Large Animal Medicine
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Introduction, (work in progress - shadowed)

1. Common ways to categorize Veterinary Medicine and Veterinary Practice
   By species
   Large animal, small animal, exotic, equine, bovine, feline, etc.

   By animal use categories
   • **Companion animal medicine**
     Small animals primarily
     Horses, cattle, sheep, goats, deer, llamas, etc. can all be companion animals in some situations

   • **Industrial animal medicine**
     Primary goal is to assist the clients in producing a profit or limiting losses. This may limit the treatments and procedures that may be used to only those that are economically reasonable compared to the value of the animal. Many advanced and expensive procedures may be utilized in valuable animals if there is a reasonable opportunity for success and an expectation for a return on the investment made in veterinary therapy or surgery.
     Food animals
     • Dairy cattle, Beef cattle, Swine,
     • Small Ruminants,
     • Captured or farmed wildlife such as buffalo, deer, elk, and other
     • Camelids,
     • Fowl,
     • Fish species
     Generally industrial medicine is a characteristic of food animal agricultural practice, but not always. It can include breeders of companion animals and animal athletes.
     • Running horse breeders
     • Pleasure horse breeders
     • Dog and cat breeders
     • Greyhound running dog breeders
     • Camelids

   • **Zoo animal medicine**
     Zoological medicine is a type of veterinary practice that includes exotic, rare and frequently valuable endangered wildlife animal species. There are fewer economic limitations on the veterinary and husbandry procedures used for these species, compared to agricultural species. A sound knowledge of agricultural practice and experience working with large farm animals can be a good background in preparation for working with large exotic species.

   • **Laboratory animals**
     • Can include any species from classical laboratory animal such as mice and primates to large species such as cattle, horses and others.
     • Pharmaceutical companies, universities and government entities all utilize animals in research

   • **Wildlife medicine**
     This area of veterinary medicine is closely allied to wildlife biology and wildlife management. Wildlife species can act as reservoirs for diseases that are important in domestic livestock species. Wildlife can also harbor zoonotic infectious and parasitic diseases.
     Following are some examples of trans-species diseases:
Brucella abortus

Tuberculosis, Mycobacterium bovis has been frequently identified in wild ruminants.

Equine protozoal myeloencephalitis,

West Nile virus and other encephalitides from avian species via mosquitoes,

Rabies, from many species to livestock and people,

Plague, Yersinia pestis, from wild rodents via fleas,

Hantavirus infection transmitted from rodents,

Echinococcosis sp. - tapeworms, from domestic and wild carnivores,

Trichinella - a zoonotic nematode parasites, from bear and swine,

& many others.

Although not currently considered to be a zoonotic diseases; chronic wasting disease in wildlife and bovine spongiform encephalitis in cattle are considered to be potentially dangerous to human due to their similarity to the human spongiform encephalopathy known as "variant-Creutzfeldt-Jakob Disease" which is abbreviated as: v-CJD.

Usually this wildlife veterinary medicine utilizes epidemiological method to evaluate and limit the spread of wildlife diseases to domestic animals and humans. Wildlife veterinary medicine is almost exclusively population level, preventative medicine, with an emphasis on research and public health. Much research is being devoted to applying vaccination programs, anti-parasite procedures and population control procedures that do not require gathering and treating animals individually.

One other specialized area in wildlife medicine is wildlife rehabilitation. In some situations this could include the rehabilitation of injured large animal species such as deer, elk, and large aquatic mammals.

**Functional Divisions of veterinary medicine**

- **Classical Veterinary Medicine**
  - Diagnostics and therapy,
  - Internal medicine,
  - Surgery,
  - Obstetrics, etc

Classical veterinary medicine is what the majority of veterinary education is all about. A good foundation in basic sciences and veterinary medicine is obviously essential in order to effectively perform techniques required for diagnosis, treatment, surgery and rehabilitation of animals in a veterinary practice. Also obvious, is the importance of that same knowledge when implementing effective preventative medicine and production medicine practices. Understanding physiology, anatomy, nutrition and veterinary medicine at the highest level possible is equally valuable for people involved with the husbandry (production, care and breeding) of livestock in unusual or specialized environments; such as horses in competition, cattle in dairy production, hospitalized animals, animals rehabilitating from disease and injury, and even beef cattle on pastures or in feedlots.

- **Preventative Veterinary Medicine**
  - Vaccination, parasite control, etc.

Vaccination programs, (discussed later) and parasite control programs (also discussed later) are the two usual candidates that people dwell on when they discuss preventative medicine. Preventative medicine really goes beyond that.

- Pre-purchase exam

Pre-purchase examinations, are performed on behalf of the purchaser to verify the suitability and soundness of a prospective animal for its intended purpose. If a prospect is identified as sick, injured or unable to fulfill the buyer's expectations, this exam is certainly a preventative medicine function.

- Pre-sale exam

Pre-sale examinations, these examinations are performed on behalf of the seller. Many times a seller will have an animal, that is to be sold, examined in order to verify their soundness for their intended purpose. An excellent example is a seller having the veterinarian perform breeding soundness exams on a group of bulls that he hopes to sell for use as herd sires. If a bull were not reasonably likely to be successful at breeding cows, it would be foolish for a breeder to offer him for sale, as it would damage his reputation.
and affect future sales. These exams should be scheduled prior to listing or showing the animals for sale, so that a buyer is not falsely led on by a beautiful, but non-functional animal.

- Quarantine and isolation procedures are used in many disease prevention protocols. Quarantine, is separation of an animal for the purpose of observing it for signs of infectious disease. A quarantined animal may have been exposed to a contagious disease, or it may be an apparently healthy new addition to a herd, where the health status in the herd of origin is unknown or suspect. In order to prevent the introduction of an infectious disease, new additions to a herd should be observed for infectious diseases, for a reasonable period of time, prior to incorporation into the herd. Quarantine is derived from the word for forty in Latin languages. A forty-day period of time is reasonable; thirty to forty days is typical. During quarantine a new addition can also be tested for diseases that are of concern. The time can be used to administer initial vaccinations and booster doses of vaccine to meet minimum herd standards. Intestinal parasite control medication can be administered at the beginning of a quarantine period and a second larvacidal dose of anti-parasitic medication can be administered at the end of quarantine for a very effective reduction in parasite ova shedding and contamination of pastures. If an animal becomes ill during quarantine that animal is then held in isolation.

Isolation is separation of an animal that is actively infected with a confirmed, suspected, or unknown contagious disease. Isolation will prevent spreading the disease to other animals in the herd or hospital. Isolation should continue until the animal is unlikely to be contagious.

- Animal welfare, humane handling and humane management

Animal welfare, humane handling and humane management of livestock are also associated with preventative veterinary medicine. Good animal health, proper nutrition, normal inter-animal interaction, non-excitatory human - animal interactions, limitation of stressful situations and overall animal comfort are required for maximum production, performance, growth and reproduction.

- Good health care and high quality management procedures are usually profitable.
- Successful cattlemen and horsemen generally understand that healthy and comfortable animals produce and perform better.
- See: http://www.grandin.com/ for data and insight on humane management practices. Dr. Temple Grandin is a Colorado State University, Animal Science professor; who has dedicated her life to the study of animal behavior and humane management of livestock.
- Safe handling, keep fences, pens, stalls, chutes, stocks & other animal handling equipment and working facilities clear of protruding, sharp or otherwise dangerous objects or obstructions. Make sure all moving parts are lubricated and working easily. Repair broken fencing and containment structures immediately. Never leave a vertical fence post without a securely attached top rail, as it is possible for a jumping animal to impale itself on such an object. When farm animals are being handled, there are likely to be excitable individuals that may attempt to escape the containment facility, especially if there are weak (and dangerous) points that invite such attempts.
- Proper restraint, when performing a procedure on a large animal it can be dangerous to the animal, and attending humans, if the patient is not properly restrained. An adjustable horse stock with a way to crosstie or otherwise restrain the horse is beneficial with many procedures. A proper cattle chute and head catch serves the same purpose for cattle. Many homemade stocks, chutes or tie-ups are acceptable for both large and small ruminants and camelids.
- Safe housing

Ventilation is essential in enclosed barns, when livestock are confined. Ventilation can be of two basic types; active or passive.

