. Motor to extrinsic muscles of the shoulder and intrinsic muscles of larynx
d. Damage:
    i. Cervical muscle atrophy and inability to move neck

XII. Hypoglossal

a. Muscles of the tongue – intrinsic and extrinsic
b. Exits the skull via the Hypoglossal canal
c. Assess
    i. Pull on tongue

ii. Tongue deviates toward lesion

Number	Name	Exit Strategy
I	Olfactory	Cribriform Plate
II	Optic	Optic Canal
III	Oculomotor	Orbital Fissure
IV	Trochlear	Orbital Fissure
V	Trigeminal-ophthalmic	Orbital Fissure
	Trigeminal-Maxillary	Round Foramen
	Trigeminal-Mandibular	Oval Foramen
VI	Abducens	Orbital Fissure
VII	Facial	Internal Acoustic Meatus to stylomastoid foramen
VIII	Vestibulocochlear	Internal Acoustic Meatus to inner ear
IX	Glossopharyngeal	Jugular Foramen
X	Vagus	Jugular Foramen
XI	Accessory	Foramen Magnum
XII	Hypoglossal	Hypoglossal Canal

Normal Pancreatic Anatomy

Submitted by: NC State Residents 2003

The pancreas of dogs and cats has a right and left lobe with a small central body, closely related to the duodenum. The slender right lobe runs within the mesoduodenum alongside the descending duodenum; the thicker and shorter left lobe extends over the caudal surface of stomach toward the spleen, within the greater omentum. The body lies in the bend of the cranial duodenum where it is crossed dorsally by the portal vein. In a beagle type dog, the pancreas is approx.1-3 cm wide x 1cm thick x 15 cm length.

The pancreatic duct (Wirsung's duct) drains the part of the pancreas that arises from the right, ventral primordium and opens adjacent to the bile duct into the duodenum via the major duodenal papilla. In the dog a lesser/accessory duct (Santorini's) emerges from the part formed by the left, dorsal primordium and opens in the opposite aspect of gut in the minor duodenal papilla 3-5 cm farther down the gut. Sometimes in the dog, one of the ducts regresses – confusingly, it is usually the pancreatic duct that is absent and not the accessory duct. The 2 ducts usually intercommunicate.

In the cat, the greater duct usually persists and fuses with the bile duct before opening with the major duodenal papilla. In 20% of cats, accessory is also present.

BLOOD SUPPLY

Celiac — cranial pancreaticoduodenal

Caudal vena cava — portal vein

Cranial mesenteric — caudal pancreaticoduodenal

NORMAL EXOCRINE PHYSIOLOGY

Exocrine functions are larger than endocrine (98% of pancreatic mass). Microscopic pancreatic lobules are composed of cells that produce digestive enzymes which are stored in zymogen granules. Pancreatic juice also has bicarbonate that helps neutralize gastric acid and other factors that help absorb cobalamin and zinc. The juice inhibits bacterial proliferation by helping normal degradation of exposed brush border enzymes and exerting a trophic effect on mucosa.

To prevent autodigestion: - proteolytic and phospholipolytic enzymes are synthesized, stored and secreted in the form of catalytically inactive zymogens

- from the moment of synthesis, enzymes are segregated in the rough e.r.

- acinar cells contain trypsin inhibitor that is synthesized, segregated, stored and secreted with the digestive enzymes = pancreatic secretory trypsin inhibitor (PSTI)

The zymogens are activated by enzymatic cleavage of the activation peptide. Enzymes from several sources, including some lysosomal proteases can be activators. Enteropeptidase enzyme (synthesized by duodenal mucosal enterocytes) is great at activating trypsinogens, which activates other zymogens.

The pancreatic basal rate of secretion is about 2% (bicarb) and 10% (enzymes) of maximal secretory rate in response to a meal. Initially after a meal, enzyme rich fluid secretion peaks at 1-2 hours, then a bicarb rich voluminous secretory phase peaks at 8-11 hours.

Secretion is a response to cephalic stimulation (anticipation, smell, presence of food in stomach/SI). SI releases secretin and cholecystokinin into blood when acid and food are emptied from stomach into duodenum. These stimulate bicarb and enzyme rich pancreatic juice secretion.