1. Active ventilation uses fans to replace and remove stale air. Air is usually drawn in through side wall vents that are equipped with fans, and air is often exhausted through ceiling vents or vents that are placed higher on the side walls.
2. Passive convective ventilation uses the natural buoyancy and rising of warmed air from the area around the livestock for exhaust of stale air through vents in the roof, and replace fresh through air inlets low on
the sidewalls of the barn. Regardless of the type, one of the goals of ventilation is to limit air drafts and allow air to pass over a minimal number of animals prior to being exhausted, usually through the roof. This prevents noxious air accumulation and infectious disease transmission. ** closed barn design **

3. Poor ventilation can result in many problems. Pulmonary irritation, allergy and due to noxious gas and dust accumulation. Viral and bacterial respiratory disease can also become a problem. Heat stress and many other conditions.

4. There is no question about ventilating barns in warm or hot weather. Cows are intolerant of heat and susceptible to heat stress at temperatures as low as 72F. In warm weather; fans work well in addition to passive ventilation building design.

5. There is concern about ventilating barns during cold weather in the winter. Cows and horses are more cold tolerant than humans and require ventilation even in cold weather. In cold weather it is important to prevent drafts; passive ventilation works best in cold situations.

Ø Good floors design, i.e. good footing with minimally abrasive surfaces is important when hoofed animals are housed on concrete or other hard composite flooring. Grooves, ½” to 1” wide, cut in the floor at an interval of less than ½ the length of the animals stride (~<1’) is probably the best. Epoxy surface coating is also good, but can be very expensive. Recycled grounded rubber and urethane can be incorporated into concrete to produce excellent footing. A roughened concrete surface is cheap and prevents slipping, but is too abrasive and can do damage to the hoof wall and sole of livestock feet. Roughened concrete, is common, and may be acceptable in area where livestock are only occasionally worked, but it is the least desirable method of concrete treatment in areas were cattle spend significant time. Some cattlemen and horsemen use smooth concrete and rely on handling animals gently on these surfaces. There is a greater risk of injury on smooth or slick flooring, but animals, once used to it, are very easy to handle.

Ø Good drainage in buildings, dirt pens and paddocks is also important for a safe and healthy environment. Most modern livestock pens are constructed with high areas where the animals are fed and have access to water and a low swale or ditch for drainage of the pen to a sewage confinement lagoon.

Ø Regular manure management or removal is essential especially in low wet areas where animals have access.

- Proper feed storage usually means prevention of weather related feed spoilage. Feed and feeding facilities can also be contaminated by feces, urine and bedding debris of rodents, pets and wildlife species. Farm cats are capable of transmitting Toxoplasmosis. Dogs and perhaps wildlife species can transmit Neospora. Sarcocystis neurona; causing Equine Parasitic Myeloencephalitis (EPM) can be transmitted to horses and ruminant species by opossums. S. neurona, Sarcocystis spp, Toxoplasma spp, Neospora spp and other parasitic organisms can infect farm animals causing illness, abortion and potentially public health risk to the food supply. Dogs, cats and carnivorous wildlife must be prevented from entering and soiling feeding and feed storage areas whenever possible.

- Proper balanced nutrition. This is an area that would require a text book to cover in any way but superficially. Animal health is, in many ways, pre-determined by the nutritional status of the animal or the animal population.

- Use of proper administration techniques (i.e. when using injectable vaccines and drugs, intra-nasal vaccines, oral vaccines and medications, topical and pour-on drugs) for preventative and therapeutic procedures.

- Eliminate intractable, wild or disruptive animals. Wild animals cause stress and influence the behavior of all animals in a herd. Wild animals should be culled from
the herd if they adversely influence production from other animals or if they are a danger to workers or animals on the farm or ranch.

- **Regulatory Veterinary Medicine**
  
  In the US, this is a government function; performed by US department of agriculture (USDA) veterinarians, state departments of agriculture (i.e. Colo. Dept. of Ag.) veterinarians, and private practicing veterinarians that are accredited by the U.S. and state departments of agriculture.
  
  - Regulatory Veterinary Medicine is performed by government and accredited private veterinary practitioners; enforcing state and federal regulations regarding domestic animals. Most of these laws involve farm animals but pets are also regulated in many ways regarding interstate and international movement. Wildlife species can also be covered by federal and state laws regarding harvest and use.
  
  - One goal of regulatory medicine is to keep the US livestock herd healthy and free of domestic contagious diseases that affect profitability.
  
  - Examples of economically important domestic diseases that are reportable to the USDA and State Departments of Agriculture are:
    - **Cattle**
      - Tuberculosis
      - Brucella abortus
      - Trichomonas fetus
      - Vesicular Stomatitis
    
    - **Equine**
      - Equine Infectious Anemia
  
  - Another goal is to prevent the introduction of foreign animal diseases into the U.S. Foreign animal diseases (also referred to as exotic animal diseases), if introduced into the U.S. can have a dramatic negative effect on animal health. An adverse health issues, such as a reportable disease outbreak, will affect livestock sales, food exports, livestock exports and the profitability of farm animals and food products.
    
    For example; the occurrence of a single case of “Bovine spongiform encephalitis” (BSE) in the U.S. in December of 2003 caused a dramatic reduction of beef exports. U.S. beef exports in 2003 were 1.3 million metric tons to 65 countries. In 2004 beef exports dropped to only 0.32 million metric tons to only a fraction of the U.S. beef customers. That single animal with BSE was actually imported into the U.S. from Canada.
  
  - **Regulations:** Federal and state departments of agriculture officials draft regulations that protect the U.S. national livestock herd. Many of these officials are specialists in epidemiology or other infectious disease disciplines. All of the Federal regulations are published in the "Code of Federal Regulations Title 9 - Animals and Animal Products". State regulations are published in similar state laws and regulations publications.
  
  - **Accreditation:** A licensed veterinarian must become accredited by the USDA prior to performing regulatory functions on behalf of the federal or state government. Accreditation requires that a licensed veterinarian pass an accreditation exam covering domestic and foreign animal disease control issues and also must meet continuing education requirements.
  
  - A partial list of regulatory functions would include:
    - **Interstate health certificates** - The issuing veterinarian must determine that (1) all animals are adequately identified with a permanent ID (cattle and other food animals) or a picture or drawing showing all markings and brands (horses). Food animal identification tags must be approved by the USDA when used for interstate health certificates. (2) that the animals meet all pre-entry requirements for the state of destination, such as brucellosis vaccination, which is required by many states, (3) that the animal is determined to be healthy through a physical exam or visual observation, (4) that suitable results (usually
negative) for all required disease tests have been obtained and (5) any treatments or vaccinations required by the state of destination have been administered. Assuming that all entry requirements for the state of destination have been met, the issuing veterinarian can then call the state veterinarians office, in the state of destination, and obtain an entry permit, if one is required. Then a health certificate or “Certificate of Veterinary Inspection” (referred to as a CVI) is completed. The CVI lists the consignor’s and the consignee’s relevant physical addresses and other contact information. The species, breed and number of animals is noted. The date and method used to transport the animals is noted. The name and address of the person responsible for transportation (usually the person driving the truck) is noted. All animals to be shipped are usually individually listed on the health certificate. All tests performed, including the result, and abnormal health exam findings are listed for each animal. The health certificate should also include a listing of all treatments and vaccinations administered prior to the shipment. The accredited veterinarian who performed the health examinations is responsible for filling out the CVI on the livestock and must sign the health certificate. As soon as possible (certainly within 2 weeks) after the shipment two copies of the CVI must then be mailed to the state veterinarian in the state of origin of the shipment. The state veterinarian then will forward a copy of the certificate to the state veterinarian in the state of destination. Currently electronic CVIs, transmitted via the World Wide Web are being used by many states to streamline interstate movement of livestock. These eCVI documents resemble closely the paper ones and are transmitted as secured unalterable .pdf files.

**International health certificates** - An International Health Certificate is often referred to as an IHC. The requirements for an IHC is similar to, though frequently much more complex, to those required for a CVI issued for interstate purposes. The animals for export and animals producing animal products for export (such as semen, embryos, hides, meat, etc.); require proper identification, testing, quarantine and treatments according to protocols that are dictated by the importing country. It is not uncommon for an importing country to require quarantine or isolation of live animals prior to their importation and during the period of time that all export testing, treatments are performed. Also, animals that are producing animal products for exportation usually must be placed into isolation during the testing and production periods of those products. Once testing and other procedures such as quarantine or production of the products to be exported is complete; an IHC can be written, documenting all procedures and then the IHC must be approved and endorsed by a USDA veterinarian. At that time the animals or animal products must be exported, in continued isolation during transport, according to the importing countries requirements.