PATHOPHYSIOLOGY OF PANCREATITIS AND PANCREATIC TUMORS

Pancreatitis

Usually divided into acute vs. chronic. Acute pancreatitis is defined as inflammation of the pancreas with a sudden onset (duh). Recurrent acute disease is defined as repeated bouts of inflammation with little or no permanent pathologic change. Chronic pancreatitis is defined as a continuing inflammatory disease characterized by irreversible morphologic change, possibly leading to permanent impairment of function. Post acute episodes may completely resolve or may lead to a smoldering inflammation.

Cats traditionally get chronic mild interstitial pancreatitis and this is often accompanied by cholangiohepatitis.

General belief holds that pancreatitis develops when there is activation of digestive enzymes within the gland, resulting in autodigestion. This likely starts with zymogen activation within acinar cells via abnormal fusion of lysosomes and zymogen granules (basically a failure of the secretory process or of normal subcellular mechanisms for effective segregation of zymogens and lysosomes).

Progression of disease is helped along by free radicals, which peroxidize and damage lipids in cell membranes. (usually in a normal patient before this happens, free radicals are detoxified before harm can be done) The injuries lead to increased capillary permeability due to endothelial cell membrane damage and results in pancreatic edema.

Once the lysosomal enzymes activate trypsinogen (trypsin inhibitor is ineffective at acid pH in lysosomes), there is further activation of all zymogens, and things go to hell in a handbasket – edema progresses to hemorrhagic or necrotic pancreatitis with multisystem involvement and consumption of plasma protease inhibitors. This sucks, because the inhibitors (esp. alpha macroglobulins) are what protect us against proteolytic enzymes in the vascular space.

Causes are generally unknown, but low-protein/high-fat diets, refeeding after prolonged fast, idiopathic hepatic lipidosis in cats and hyperlipoproteinemia may cause or be associated with pancreatitis. Hyperlipidemia maybe causes pancreatitis with toxic FA generated by the action of lipase on abnormally high concentrations of triglycerides in pancreatic capillaries. Suspect drugs include thiazide diuretics, Lasix, azathioprine, L-asparaginase, sulfonamides, and tetracycline. Possible drugs include steroids, and H2 receptor antagonists. Duct obstruction (via inflammation, edema, fibrosis, biliary calculi, parasites, neoplasia, trauma, surgery, etc.) is another possible cause especially in cats. And let's not forget duodenal reflux, trauma, ischemia and viral/mycoplasmal/parasitic (oh that pesky *Eurytrema procyonis* in cats) infections.

Neoplasia

Pancreatic adenocarcinomas may be acinar or duct cell in origin, but are uncommon and very rare in cats. Which is good, since they're usually really malignant and frequently metastasized to duodenal wall, liver and lymph nodes. Patients can have signs of pancreatitis, DM or EPI. Prognosis is crappy.

Insulin secreting neoplasias are functional Beta cell tumors that are independent of normal feedback control via hypoglycemia. Pancreatic gastrinomas can be part of the Zollinger-Ellison syndrome (= increased gastric hypersecretion, multiple GI ulcers and a non-beta cell tumor of the pancreas). Normally adult pancreas doesn't secrete gastrin, but this appears to be a reversion back to fetal D-cell function. Gastrinomas can also secrete insulin, ACTH and insulin.

Pancreatic gastrinomas (most commonly) and insulinomas (2nd most common) can be part of the Multiple Endocrine Neoplasia syndrome; this consists in one form of parathyroid, pancreatic islet and pituitary hyperplasia or neoplasia.

Glucagonomas associated with superficial necrolytic dermatopathy has been described in 2 dogs.

Pancreatic polypeptide tumors have not been documented but PP has been found in high concentrations in many types of pancreatic endocrine tumors.

NORMAL ENDOCRINE PHYSIOLOGY

Islets of Langerhans are “small islands” have four distinct cell types: alpha, beta, delta and F cells.

Alpha cells → glucagon → liver glycogenolysis, gluconeogenesis, Ketogenesis, ureagenesis
Adipose tissue increase output of FA and glycerol
Direct anti-insulin actions

Beta cells → insulin → Increases glucose (liver and peripheral tissues) and amino acid transport into cells.
Promotes increased deposition of glycogen, protein and fat
Glycogen synthesis and lipogenic
Increases potassium uptake into tissues

Delta cells→somatostatin → = hypothalamic somatostatin, inhibits pituitary GH secretion, thyrotropin and gastrin secretion in GI
Inhibits insulin and glucagon secretion

F cells → pancreatic → Regulates exocrine pancreatic function: inhibits pancreatic Polypeptide enzymes and fluid secretion.
By protein meals and cholinergic reflexes

Beta cells monitor and control BG concentration. Entrance of glucose into beta cells is independent of insulin. If BG>110mg/dl, insulin is secreted. When BG<60mg/dl, insulin secretion is depressed.