**Brucellosis vaccination** - of heifers only, should be performed prior to the heifer achieving 12 months of age. The heifers should be identified with a proper vaccination tattoo in the right ear’s concave area and usually a metal vaccination tag. The tattoo includes an “R” indicating that the RB51 Brucella abortus vaccine strain was used. The tattoo also includes a “US Vaccination Shield” and the last digit of the year of vaccination i.e. “3” for 2013 (and 2003). The tattoo is noted as “RV3” on the vaccination document. The vaccinated heifers are tagged with an orange metal “clip” tag with a number that is unique in the USA. In Colorado, that number begins with 84, then 3 letters and 4 digits. For example 84VAB1234, the 84 means that the tag is a Colorado origin tag. The V stands for “Vaccinate” and is present on all vaccination clip tags. A sequence of tags, ordered by a veterinarian, are registered to that accredited veterinarian so that an animal can be traced back to the veterinarian and from there back to the farm of origin. Keeping an accurate record of the application of these ear tags is important for the purpose of animal trace-back if it is ever
necessary. It is required that all Brucella abortus vaccinations be reported to the State Department of Agriculture.

The vaccination tattoo is applied to the concave skin surface of the right ear. An orange official identification clip tag is also applied to the right ear. There are also official electronic ID (EID) tags that can be used.

- **Brucellosis testing** of infection suspects and cattle to be sold or moved interstate or internationally may be required. All tested animals must have acceptable identification or else they must be ear tagged with a unique, shiny metal, “bright” clip tag. Like the vaccination tag, in Colorado, the test tag begins with 84, then 3 letters and 4 digits, for example 84ABC1234.
  - A blood test is used for routine testing of beef and dairy individual animals prior to sale, interstate movement or international movement. A blood test can also be used on all animals on a beef or dairy farm for certification of the farm as “Certified Brucellosis Free”. A blood sample is also used for diagnostic purposes, usually after a cow has aborted.
  - All tested individual animals must have acceptable official identification or else they will be ear tagged with a unique “bright” test tag with the 84ABC1234 pattern.
  - A Brucellosis “Milk Ring Test” on a milk sample from the milk bulk tank allows a dairy farm to test the entire herd for Brucellosis with a single test. This is for food safety purposes as well as for simple periodic survey testing of the dairy cattle for maintenance of the dairy farms certification as “Certified Brucellosis Free”.
  - The brucellosis eradication program also requires all cattle sent to slaughter at registered slaughter plant to be tested for Brucellosis.

- **Tuberculosis testing** is required for all dairy cattle periodically, usually every other year, to meet FDA and USDA health standard. TB suspects, cattle to be shipped interstate and cattle to be shipped internationally may also be required to be tested for TB.
  - The routine survey test used for tuberculosis is the “Caudal Fold Intradermal Skin Test”. Any suspicious reactions to this survey test...
will require that the animal be tested with the "Comparative Cervical Test" within 2 weeks of the caudal fold test.

- All tested animals must have acceptable official identification or else they will be ear tagged with a unique “bright” test tag with the 84ABC1234 pattern.

- The primary method for routine survey of US cattle for tuberculosis is the post mortem exam that all cattle receive at the time of slaughter in registered slaughter plants.

- **Trichomonas fetus testing**, required for all sale bulls, bulls moving interstate, and bulls used on public lands for breeding purposes. Virgin bulls under 18 months of age may be exempt. "Trich" testing is routinely done primarily on bulls, using a prepuce for scraping and aspiration technique to obtain the sample for culture and PCR test Trichomonas fetus DNA. Trich culture and PCR is done primarily on bulls because will become persistently infected when they are exposed to an infected cow. It is bulls that maintain the infection within the herd. After a non-immune cow becomes infected she will abort or not become pregnant. She will continue to cycle and transmit the infection to other bulls when she is in estrus. She will develop an endometritis that results in a strong immune response and clearance of the infection, after which she is no longer infectious and may go on to conceive but have a very late calf.

- **Equine Infectious Anemia (EIA) testing**: Testing for EIA requires serum for a serologic AGID “Coggins Test” or an EIA ELISA test. EIA testing is required by all states for interstate movement of horses into their state. An EIA test is also required by most horse shows and competitive events prior to admittance to the event.

- **Testing for or diagnosis of any other disease that must be identified and prevented from being introduced into an importer's state or country by animals or animal products from our clients. These are usually diseases of concern by the importing entity and are spelled out by the state or countries importation requirements.**

- **Prevention of the introduction of exotic (foreign) animal diseases into the U.S.**

  Probably the most important regulatory functions of the USDA and all veterinarians

  The USDA has developed and enforces import requirements for animals and animal products originating in foreign countries that are to be imported into the U.S.

  These entry requirements are country of origin specific. They are based on knowledge of the exporting country's status with respect to contagious diseases of concern.

  Diagnostics, testing, and quarantine of animals both prior to importation and after importation can assure identification and prevent the importation of exotic animal diseases.

  One other concern is the possibility of foreign animal diseases being brought into the U.S. on the clothes and shoes of international visitors. These travelers may have been exposed to contagious animals on farms and food markets in foreign countries. U.S. Immigration and Customs Enforcement and the USDA are addressing this issue during customs inspections upon arrival in the U.S. Any prohibited agricultural product are identified through declarations, interviews and inspections of baggage and shipment arriving in the U.S. Any prohibited or suspicious foodstuffs arriving from foreign counties are confiscated and destroyed. This is particularly important with arrivals from countries that are infected with animal (or plant) diseases and parasites of concern.
The USDA monitors the health status of foreign countries through membership in the World Organization for Animal Health (OIE – French for ‘Office International des Epizooties’), along with 178 other countries. All members are required to report epidemiologically significant disease occurrences in their country so that OIE members are aware of international disease status.

**Production veterinary medicine** -
A scientific approach to animal husbandry and veterinary medicine. The goal of a production medicine program is to increase production and therefore profitability of an animal enterprise. The client served is a "Industrial animal" producer; such as a dairy, beef cow-calf ranch, a beef feedlot, swine producer or feeder, performance horse breeder and any other client that produces animal based products.

Production Veterinary Medicine incorporates:
- Classical Veterinary Medicine
- Preventative Veterinary Medicine
- Animal Sciences
- Nutrition
- Reproduction
- Production record systems
- Phenotypic selection assistance
- Genetic selection assistance
- Client and employee education
- Artificial Insemination (AI); breed livestock to the best sires available in the U.S.
- Embryo Transfer (ET); utilizing the top female genetics within the herd or flock.
- Estrus cycle control e.g. Estrus Synchronization; this will allow for greater efficiency and labor savings in implementing AI and ET programs.

Production monitoring, data record systems
- Birth weight
- Calving difficulty score
- Weaning weight (adjusted to 205 days ~ 7 mo.)
- Yearling weight (adjusted to 365 days)
- Mature weight and frame score (how heavy and how tall?)
- Rate of gain (Lbs. / day)
- Feed efficiency (Lbs. of feed / Lb. of gain)
- Periodic (or daily) milk weights for producing dairy cattle
- Somatic cell counts in milk

Phenotypic trait selection
- Selection and culling based on the visible and measurable traits exhibited by an individual animal
- Phenotypic traits are the result of expression of an animals genetic make up and the environmental influences on the expression of the genetics
- In addition to conformation and outward appearance, some measurable phenotypic traits in beef cattle include: birth weight, growth rate (ADG), 205 day weaning weight and 365 day yearling weight. Carcass traits are measured and analyzed for animals that are slaughtered and also living, non-terminal, breeding animals whose carcass is evaluated using ultrasonography. The carcass traits of interest include: meat quality grade or marbling (% of intramuscular fat), ribeye cross sectional area, between the 12th and 13th rib (ribeye area correlates with overall yield of meat in the carcass), and back fat thickness (a measure of waste; the less back fat the better!)
- In dairy cattle measurable phenotypic traits include: milk quantity (pounds of milk in a 305 day lactation), milk fat (lbs and %), milk protein (lbs and %), and somatic cell score (white cell count in the milk is an indicator of mastitis susceptibility).
cattle, body conformation, including udder conformation and feet and leg conformation is also evaluated.

- Numeric analysis of an animal's performance trait measurements can be performed if several simple rules are followed when obtaining the measurement and performing any analysis.

  ñ Contemporary Group (CG): The animals that are to be analyzed must all be measured and recorded together as a Contemporary Group (CG). A CG is a group of animals of the same sex, similar age, and must have been housed together in the same geographic area with identical environmental and management conditions. Direct analysis phenotypic measurements can only be done between animals that belong to a common contemporary group.

  ñ Before performing a mathematical or statistical analysis of a phenotypic trait that involves animal performance over a period of time; the measurements must be adjusted to a common number of days, or to a common age. For example the amount of milk produced in a lactation must be adjusted to a standard lactation length of 305 days.

  ñ A good example of a mathematical analysis of a performance trait is the comparison of the weaning weights of a group of beef calves on a ranch. The first step is to adjust the weaning weight to a common number of days of age. In this case 205 days is used as the standard. The actual age of the calves may vary +/- 40 days on the day that their weaning weight is obtained. To adjust the Actual Weaning Weight (Actual WW) obtained at an Actual age of 165 to 245 Days Of Age (Actual DOA) into an Adjusted 205 day Weaning Weight (Adj WW), the following data and calculations are required:

    1. Relevant data: Animal ID, Sire ID, Dam ID, Birth Date (BDate), Birth Weight (BW), Actual Weaning Date (AWDate), Actual Weaning Weight (ActWW). When a computer spreadsheet is used to do the analysis; the data for each animal are entered into separate cells on the animal's spreadsheet row.

    2. That data is then used in to calculate the following:

    3. Days Of Age (DOA) = AWDate − BDate

    4. Weight Gain (WG) = ActWW − BW

    5. The real phenotypic trait that determines the weaning weight of a calf is the "Average Daily Gain" (ADG) = WG / DOA

    6. The final calculation is: Adj WW = (ADG * 205) + BW (Birth Weight)

    7. On the animal's spreadsheet row, each of the 4 formulas can be written individually into separate cells. Alternatively the formulas can be easily combined into a single formula.

    8. Adj WW=((ActWW − BW) / (AWDate − BDate)) * 205 + BW

    9. The formulas for the first animal entered into the spreadsheet are then copied to the rows for each animal in the analysis.

    10. Finally statistics such as overall mean, median, standard deviations, and sire and dam relative mean, median, standard deviations

  ñ Once the adjusted measurements are calculated an index (or ratio) may be calculated, using the adjusted data from animals within the CG. The index is a measure of performance of an animal above or below average, expressed as a percentage of average, within the CG. It can be used to compare animals within the CG, but even more important; it can be used to compare animals outside of the contemporary group if they are also indexed within their own CG. The effect of environmental influences between two CGs; such as topography, elevation, weather, feed quantity, feed quality, and other management factors, are compensated for when two animals from different CGs are compared using an index. The index can also be used in calculating genetic merit statistics.
For example: In one CG, the average Adjusted WW is 600 Lbs. An individual animal, which has an Adjusted WW of 640 Lbs, will have a performance ratio of: 640/600 (1.0666), or a weaning weight index 107. (value of: (640/600) * 100 = 106.66 (~107% of average)

Another CG has similar cattle but different environmental conditions. In this CG the average Adjusted WW is 500 Lbs. An individual animal in the latter CG has an Adjusted WW of 540 Lbs. This animal has a performance ratio of: 540/500 (1.08), or a weaning weight index 108.

Even though the latter animal is 100 pounds lighter than the first, it has a similar (slightly better) index value. One would conclude that both of these animals are likely to have similar genetic merit. By indexing the animals we are able to compare animals from different CGs or herds, even from different areas of the country.

- Racehorse breeders and enthusiasts can use race results and speed indexes in a similar fashion, when deciding how to breed race horses; or which horse to “wager on” at the track.

Genetic trait selection using: Expected Progeny Differences (EPDs for beef breeds of cattle), Predicted Transmitting Ability (PTAs for dairy breeds of cattle), and Speed indexes (for horse breeds).

- Selection, breeding and culling through the use of genetic statistics that are based on analysis of the phenotypic trait data from an animal and related animals. Genetic statistics for a performance trait are calculated for an animal by performing a weighted average of the ratio values for that animal it’s ancestors, it’s siblings, and most importantly, it’s offspring. Once the breed association has calculated the genetic statistic for an individual, that animal can then be compared for that trait to all animals in the population, regardless of location or management. The population is defined as all animals within the same breed.

- By including the performance data of ancestors, siblings and offspring in the statistical analysis, the effects of environmental influences are reduced and a truer picture of an animal's genetic value can be obtained.

- Breed associations, the USDA or dairy herd improvement associations (DHIA) usually perform the statistical analysis utilizing phenotypic data submitted by producers. These organizations are able to calculate the genetic statistics since they have access to data from thousand or even millions of animals; including all of the related animals whose phenotypic data has been submitted.

- The statistics, which are returned to the producers and to breed associations are:

  ¡ In dairy breeds the genetic statistic for an individual trait is referred to as a “Predicted Transmitting Abilities” (PTA). For example the Holstein Association computes the PTA genetic evaluations for production traits (Milk production and composition), type traits (conformation), and longevity. PTA statistics for milk production traits attempt to project expected performance of the animal's progeny above or below a baseline animal's progeny average production in a 305-day lactation. The PTAM (milk) is expressed as pounds of milk above (or below) the baseline animal's progeny 305-day lactation. PTAF (fat) estimates progeny Lbs. and % of fat produced above (or below) the baseline animal's progeny average in a 305 day lactation. PTAP (protein) estimates Lbs. and % of protein produced above (or below) the baseline animal's progeny average in a 305 day lactation. Somatic Cell Score and Productive Life PTAs are also calculated. The US Department of Agriculture’s Animal Improvement Programs Laboratory (USDA–AIPL) is also involved in calculating dairy PTAs for non-registered cows.
“Total Production Index” (TPI) and Net Merit statistics are overall composite merit statistics calculated by dairy breed associations for dairy cattle. TPI statistics attempt to project overall performance above or below average for pounds of milk, butterfat, milk protein and other measures.

“Expected Progeny Difference” (EPD) statistics are calculated by beef breed associations for beef cattle. EPD statistics attempt to project expected performance of progeny above or below average for weaning weight, yearling weight, carcass weight, marbling (percent of intramuscular fat), back fat thickness and other measures.

The PTA and EPD statistics are assigned to individual animals but as the name implies they predict the performance of that animal's offspring. The accuracy of these statistics increase as the animal has more offspring. Since the progeny performance is weighted the heaviest in calculating an animals EPD or PTA statistics, a bull's statistics will have much higher accuracy than a cows since bulls will have many more offspring than cows will have in a lifetime. Even a young 3 year old bull used only for natural service may have 60 to 100 offspring compared to a cow which will have only 2.

The final analysis and best use of these statistics is comparing a cow's or a bull's numbers to other animals when making breeding decisions that will maximize improvement in performance of the offspring.

DNA tests can identify carriers of genetic defects, verify parentage and evaluate genetic variation for several performance traits. These tests can be performed using simple samples such as dry blood spots on an “FTA” card, or hair samples from large tail hairs that include the hair root. These samples can be stored for years in a file cabinet without refrigeration.

DNA “Marker Assisted Selection” (MAS) tests for characterization of performance traits, augment the value of traditional phenotypic measurements and computational genetic selection. These DNA tests are also sometimes referred to as genomic testing. These genomic tests correlate well with measurable phenotypic traits in many cases.

Beef cattle with superior growth and carcass traits and dairy heifers (and bulls) with superior milking characteristics can be identified using MAS. Animals that have genomic test data on file will have increased accuracy of EPD and PTA statistics.

Young calves can be identified as superior with respect to a economically important trait, many months before that trait can even be measured phenotypically.