See table

PATHOPHYSIOLOGY OF ENDOCRINOPATHIES ASSOC. WITH THE PANCREAS

Diabetes Mellitus

DM results from relative or absolute deficiency of insulin secretion by beta cells. Insulin deficiency causes decreased tissue utilization of glucose, aa and FA. Glucose from diet or hepatic gluconeogenesis accumulates in circulation. As hyperglycemia builds, renal tubular ability to resorb glucose from ultrafiltrate is exceeded, causing glycosuria. (180-220 mg/dl in the dog, 200-320 mg/dl in the cat). Glycosuria creates an osmotic diuresis, causing PU. Compensatory PD follows. Diminished peripheral tissue glucose utilization results in weight loss as body tries to compensate for perceived starvation state.

The "satiety center" in the ventromedial region of the hypothalamus interacts with the "feeding center" of the lateral region of the hypothalamus to control food ingestion. The feeding center can be transiently inhibited by the satiety center of eating. Glucose entering cells of the satiety center lessens the feelings of hunger. Glucose entrance is mediated by insulin so in diabetics w/o insulin there is no inhibition of the feeding center; thus, polyphagia.

In the dog and cat, the most common disorder of the endocrine pancreas is diabetes mellitus. It is classified as Type 1 or 2, based on pathophysiologic mechanisms. This is completely different from IDDM or NIDDM, which is based on a clinical diagnosis; this classification is used more often in veterinary medicine.

Type 1: characterized by destruction or loss of beta cells with progressive and eventually complete insulin insufficiency. This can be a sudden or gradual process (they can be initially non-insulin dependent) but eventually they become IDDM.

Type 2: characterized by insulin resistance and "dysfunctional" beta cells. Total insulin secretion can be increased, decreased or normal compared to a normal fasting animal. Usually the insulin present is enough to prevent ketoacidosis.

Some diabetics, especially cats, can flip back and forth between IDDM and NIDDM. Changes in diabetic state vary because:

- islet pathology may be mild to severe and progressive or static
- the ability of the pancreas to secrete insulin depends on severity of islet pathology and can decrease with time
- the responsiveness of tissue to insulin varies, often with the presence of other disease.

Secondary diabetes mellitus is carbohydrate intolerance secondary to concurrent insulin antagonistic disease or medicine (remember the bitch in diestrus, or cat treated with megestrol acetate). If the insulin antagonist sticks around, beta cell function can become impaired and permanent DM can develop.

IDDM

Causes in the dog include genetic predisposition. This can be a congenital absolute deficiency of beta cells to a less severe change in the beta cells which can predispose the latter animal to DM after exposure to other factors. Immune mediated causes can occur; islet cell autoantibodies and anti-beta-cell antibodies have been found in dogs. Pancreatitis can cause destruction of islet and lead to DM.

The cause of beta-cell degeneration in cats is not known, but islet-cell amyloidosis and beta-cell vacuolation are common histologic abnormalities in cats with IDDM. Chronic pancreatitis occurs in 51% of DM cats (1 study). Other cats have a reduction in number of islets or beta cells so immune destruction is another possibility.

NIDDM

More common in cat, and is probably multi-factorial. Obesity induced carbohydrate intolerance and islet specific amyloidosis may be causative factors. Obesity causes reversible insulin resistance from downregulation of insulin receptors, impaired receptor binding affinity and postreceptor defects in insulin action and abnormal insulin secretory response. These things can be found in the dog, too but is much less common.

DKA

FFA release from fat stores occurs with low circulating insulin and increased glucagon concentrations. Catecholamines – epi and norepi – also release FFA by stimulating lipolysis. Cortisol, GH, dopamine and thyroxine can all act the same way.
They are used extrahepatically as oxidative fuels and intrahepatically incorporated into triglycerides, metabolized to CO2 and H20 or converted to ketone bodies. Ketone production is due to FFA availability and ketogenic capacity of liver. When the liver make ketones, an equivalent number of hydrogen ions is made; these titrate with bicarb and can lead to acidosis. Ketones are substrates for energy metabolism in most tissues but excessive production results in accumulation.