A list of beef profile genetic tests for “Economically Relevant Traits”, (From Igenity, a DNA testing laboratory) include:

- Residual Feed Intake; a measure of feed efficiency
- Average Daily Gain;
- Beef Tenderness; in lbs. - Warner-Bratzler Shear Force (WBSF)
- USDA Marbling Score
- % Choice & higher
- Yield Grade
- Back Fat Thickness (in)
- Ribeye Area (in²)
- Heifer Pregnancy Rate (%)
- Stayability (%)
- Maternal Calving Ease (%)
- Docility (%)

A list of ‘Igenity’ dairy profile genetic tests for “Economically Relevant Traits” include:

- Milk Yield
- Fat
- Protein
Below is a list of beef cattle genetic defects tests

<table>
<thead>
<tr>
<th>Full Name</th>
<th>Abbreviation</th>
<th>Result Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthrogryposis multiplex</td>
<td>AM</td>
<td>AMF: free, AMC: carrier, AMA: affected</td>
</tr>
<tr>
<td>Congenital Contractural Arachnodactyly</td>
<td>CA</td>
<td>CAF: free, CAC: carrier, CAA: affected</td>
</tr>
<tr>
<td>Chondrodysplasia</td>
<td>CHO</td>
<td>CHOF: free, CHOC: carrier, CHOA: affected</td>
</tr>
<tr>
<td>Coat Color Diluter</td>
<td>DL</td>
<td>DLF: free, DLC: carrier, DLH: homozygous</td>
</tr>
<tr>
<td>Dun</td>
<td>DN</td>
<td>DNF: free, DNC: carrier, DNH: homozygous</td>
</tr>
<tr>
<td>Idiopathic epilepsy</td>
<td>IE</td>
<td>IEF: free, IEC: carrier, IEA: affected</td>
</tr>
<tr>
<td>Alpha-mannosidosis</td>
<td>MA</td>
<td>MAF: free, MAC: carrier, MAA: affected</td>
</tr>
<tr>
<td>Neuropathic hydrocephalus</td>
<td>NH</td>
<td>NHF: free, NHC: carrier, NHA: affected</td>
</tr>
<tr>
<td>Osteopetrosis</td>
<td>OS</td>
<td>OSF: free, OSC: carrier, OSA: affected</td>
</tr>
<tr>
<td>Pulmonary hypoplasia with anasarca</td>
<td>PHA</td>
<td>PF: free, PC: carrier, PA: affected, PNR: no result</td>
</tr>
<tr>
<td>Tibial hemimelia</td>
<td>TH, THO</td>
<td>THF and THFO: free, THC and THCO: carrier, THA and THAO: affected</td>
</tr>
</tbody>
</table>

Merial's Igenity genetic testing laboratory: [http://www.igenity.com/pdfs/forms/Igenity Results Key Beef.pdf](http://www.igenity.com/pdfs/forms/Igenity Results Key Beef.pdf)

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<tr>
<th>Full Name</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Brachyspina in Holsteins</td>
<td>BY</td>
<td>TY: free, BY: carrier, Affected is lethal</td>
</tr>
<tr>
<td>Bovine Leukocyte Adhesion Factor Deficiency</td>
<td>BLAD</td>
<td>TL: free, BL: carrier, BLH: homozygous affected is lethal</td>
</tr>
</tbody>
</table>
Complex Vertebral Malformation (CVM) is a lethal recessive condition. TV: free, CV: carrier, CVH: homozygous affected.

Deficiency of Uridine Monophosphate Synthase (DUMPS) is a lethal recessive condition. TD: free, DP: carrier, Affected is lethal, fetus is aborted.

Mule Foot or Syndactylism (MF) is a non-lethal recessive condition. TM: free, MF: carrier, MFH: homozygous affected.

Factor XI deficiency

Citrullinemia

Haplotype 1 Impacting Fertility (HH1) is free in HH1F, carrier in HH1C, not tested in HH1N.

Haplotype 2 Impacting Fertility (HH2) is free in HH2F, carrier in HH2C, not tested in HH2N.

Haplotype 3 Impacting Fertility (HH3) is free in HH3F, carrier in HH3C, not tested in HH3N.

Links: to come

Equine Genetic Defect ************

- **Hyperkalemic Periodic Paresis (HyPP)** - Quarter Horse descendants of a stallion known as “Impressive” may carry a defective gene that results in HyPP. Heterozygotic animals are moderately affected while homozygotic animals are severely (sometimes fatally) affected. These horses have abnormal sodium channels, with abnormal (slow) sodium expulsion and potassium re-uptake in muscle fibers, causing muscular hyper excitability, and eventually muscular weakness and apparent “paresis”. Diagnosis of the HyPP genetic condition is by submitting tail hairs (with good hair follicles intact) to the veterinary genetics laboratory at Univ. California, Davis.

- **HERDA: Hereditary Equine Regional Dermal Asthenia (HERDA)** - Is a genetic disease caused by an abnormal simple recessive gene. It causes affected horses to have abnormal skin collagen resulting in hyper-extensible, fragile skin. When the skin is damaged it heals very slowly and scars excessively. These horses frequently are euthanized because of disfiguring scars and skin infections.

- **Cerebellar Abiotrophy (CA):** Genetic Cerebellar degeneration. Usually starting shortly after birth, and visibly present by 1 year of age. Result in a stiff ataxic gait. Occurs primarily in Arabian horses but has been reported in some other breeds also.

- **Lavender Foal Syndrome (LFS),** is a fatal recessive condition of Arabian horses. It causes neuromuscular dysfunction, obvious at birth, and also a coat color dilution resulting in the "lavender" color. The neurologic symptoms include seizures, opisthotonus, leg paddling, nystagmus and the resulting inability to stand. These symptoms are present in utero, and often cause dystocia.

- **Overo Lethal White Syndrome (OLWS)** is a fatal recessive condition of foals that are homozygous for the "frame overo" gene which occurs in the Paint Horse breed. Two copies of this gene cause the solid white coloration and also results in parasympathetic malfunction of the digestive system, preventing normal passage of ingesta through the colon. Foals show profound colic soon after consuming milk, and are usually euthanized or die within a few days of birth.

- **Severe Combined Immunodeficiency (SCID)** is a lethal, autosomal, recessive genetic trait that occurs in Arabian horses. Homozygous foals are unable to mount a humoral or cellular immune response to invading bacteria or viruses.
Polysaccharide Storage Myopathy (PSSM) is a dominant autosomal genetic mutation that will cause an intermittent, recurring form of tying-up. The typical, and correct, physical diagnosis of an episode of myopathy is exertional rhabdomyolysis, or myoglobinuria. Horses experiencing an episode of PSSM will present with the typical muscle damage, pain, inability to move and myoglobin discoloration of the urine. Not all animals that experience exertional rhabdomyolysis are carriers of this genetic defect.

Malignant Hyperthermia

- Parentage verification, using DNA typing technology (previously referred to as DNA “fingerprinting”) is required by breed associations to be used on all artificial insemination sires and on embryo or oocyte donor mares and cows. DNA typing of parents allows for the confirmation of the parentage. If there are discrepancies that bring parentage into question the offspring resulting from ET and AI can be DNA typed to confirm or dis-prove parentage with a 1 in hundreds of thousands chance of error. This is many orders of magnitude more accurate than the previous technology used for parentage verification; blood typing.
- As DNA typing technology has become cheaper, it has been used on commercial (unregistered) offspring from valuable bulls. This allows a rancher to identify a calf’s sire even when multiple bulls have been used for natural breeding in large pastures. This allows for phenotypic and genetic evaluation of the offspring from valuable sires and dams. Parentage verification will also provide important information on fertility and the serving ability (breeding ability) of the sires. Fertility and libido are important genetic traits for natural breeding sires. These traits can be quantified in multi-sire situations by identifying the sire of the calves.