Ketoacids are metabolized/excreted via 4 main routes. They can be oxidized to CO2 and H20 (muscle, brain, kidney). Some is decarboxylated and eliminated in the lungs. Acetone can be converted to glucose via a circuitous route. Last but not least, they can be excreted in urine.

4 conditions are assumed to contribute to development of ketogenesis and gluconeogenesis: relative or absolute insulin deficiency, diabetogenic hormone excess, fasting and dehydration.

When the body perceives low insulin levels and thus a “starvation” state, initial conversion of FFA to ketone bodies is good at first since they can be used as energy source. As they accumulate though, the buffer system gets overwhelmed. This causes an acidotic state which has bad effects on the cardiovascular system. When they spill into urine, they create an osmotic diuresis. This is compounded by diuresis secondary to glycosuria and create a depletion of body water, sodium, potassium, phosphorus and chloride. Vomiting and diarrhea that accompany DKA also contribute to the losses. Meanwhile, the liver is making glucose like crazy. So, we get hyperosmolality of extracellular fluid, volume contraction and underperfusion of tissues. The hyperosmolality leads to osmotic shift of water out of cells and thus cellular dehydration. This can lead to cerebral edema if the BG decreases too rapidly with therapy.

Epi, norepi, glucagon, cortisol and GH are very increased in humans with DKA, as are plasma AA and FFA. The stress hormones and elevated plasma FFAs themselves contribute to insulin resistance and stimulate hepatic gluconeogenesis as well as lipolysis. Insulin deficiency and increased counterregulatory hormones stimulates protein catabolism. The increased AA then also contribute to insulin resistance and hepatic GNG.

Usually the DKA patient has some other coexisting disorder. For instance, concurrent infection elevates secretion of cortisol and glucagon. Diabetic animals that aren’t eating and get their insulin withheld augment their gluconeogensis and ketogenesis. (a 50-75% insulin dose is most likely more appropriate).

<u>SOURCES</u>

I used Miller’s anatomy, the Dyce, Sack and Wensing anatomy book, physiology notes from class, 4th edition Ettinger and Felman and Nelson’s endocrinology book.

Microstructural anatomy of the Lung

Submitted by: Matt Wright

General:

- Airways are pliable tubes lined by respiratory mucosa with variable amounts of muscle and cartilage
- Respiratory epithelium
    - Larynx/trachea: tall pseudostratified columnar ciliated
    - Small airways: simple cuboidal nonciliated
- Goblet cells: mucous secreting cells that decrease in number as you go deeper in the airways
- K-cells: neuroendocrine function cells
- MALT: lymphatic tissue that produces IGA
- Smooth muscle: more prominent as airway size decreases. ANS, circulating catacholamines, and local factors can affect this smooth muscle

Comparitive anatomy of the distal airway:

- Terminal bronchioles mark the end of the conducting portion of the bronchial tree. Thus, the part of the bronchial tree distal is concerned in part or in whole with gas exchange and is subject to species modification
- The general pattern which is found in humans
    - Each terminal bronchiole divides into two respiratory bronchioles
        - A respiratory bronchiole is characterized by the presence of simple sac like alveoli opening off its walls
        - Respiratory bronchioles may undergo three orders of divisions so there may be three orders of respiratory bronchioles.
            - Generally, each third order respiratory bronchiole gives off a number of alveolar ducts from which the alveoli arise
            - However, alveoli can arise from any level of the respiratory bronchiole

Species Specifics;

- Dog: several generations of respiratory bronchioles are present. These respiratory bronchioles show considerable alveolar budding and end in alveolar ducts or alveolar sacs
- Horse: respiratory bronchioles are absent and alveolar ducts arise from divisions of the terminal bronchioles
- Ox: same as horse
- Cat: there is one and maybe two generations of respiratory bronchioles.

Bronchiole arterial supply:

- Branches off of the broncho-esophageal artery (a direct branch off the aorta) closely follow the bronchial tree and supply the entire wall of the bronchi and bronchioles. However, the peripheral distribution of the bronchial branches differs among species
    - Cow: bronchial branches terminate in the distal portions of the terminal bronchioles in a common capillary bed with the pulmonary arteries
    - Horse: Bronchial branches also follow the pulmonary arteries and supply blood to the alveolar capillary bed
    - Dog and Cat: bronchial branches terminate in a capillary bed in common with the pulmonary arteries at the level of the respiratory bronchioles.