Culling programs;
- Purpose is to remove non-productive or unprofitable animals from the herd.
- Culling can be based on many different criteria.
- Typical criteria are listed below, in an order that reflects the most common reasons on top:
- Reproduction failure
- Individual animal reproductive fertility
- Sickness
- Health criteria such as disease resistance (brisket disease, metabolic disease in dairy cattle, etc.)
- Performance
  - Milk production
  - Beef cattle growth performance
  - Carcass quality of offspring
  - Racing & other athletic performance in horses
- Genetics

Integrated Resource Management
- A program where ranchers, dairymen, and farmers use economists, bankers, veterinarians, nutritionists, agronomists, academicians and other specialists in a team approach to farm and ranch management assistance
- Economic analysis of all resources and procedures that may be used allows the producer to exclusively make decisions that have a high probability of enhancing profits.
- These programs are sponsored by university extension services and agricultural companies
2. Physical Examination

Purposes
- Diagnosis
- Pre-purchase exam
- Insurance exam
- Performance problem analysis

Types
- Physical exam
  - Observation
  - Auscultation
  - Palpation
  - Smell
  - Special tests
- Surgical, exploratory
  - Laparotomy
  - Laparoscopy
  - Arthroscopy
- Laboratory
  - Hematology
  - Serum Chemistry
  - UA
  - Microbiology
  - Pathology
- Necropsy

Two common approaches to physical exam
- 1. Body systems approach to physical exam; this is always the best approach in most situations. Each body system exam is completed prior to moving on to the next system. The main systems are:
  - Integument
  - Musculo-skeletal
  - CNS
  - Ocular
  - Respiratory
  - Cardiovascular
  - Lymphatic, Immune
  - Digestive
  - Reproductive
  - Urinary
  - Mammary glands

- 2. Regional approach to physical exam; this approach to physical exam is common on large animals. In this approach all systems are examined in a region by region order. The body systems must still be examined thoroughly. Using a systematic physical exam form is helpful when doing a regional physical exam.
  - Head and neck
  - Left Side
    - Legs
    - Thorax
    - Abdomen
  - Perineal area, rectum and genitalia
  - Right Side
    - Abdomen
    - Thorax
    - Legs
Common to all approaches to physical diagnosis

- **Complaint, Signalment and History**
- **Observe from a distance – unrestrained**
- **Body system examinations**

Special tests and procedures for various body systems

- **Musculoskeletal Systems**
  - **Localization of Lameness**
  - Trotting the horse on a hard surface
  - Observation of asymmetrical movement of legs
  - Asymmetrical movement of the head in relation to the animal’s stride
  - Asymmetrical positioning or movement of the rump relative to the hind leg stride
  - Identification of the lame leg and sometimes the region of the leg injury

- **AAEP System of Grading Lameness**
  - Grade 0: Lameness not perceptible
  - Grade 1: Lameness difficult to observe; not consistently apparent regardless of circumstances.
  - Grade 2: Lameness is difficult to observe at a walk or while trotting in a straight line, consistently apparent under certain circumstances.
  - Grade 3: Lameness is consistently observable at a trot under all circumstances.
  - Grade 4: Lameness is obvious; marked head nodding, hitching or shortened stride.
  - Grade 5: Minimal weight bearing in motion and/or at rest, inability to move, “three-legged lame”. This type of lameness is often associated with fractures, subsolar abscesses, severe tendonitis, and septic arthritis.

- **A locomotion score in dairy cattle, is very similar to an equine lameness grade.**
  1. Normal: The cow walks and stands with a flat back. She walks normally.
  2. Slightly abnormal gait: The cow stands with a flat back but arches her back while walking. She walks normally.
  3. Moderately lame: The cow stands with an arched back and walks with an arched back. She short-steps while walking.
  4. Lame: The cow stands with an arched back and walks with an arched back and walks with decreased weight bearing on one limb (limps).
  5. Severely lame: The cow stands and walks with an arched back and refuses to bear weight on a limb.

- **Nerve blocks / Perineural anesthesia**
  - By blocking nerves involved in pain perception at different levels of the leg, lameness can frequently be localized to certain areas of the leg
  - Always begin a series of nerve block distally in order to desensitize the smallest area possible. Typically the 1st injection is in the posterior branch of the digital nerve in the area just above the bulbs of the heel. Injections are usually given both medially and laterally on these distal nerves.
  - Usually just clipping the hair and an alcohol prep is all that is required. When injecting near a synovial structure a surgical scrub may be warranted.

- **Arthrocentesis**
  - Placing a needle, with sterile technique, into a joint in order to obtain and evaluate the synovial fluid. Sterile injections can be administered subsequent to the aspiration, through the same needle placement.
Synovial fluid analysis
- Color and Clarity: transparent, slightly yellow
- String test: Fluid viscosity observation, - a drop of normal synovial fluid should form a long, 3cm to 10cm string
- Mucin clot test: hyaluronic acid and other glycosaminoglycans will form a firm clot content of fluid
- Cytological analysis: normal nucleated cell count <100 cells/ml
- Protein content
- Culture
- Diagnostic intra-articular anesthesia can be performed at the same time as sampling
- Therapeutic agents should be readied for infusion at the same time that a synovial sampling is done.
- Surgical, 3 scrub, preparation should always be used prior to arthrocentesis or arthroscopy.

Arthroscopy
- Imaging of a joint using a small diameter, short endoscope.
- Arthroscope are
- Surgical, 3 scrub, preparation should always be used prior to arthrocentesis or arthroscopy.

Serum chemistry
- CPK
- AST
- Ca++
- Phosphorus

Muscle biopsy

CSF evaluation

Radiography

Leg lift restraint to assist in examination and treatment
- Lift the left front leg when working on the right front leg; and visa-versa
- Lift the right front leg when working on the right rear leg
- It is highly recommended to not lift a rear leg for restraint purposes due to the strength of the rear legs.

Ultrasonography
- Ultrasonography in B Mode, or real-time (20-60 video frames per second), cross sectional imaging mode is often used in evaluating portions of the musculoskeletal system
- Primarily muscle, tendon and ligament imaging.
- Imaging skeletal component is not very useful since bone is nearly 100% reflective and thus impervious to sound.
- What is ultrasonography?

An ultrasound machine in “brightness mode” (B mode) is the basis for veterinary ultrasonography. The most popular transducers contain a linear array of ceramic (piezo-electric) crystals. These crystals will vibrate for a short period of time when they receive an electrical charge. That vibration will produce sound frequencies of 3 million to 15 million hertz (cycles per second), depending on the size and thickness of the crystal. The ultrasound frequency is referred to in a unit called a megahertz; i.e. 3 MHz to 15 MHz. The transducer array will emit a “thin sheet” of rapidly repeating ultrasound pulses. The pulse frequency is typically 20 to 60 pulses per second. These short sound pulses will penetrate the tissues that is to be imaged, distal to the probe. Some of the sound will be echoed back to transducer's crystal array, during the relatively long period between the emitted pulses. The transducer's electrically charged piezo electric crystals will receive these echoes and convert that sound back into an electrical signal. That signal, from the reflected sound, will then be interpreted by the machines CPU and used to
produce a video image that represents a two dimension cross sectional, image of the tissues distal to the probe.


- **Alimentary System**
  
  Mammalian Digestive physiology
  
  Carbohydrate digestion
  
  Monosaccharides are a single sugar molecule. The main biological sugars are hexose - 6 carbon - sugars; and pentose - 5 carbon - sugars. Examples are
  
  Hexose: Glucose, Galactose, Fructose
  
  Pentose: Ribose, Deoxiribose
  
  Polysaccharides include the important nutritional disaccharides such as:
  
  Sucrose: is a disaccharide polymer of the monosaccharides D-glucose and fructose, that is produced by plants.
  
  Lactose: also is a disaccharide polymer of the monosaccharides glucose and galactose. It is produced by mammals and is present in milk.
  
  Maltose is another disaccharide. It consists of two glucose molecules. It is present in many cereal grains, sweet potatoes, molasses and barley malt used for beer production. But, it is primarily a breakdown product of starch degradation in these high starch foods, and in mammalian digestion of starch.
  
  Important nutritional polysaccharides also include large complex polymers of glucose and other monosaccharides molecules
  
  Starch is a large polymer of glucose where adjacent glucose molecules are joined by a 1-4-alpha glycosidic bond into very long chains of glucose that is called amylose. Adjacent chains are cross linked with 1-6-beta bonds forming amylpectin which is a synonym for starch
  
  Glycogen is the animal version of starch utilizing the same glycosidic bonds.
  
  Cellulose is a complex polymer of glucose where adjacent glucose molecules are joined by a 1-4-beta glycosidic bonds which are not digestable by mammals. Many bacteria, fungi, yeast and protozoa in insects are able to digest cellulose with cellulase enzymes.

- **Basic herbivore digestive physiology**
  
  Equine
  
  Simple stomach
  
  Stomach tube
  
  Used simultaneously for diagnosis and therapy
  
  Always be prepared to obtain gastric decompression samples
  
  Equine stomach tubes are passed through a nostril into the nasal pharynx and then into the esophagus. This is referred to as a naso-gastric tube.
  
  It is common for the N-G tube to enter the trachea, by accident. Extra caution should be exercised to ascertain that the NG tube is in fact in the esophagus before forcing the tube further, and certainly before administering any medication. So how do you tell if the tube is in the trachea? If the tube is in the trachea air will move in and out, through the tube, with nearly no resistance. If the tube is in the esophagus there is greater resistance to air being blown into the tube and essentially no air can be sucked out of the tube, since the esophagus collapses over the distal end of N-G tube due to the negative pressure. (Yes use your mouth to suck on the stomach tube). These techniques, i.e. blowing and sucking on the stomach tube while passing the tube into the esophagus, work equally well on horses and cattle.
• Mineral oil for instance, administered into the lungs, even in a small 1-3 ounce dose, will cause severe and occasionally fatal foreign body pneumonia.
• In cattle a frick speculum is placed in the mouth prior to passing the stomach tube. This protects the N-G tube from the cow’s teeth and the inevitable chewing damage that would occur to the plastic tube, if no frick speculum was used.

Hematology and serum chemistry
• Liver enzymes, pancreatic enzymes

Abdominocentesis
• Usually performed on a horse with progressive colic or other acute abdomen symptoms.
• Usually a single 16 to 18 gauge, B bevel needle is inserted on the ventral midline at the most ventral location, or 5 to 10 cm posterior to the xyphoid cartilage.
• Prep is usually 2 or 3 pre-surgical scrubs, and then alcohol.
• Cytological evaluation
• Chemical composition
• Culture samples
• Gram stain

Glucose absorption testing for malabsorption
• Oral monosaccharides require no digestion prior to absorption from intestines. 10% Glucose is administered via stomach tube to the horse after a 12 hour fast, 10 ml/kg. A baseline blood glucose level must be obtained prior to dosing.
• Serial blood glucose concentrations taken every 30 minutes
• 50% increase over baseline in 2 to 4 hours in normal horses

Lactose absorption testing for lactose intolerance in foals
• Lactose is a disaccharide, which requires lactase for digestion, prior to absorption from the small intestines.
• Serial blood glucose concentrations taken every 30 minutes
• 35 mg/dl increase in 1 to 2 hours in normal foals

Liver biopsy
• Percutaneous route for general hepatic disease and diagnostics
• Laparoscopic or laparotomy for biopsy of specific lesions

Rectal exam
Endoscopy
• Esophagus
• Stomach, especially in diagnosis of gastric ulcers
• Colonscopy
• Laparoscopy

Exploratory laparotomy

• **Respiratory System**
Common conditions of the respiratory system
• Pharyngitis, Laryngitis,
• Tracheitis, Bronchitis
• Pneumonia
  Ÿ **Bronchopneumonia**
  In the bronchial tree and alveoli
  Lung consolidation
  Red hepatization
  Grey hepatization
  Ÿ **Fibrinous pneumonia**
  Inflammation in the pleural cavity
  Ÿ **Interstitial pneumonia**
  Inflammation in the interstitial space (between the bronchioles and alveoli.
Causes a shortness of breath that is characterized as expiratory dyspnea. There are numerous causes all leading to interstitial pneumonia including:

- Atypical interstitial pneumonia of cattle caused by the toxic effects of 3 methyl-indole,
- Viral Interstitial pneumonia in cattle, usually caused by parainfluenza viruses,
- Heaves in horses which is an allergic asthma like condition.
- Pleuropneumonia, an extension of a pneumonic condition into the chest cavity causing pleuritis. Pleuropneumonia can be caused by several bacterial species.

- Laryngeal hemiplegia
- Strangles in horses

Percussion
- Detect consolidated areas of the lungs
- Detect fluid level in the thoracic cavity

Endoscopy, using a flexible endoscope
- Naso-pharynx
- Guttural pouch opening and guttural pouch
- Larynx
  - Laryngeal hemiplegia is a common cause of exercise intolerance in horses. It is referred to as “Roaring”.
  - Damage to the recurrent laryngeal nerves, innervating the abductor muscle of the arytenoids cartilage, is one cause of laryngeal obstruction
- Trachea
- Bronchi

Hematology

Radiology
- Particularly useful in smaller animals under 400 pounds
- Large horses require a LARGE X-ray machine

Blood gasses

Transtracheal wash and Bronchoalveolar Lavage
- Culture samples
- Gram stain
- Cytological evaluation

Thoracocentesis
- Culture samples
- Gram stain
- Cytological evaluation
- Chemical composition

Ultrasonography
Guttural pouch catheterization

- **Cardiovascular System**
  Electrocardiography
  Echocardiography
  - Ultrasonography
  - 2 dimensional, real time image of heart
  - 1 dimensional X time image of specific area of heart, M-mode
  - Doppler real time imaging reveals rate and turbulence in blood flow (using color enhancement of fluid flow)

  Exercise testing, tread mill
  Radiography
  Cardiac catheterization
  - Pulmonary Arterial Pressure test (PAP test), in cattle
    - Hypoxia that occurs at high elevation causes increased cardiac output
• Genetic predisposition to thickened pulmonary arteries with decreased elasticity leads to elevated PAP especially at altitudes over 5000 feet
• Chronically Elevated PAP causes right heart failure
• Right heart failure in cattle living in the mountains is called “Brisket Disease” or “High Mountain Disease”
• Peripheral edema especially ventral edema, dyspnea and weakness are main symptoms. Brisket edema is common.
• PAP testing bulls and elimination of those with elevated PAP reduces the incidence of this deadly condition

• Reproductive System

Vaginal Speculum exam
- Caslick Speculum for mares
- Frick Speculum for cattle and mares
- Modified Balfour retractor with 8”-10” blades for intravaginal surgery

Microbiologic exam, vagina, uterus, male urethra, semen
- Gram stain of exudate, and discharge
- Culture of exudate, vaginal / uterine discharge and semen
- Tiegland culture swab for uterine culture

Direct palpation
- Vagina
- Cervix
- Scrotum and Penis

Indirect palpation
- Testicular exam
- Portion of the penis that are under the perineal skin
- Rectal exam, the most common reproductive exam done in large animal medicine
  - Indirect palpation of many structures through wall of the rectum
  - Female: Cervix, Uterus (and the embryo or fetus in pregnant animals), Ovaries, Oviduct
  - Male: Seminal vesicles, Prostate, Ampulla, Inguinal rings, Root of the penis
- Pregnancy diagnosis through rectal palpation of the uterus is probably the most common reproductive exam performed in large animals.

Ultrasonography, in recent years this is probably the second most common reproductive procedure that is done in LA medicine
- Most reproductive ultrasonography is done via the rectum, using a straight linear array probe in B-Mode. Advanced pregnancies (> 4 months) can be examined through the abdominal wall, sheep and swine can be examined trans-rectally or trans-abdominally
- Early pregnancy diagnosis (as early as 11 days in the mare, 25 days in cattle)
- Another advantage of early ultrasound pregnancy diagnosis in horses is that a twin pregnancy can be identified. Twinning in mares rarely results in two or even one live foal being carried to term. With an early pregnancy exam, performed at about 13 to 15 days after heat, one of the twins can be eliminated in order to cause a single birth. The easiest time to do this is prior to embryo fixation, while the embryos are still mobile in the uterus. The twin that is to be eliminated is manipulated away from the other and ruptured using digital pressure. The maximum number of days that should pass, after the animal ovulates or goes out of heat, in order to accomplish this is 15 days. There are other more invasive methods that can be used at later stages of gestation to eliminate one member of a twin pregnancy. Another simple method that can be used prior to 30 days is to simply administer prostaglandin F2 alpha in order to cause the mare to return to heat. This will result in elimination of the twins, and the mare can then be re-bred at that heat.
- Age estimation can be done using various measurements
  - Amnion diameter
  - Crown nose length
Crown rump length
Thoracic diameter
Orbit diameter

- Fetal sex determination
- Ovulation prediction and detection of ovulation, especially important in mares
- Ovarian pathology
- Uterine pathology
- Testicular imaging

Endometrial biopsy, cytology and histology
- Uterine biopsy of mares is common for determining the prognosis for conception
- Uterine biopsy is less commonly done in cattle since the placental sites are discreet and only comprise a small percentage of the endometrial surface

Semen evaluation
- Evaluation of the semen is commonly done pre-breeding or pre-purchase to help assure fertility of a potential sire.
- Semen is also evaluated every time that a semen ejaculate is collected for artificial insemination (AI) purposes. This semen exam includes: semen volume, motility (% of progressively motile sperm cells) concentration (number of cells per ml), and for normal morphology. This exams allows for the calculations that are needed to properly extend and divide the ejaculate into appropriate inseminating doses with an adequate number of normal motile sperm cell to achieve the maximum possible conception rate.
- Stallion semen is typically collected with an artificial vagina (AV)
- Bull semen can be collected with an AV or can be sampled using “Electro-Ejaculation”

Embryo evaluation
- Embryos can be collected at 6 to 7 days after ovulation by catheterizing the uterus and flushing it with a tissue culture solution. They are evaluated for fertilization, stage of development and general quality.
- Embryo collection can be used as a diagnostic protocol to evaluate fertilization and embryo development in a valuable female that is displaying signs of sub-fertility. Evidence of sub-fertility could include repeated return to estrus after breeding with a proven fertile sire or artificial insemination (AI) using fertile semen.
- Embryo collection is most frequently done for strict the production purpose of genetic improvement of the herd. Regardless of the reason, every embryo collected is examined for the embryo developmental stage and quality (normality) of the embryo.