Comparative thickness of the pleura

- Cow: thick
- Dog: thin
- Horse: probably thin

Anatomy of the alveolus

- Alveolar pores: openings in the alveolar septa to permit equalization of pressure and allow collateral circulation in case of bronchial obstruction
    - Cows do not have these pores
    - Dogs and horses have them
- Epithelium is composed of two cell types:
    - Type 1 pneumocytes: flat cells that cover most of the surface that serves as the gas diffusion barrier
    - Type 2 pneumocytes: small round cells that cover less than 5% of the alveolar surface
        - Secrete surfactant
        - Can differentiate into type 1 pneumocytes is there is epithelial damage
- Blood vessels form a plexus around each alveoli and the basement membrane supporting the capillary endothelium is directly applied to the basement membrane of the surface epithelium. In these areas the basement membranes fuse and supporting tissues are absent.
- Alveolar macrophages are found in the alveolar wall or free in the alveolar space.

Caution: This was a pretty general objective. I do not know what they were looking for but this is my best guess ☺ **MW**

Anatomy of the Kidney, ureter, and lower urinary tract including small ruminants and llamas

Submitted by: Matt Wright

Kidneys:

- Dog and general
    - In the cat there are normal subcapsular veins
    - Both of the cat's kidneys are located caudal to the dogs kidneys.
    - Fibrous capsule
    - Cortex: renal corpuscles and convoluted tubules
    - Medulla: contains the collecting ducts and loops of Henle.
        - **Gross arrangement varies with the species, however, the pattern is a medullary pyramid capped with cortex. The apex of the pyramid is the renal papilla. The papilla is the part of the lobule that drips urine into the proximal end of the ureter.**
            - Unipyramidal (DOG, HORSE, SHEEP)
                - A single medullary mass confines the cortex to the periphery where it forms a continuous shell.
                - The origins of the kidney are demonstrated by the scalloping of the corticomedullary junction and the arteries the mark the Interlobar boundaries.
                - The fusion joins the papillae in a **common renal crest.** This is the ridge resulting from the fusion of the pyramids. (Found in dogs, horses and small ruminants.)
                - The **renal pelvis** is the expanded proximal end of the ureter with a terminal end renal crest.
                - The **renal sinus** is the potential space occupied by the ureter, renal aa, vv, and lymphatics, and nerves entering the kidney.
                - The **renal hilus** is the opening into the renal sinus.
    - Ureter
        - In most species the ureter is a continuation of the renal pelvis into which the papillary ducts open.
        - IN the dog and cat, the renal pelvis is molded upon the renal crest and extends flanges (**renal diverticula**) dorsal and ventral to this
            - Each flange shows a number of local expansions or recesses that are divided from each other by projections of renal tissue.
        - On reaching the pelvic cavity, the ureter
            - Male: bends medially to enter the genital fold
            - Female: bends medially to enter the broad ligament.

        - In both, the ureter is carried over the dorsal portion of the bladder and enters obliquely
            - The length of intramural travel in the bladder wall prevents reflux because the ureter collapses when vesicular pressure rises. However, this closure can be overcome by peristalsis in the ureter.

    - Bladder:
        - The inner surface will become smooth with filling but there are two folds that resist effacement
            - **<u>Urethral Crest</u>**: continues into the pelvic urethral
                - A convergence of the slit like ureteral orifices.
        - The **<u>trigone</u>** is the internal area of the dorsal bladder between he two ureteral openings and the start of the urethra.
        - Ligaments of the urinary bladder
            - Medial (ventral ligament): the remnant of the urachus runs in the medial ligament so it extends from the bladder to the umbilicus
            - Two lateral ligaments
                - The round ligament of the bladder which is the remnant of the fetal umbilical arteries travels in the edge of the lateral ligaments.

- Cow:
    - Multipyramidal or multilobar: medulla is arranged in many discrete masses in which a portion of the cortex caps the base of the pyramid
        - The cortex is actually continuous.
        - **<u>The apex of the pyramid points toward the renal sinus and forms a *papilla* that fits into a cuplike expansion of the renal pelvis (*calyx)*formed by a terminal branch of the ureter.</u>**
            - Thus, individual papillae are present due to incomplete fusion of the medullary pyramids.
            - Thus, a calyx is the cup shaped thingy that receives urine from the individual renal papillae.
        - **These terminal branches eventually unite to form two major channels that converge from the cranial and caudal pole to form a single ureter.**
        - <u>There is no large central expansion consistent with a renal pelvis.</u>
        - Each medullary pyramid with it's associated cortex constitutes a renal lobe
        - In the cow, the boundaries of the lobes are revealed by fissures in the surface

- Renal Pelvis and ureter:
  - The ureter is formed by the coming together of the short passages that lead from the calices that enclose the individual renal papillae
  - *There is therefore, no renal pelvis in the cow*.

- Horse:
  - Modified Unipyramidal type
    - The numerous constituent pyramids are fused
    - The former boundaries are revealed only by the arrangement of the Interlobar arteries.
    - Some external lobation is present in the foal.
    - The apices of the fused medullary pyramids forms a common renal crest that projects into the pelvis.
    - There is a curious central expansion at the origin of the ureter and two terminal recesses toward the poles.
      - **The terminal recesses are long tube like extensions that collect and carry urine from the kidney poles to the small renal pelvis. They can be regarded as large collecing ducts or diverticulae of the renal pelvis.**
    - The pelvic mucosa produces a mucous secretion so that the urine normally contrains protein.
  - The left kidney is bean shaped and the right kidney is heart shaped.

- Small Ruminant:
  - Bean shaped and smooth (not lobated)
  - Kidneys of the dog, goat and sheep are hard to distinguish grossly.

Embryology of the urinary tract:
Submitted by: Matt Wright

During development, there are three distinct excretory organs

- Step 1: The Pronephros
    - Non functional
    - Located along the thoracic segments
    - Composed of numerous pronephros or pronephric tubules
    - These tubules turn caudally and combine with their neighbor to form the pronephric duct which empties at the cloaca

- Step 2: The mesonephros
    - The pronephric tubules regress but the pronephric duct remains
    - Mesonephric tubules arise and dump into the pronephric duct which is now called the mesonephric duct.
    - The mesonephros is functional throughout the embryonic life. They are like little individual nephrons
    - When the time comes, the mesonephros regresses in a cranial to caudal direction but parts of it are retained to be used in the male genital system.
        - **The mesoneprhic duct becomes the Ductus deferens**
- Step 3: the metanephros
    - The ureteric bud grows out of the caudal end near the cloaca.
        - The proximal end undergoes successive divisions to become the kidney.
        - There is dichotomous divisions with resorption of the first few orders into the terminal expansion in a variable fashion that account for the specific forms of the renal pelvis and calices.
    - **Again, the remnant of the metanephric duct is the ureter**

Formation of the lower urinary passages:

- Step 1: horizontal division of the cloacal region of the hindgut but the caudal outgrowth of a wedge of mesoderm called the **urorectal septum**
    - The urorectal septum eventually reaches the cloacal membrane and divides it into the dorsal anal and ventral urogenital parts.
    - The fusion site corresponds to the perineal body
- Step 2: Rupture of the anal membrane. When the anal membrane breaks down the dorsal passage is the rectoanal canal
- Step 3: rupture of the urogeniatal membrane: this provides a ventral passage with a caudal opening
    - The cranial part differentiates into the future bladder and the allantois
        - The bladder is a widening that is continued cranially by the **urachus** that exits the body through the umbilical opening to collect urine in the allantois.

        - At birth, this duct shrivels and forms a scar on the apex of the bladder.
    - The caudal part forms the urethra
        - Forms the entire female urethra
        - Forms only the pelvic urethra in the male, the rest is formed with the genital system.

Formation of the Reproductive Organs

- Initially the embryo is of indeterminate sex:
    - Gonadal primordium is formed on the medial aspect of the mesonephros
        - This invagination is invaded by cords of epithelial cells and primordial germ cells that migrate from the yolk sac.
- Formation of the male system
    - The male business gets surrounded by a tunica albuginea and there is male type differentiation of the cords within the primordial gonad
    - Later, the mesonephric duct canalized this whole business and becomes the outlet for the gametes (the ductus deferens)
    - The mesonephric duct opens on the other end to the orogenital sinus
    - After division of the cloaca, the caudal parts become the pelvic urethra.
        - Outgrowths of the urogenital sinus become the prostate and the BUGS.
    - Formation of the penis and the rest of the Urethral
        - Thickenings appear at the margin of the urogenital membrane in the indifferent stage
            - Phallic tuburcle and cloacal fold and a scrotal swelling arise laterally and medially and fuse.

- Formation of the female system
    - The gonad begins to form similar as the male, however, they develop into ovaries. Duh. Also, there is no connection that forms with the mesonephric tubules so there is no uninterrupted outlet for the gametes.
    - Ovarian descent is limited but greatest in the ruminants
    - The duct system is provides by the *paramesonephric ducts* which have only vestigial importance in the male.
        - These ducts develop as evaginations lateral to the mesonephric duct.
        - Initially there is evagination then there is active growth of the ducts.
        - The cranial end of the duct remains open to the periotoneal cavity while the caudal end grows together to end on an outgrowth of the dorsal wall of the urogenital sinus.

        - The outgrowth forms the vagina once this solid tissue forms a hole in itself.
        - The urogenital sinus becomes the vestibule.
        - Where the ducts and the outgrowth meet forms the cervix.
            - A hymen may persist at the fusion with the paramesonephric ducts.
    - Remember, the mesonphric duct regresses in the female
        - Remnants survive as the ducts of gartner and the epoophoron which may cause anomalies
    - Just like the male, the external features also form from the phallic tuburcle genital folds, and urethral folds.

- The process of testicular descent
    - The gubernaculums forms and then regresses by doing so, the testis is pushed and pulled into the abdomen through the inguinal canal.

References: The most straightforward and least painful review of this embryology was in Dyce, Sack, and Wensing's Textbook of Veterinary Anatomy

Avian Respiratory considerations and physiology

Submitted by: Matt Wright

- Lungs:
    - Paired lungs lie dorsally
    - They are attached to the thoracic ribs and fill the intercostals spaces.
    - Changes in size and position of the lungs are limited but they are not fixed and nonexpendable.
    - The course of air in the lungs is as follows
        - Syrinx (distal trachea)
        - Primary bronchi
            - Secondary Bronchi: in the ventral surface of the lung, secondary bronchi connect directly to the caudal thoracic and abdominal air sacs through ostium that can be seen during endoscopy.
                - Tertiary Bronchi
                    - Parabronchi with shallow depressions (artia) along their walls. Each atria has 3-6 funnel shape ducts (infundibula) that lead to the air capillaries.
                    - The air capillaries form an ansatamosing 3D network which is intermittently woven with the blood capillary network for oxygen exchange.

- Air Sacs:
    - **Pulmonary Air Sacs:** Most birds have four paired and one unpaired pulmonary air sacs (the exact configuration varies with species) that connect to the lung to create a large respiratory capacity.
        - Cervical
        - Cranial thoracic
        - Caudal Thoracic
        - Abdominal
        - Clavicular: unpaired located dorsal and caudal to the crop in the thoracic inlet and has intra and extrathoracic components.
    - **Cervicocephalic Air Sacs:** these air sacs are not connected to the lungs but connect to the caudal aspects of the infraorbital sinus. They function as an insulating layer for heat retention or something else but not for respiration.

- **Pneumatized bones:** humerus, clavicle, coracoids, vertebrae, ribs, and femur are connected to the respiratory tree through extrathoracic Diverticula.

Respiratory Physiology

- There is no diaphragm rather there is a thin membrane (**oblique septum)** that separates the thoracic and abdominal cavity.
- Inspiration (rib and sternum movement) create negative pressure in the ceolem
    - Therefore, if a bird cannot move its ribs, it suffocates (handling, bandage)
- The cycle
    - Inspired air flows in through primary bronchi
        - _ goes to the lung
        - _ bypasses the lung to go to the caudal air sac (bypassing the gas exchange portion
        - The air already in the lung goes into the cranial air sac.
    - On expiration,
        - Air that is in the caudal air sac enters the lung
        - Air that is in the lung and air in the cranial air sac exits through the trachea.
- Thus, two respiratory cycles are necessary for the _ volume of air that enters the air sacs to move totally through the respiratory tract. This is entirely efficient because there is always fresh air traveling into the lung either inspired or to the lung from the caudal air sac on expiration.

Gas Exchange:

- In parts of the lung: Gas exchange occurs in the air capillaries which are air tubes that branch and anastamsose which each other and are richly intertwined wit blood vessels that create a blood gas barrier.
- In other parts of the lung there is a counter current exchange mechanism which is more efficient that the former and compared to mammals.

The effect of patient positioning and anesthesia on the appearance of the thoracic radiographs

Submitted by: Matt Wright

Technique: high Kv low mAs to maximize contrast (long scale) and decrease motion

Inspiration Vs. Expiration:

- In panting dogs, hold the mouth shut then allow to open and radiograph on deepest inspiration
- Inspiration Lateral View:
    - Diaphragm caudal and more flat
    - Increased distance between heart and diaphragm
    - Lungs are larger, more inflated, and lucent
    - Slight elevation of heart from sternum – more vertical
    - More elongate, parallel, distinct, thinner CVC
- Inspiration VD View:
    - Smaller silhouette
    - Increased thoracic width and length
    - Decreased diaphragmatic contact
    - More distinct less blunted apex
    - Costodiaphragmentic angle caudal to T10

Right vs. Left Lateral: in both the dependant bronchus is dorsal

- Right lateral:
    - Heart is more egg shape
    - Crura parallel
    - Overlap between the cranial lobe vessels.
    - The right lateral may better depict the anatomy of the heart because in left lateral, the heart falls away from the sternum but in right lateral it stays pretty much on midline
- Left Lateral
    - Heart is more round
    - Crus diverge
    - Apex of the heart is displaced slightly dorsal making it appear more circular
    - Easier to distinguish cranial lobar vessels as they are parallel.

Vd vs. DV

- VD:
    - Silhouette is longer and narrower
    - Accessory lung lobe is larger
    - CVC is longer
        - It is easier to evaluate these caudal structures

    - Changes in the descending aorta and great vessels are more conspicuous on the VD.
- DV
    - Caudal lobar vessels are magnified, not silhouetting with the collapsed caudal lung, and they are more perpendicular to the primary beam making them easier to evaluate.

Some additional things from articles written on the subject…

- In cats the heart is more variable in dorsal than sternal.
- Root feels that left lateral has less distortion of the heart.
- DV has been recommended for the heart and the VD for the lungs

- **DV vs VD in cats. VRUS 1982 Carlisle and Thrall:**
    - Only marginal difference in the appearance in the heart in cats.
    - The biggest change is that in the VD the right cranial heart is more curved, and the caudal mediastinum and the accessory lung lobe region was more easily seen. Cardiac shape varied
    - The heart was more consistent in the DV
    - Bottom line is that both are satisfactory. All changes are mild and do not simulate disease.
    - Subjective and objective measurement showed that the size of the heart was independent of position. This is in opposition to a previous study where the heart was longer in the VD and wider in the DV
        - This may be because magnification is less in the cat.
    - There was little difference in the position of the base of the heart relative to the dorsal limit of the thorax (in dogs it moves) as evidenced by horizontal beam.
    - The magnitude of change in the cat heart from DV to VD is not as great as it is in the dog.

- **The effect of dorsal vs. ventral recumbency on the radiographic appearance of the canine thorax: VRUS 1981 Thrall**
    - VD: craniocaudal axis was longer. Heart has a more consistent positional relationship to the thoracic spine, larger area of the accessory lobe is visible, greater length of the CVC is visible.
        - Increased length is from magnification from increased distance
    - DV: caudal lobar vessels are more easily seen
    - The width of the heart does not change much
        - This is because the base of the heart does not move as does the apex. The width measurement is a measurement of the heart base.
    - On horizontal beam films they saw that the heart base moves toward the spine in VD. This may be hazardous with LA enlargement, hypertension, or malacia of the trachea.

- Conclusion: The choice should be made based on the clinical status of the patient and the reason radiographs were taken.

- **The effects of rotation on the radiographic appearance of the canine heart in dorsal recumbency**
  - If the dorsal spinous processes are outside the vertebra it is tilted too much

- **Influence of cardiac cycle on the radiographic appearance of the feline heart. Toal: VRUS 1985**
  - A more constant appearing size between systole and diastole in the VD than the DV
  - VD hearts were wider and of larger area.
  - Therefore, cardiac size changes during the cardiac cycle are dependant on body position.
  - In lateral recumbency, the heart was wider in diastole but longer in systole. This was difficult to explain.

- **The canine lateral radiograph: Spencer VRUS 1981**
  - The heart was longer from apex to base in the right lateral recumbency.
  - Right lateral may be most accurate.
  - In left lateral recumbency, the apex may shift away from midline slightly giving a smaller profile. This also accounts for the fact that the apex is closer to the sternum in right lateral recumbency.