Reproductive and Endocrine hormone assays
- BioPRYN is a pregnancy test for use in ruminants that tests for a pregnancy associated glycoprotein known as “Pregnancy Specific Protein B” (PSPB). The BioPRYN tests require 2-3 ml of serum.
  - In beef & dairy cattle the PSPB test is accurate at 28 days post breeding.
  - The sheep and goats BioPryn test can be used at 30 days of pregnancy.
  - There is also a BioPRYN PSPB pregnancy test for elk, moose, deer, caribou, African antelope and other ruminant animals. This test is accurate at 40 days post-breeding.
  - Idexx has a similar test for “Pregnancy Associated Glycoproteins” (PAG) for use with dairy and beef cattle. This test can be run on serum or milk samples.
- Progesterone assay, the most common test done
  - Determine the competency of a CL in producing progesterone,
  - Use to make a presumption of pregnancy in a ruminant at 21 days post breeding.
- Estradiol
- LH level, LH surge detection
- Estrone sulphate
  - pregnancy detection in swine and other species.
 Estrone sulphate can also be used to identify cryptorchid stallions or “geldings” with remnants of testicular tissue.

- **Urinary System**
  Rectal Exam and direct palpation (penis, urethra, bladder, kidneys)
  Urinalysis
  - Routine UA
  - Stone and crystal analysis
  Serum chemistry
  Catheterization
  Abdominocectentesis (male foals, rupture of bladder at parturition)
  Ultrasonography

- **Dermatologic**
  Ectoparasites
  - Chigger – larval stage of *Trombicula alfreddugesi* in the US and Trombicula autumnalis in Eurasia. The adult chigger is a free living mite in the family Trombiculidae and class Arachnida. The extreme itching that results in self inflicted trauma is caused by digestive enzymes injected by the chigger larva.
  - Ticks –
  - Mange Mites –
    - Skin scraping, deep dermal scraping using a scalpel blade or sharp knife.
  - Sucking Lice –
    - Skin scraping, superficial epidermal and hair scraping using as in shaving with a straight edge razor or sharp knife
  - Biting Lice –
  - Blood sucking flies belonging to the order Diptera.
    - Horse Fly and Deer Fly - Large blood sucking flies that belong to the family Tabanidae. They are carriers (vectors) for non-contagious viral diseases such as Equine Infectious Anemia virus
    - Biting midges, are small flies in the Family Ceratopogonidae. *Culicoides* sp. and other small biting midges or “no seeems” can act as vectors for the viruses that cause Blue Tongue, Epizootic Hemorrhagic Disease in ruminants and Vesicular Stomatitis in ruminants and horses. They can act as vector for protozoan parasites, such as *Leishmania* spp. Also. These small midges can cause allergic reactions in horses (and likely any species) to the saliva and mouth secretions deposited in the puncture wound when feeding on mammals. In horses the most commonly affected areas are the concave surface of the ear pinna and the lateral sides of the neck.
    - *Phlebotominae* – Sand flies, another family of “no seeems”
  - Mosquitoes are insects that are flies that constitute the family Culicidae.

- Ringworm
- Skin tumors and warts

Biopsy
Microbiology

- **Ophthalmologic System**
  Ophthalmoscope
  Special ophthalmologic exams, slit lamp, etc
  Auriculopalpebral nerve block
  Flourescien staining of cornea
  Microbiologic exam
Nasolacrimal catheterization & irrigation
Suture 3rd eyelid to upper lid for corneal ulcers

- **Neurologic System**
  - CSF Examination
  - Radiography
  - Neurologic response tests

- **Hemolymphatic and Immunological Systems**
  - Hematology
  - Clotting profile
  - Bone marrow biopsy
  - Serum chemistry
  - Lymph node biopsy, aspiration
  - Immune status tests
    - Combined immunodeficiency (CID) testing
    - Failure of passive transfer in foals and calves

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3. Gastro-Intestinal Tract, 03-12-2012

**The Equine GI Tract and Physiology**
- **Lips**
- **Teeth**
- **Esophagus**
- **Stomach**
  - Simple stomach
  - Cardiac region
  - Glandular fundus
  - Pyloric region
- **Small Intestines**
- **Ileocecal junction**
Cecum

Cecocolic junction

Colon

Ventral Colon
- Origin of the colons, starting at the cecocolic junction in the right posterior quadrant of the abdomen
- Analogous to the Ascending Colon
- Divided into three sections
- Right ventral colon
- Sternal flexure – common site of sand impaction
- Left ventral colon

Pelvic flexure
- Separates the Ventral Colon from the Dorsal Colon
- Easily palpated via the rectum
- Common site of fibrous impaction

Dorsal Colon
- Analogous to the Transverse Colon
- Divided into three sections
- Left dorsal colon
- Diaphragmatic flexure
- Right dorsal colon

Small Colon (also called the Descending Colon)

Rectum

Ruminant Stomach, GI Tract and Physiology

Four divisions of the ruminant stomach

Reticulum (1st compartment)
- Functions in concert with the rumen in fermentative digestion of fibrous feed components
- Sometimes referred to as the “first stomach”
- Heavy items usually end up in the reticulum due to its location ventral to the esophagus
  - Boluses, Magnets
  - Wire – the cause of “hardware disease”

Common Condition
- Hardware Disease
  - Wire may perforate the reticulum
  - Causes peritonitis
  - Can penetrate diaphragm and pericardial sac
  - Signs: severe pain, arched back, ruminal ileus

Rumen (2nd compartment)
- The largest part of the ruminant stomach
- Is actually a large fermentation vat
  - Cows do not masticate feed as they eat, swallowed whole
  - Un-masticated, unsaturated feed is lighter
  - Fresh feed lies on the surface of the rumen
  - Feed on the surface is regurgitated and masticated
  - Called “chewing the cud”
  - When cud is swallowed it is saturated, heavy and falls into the reticulum and lower layers of the ruminal contents

Common conditions
- Bloat
- Ruminal Acidosis

**Omasum (3rd compartment)**
Round basketball sized organ
Mucosal folds form interdigitating leaves
- Mucosal leaves fill entire lumen
- Very large mucosal surface area for absorption
Main action:
- Dehydration of rumen contents
- Absorption of volatile fatty acids (end products of fermentation)
Common condition
- Impaction
  - Undigestable materials such as baler twine can easily cause impaction and obstruction of the omasum

**Abomasum (4th compartment) The true stomach**
Acid and enzymatic digestion of ingesta
Anatomy is similar to the simple stomach of other species
In addition to digesting consumed feed:
- Bacteria and protozoa from rumen fermentation are digested, greatly increasing the protein and nutrient quality of the ingesta
- Ruminal microorganisms can convert ammonia, urea and other nitrogenous compounds into protein
Common condition
- Left displacement and Right displacement / torsion
  - Caused anorexia and/or GI ileus
  - Usually a secondary condition

**The Ruminant Hind Gut (Intestines)**
- Small Intestines
- Cecum
- Large (or Spiral) Colon
  - Centripetal Spiral Colon
  - Centrifugal Spiral Colon
- Descending Colon
- Rectum

**Equine Digestive Disturbances**
**Primary categories**
- Gastritis
  - Parasitic gastritis
    - Bot fly larva
    - Stomach worms
    - Both parasites perforate and cause lesions to gastric mucosa
  - Gastric ulcers
    - Primarily a disease of young horses
    - Decreased appetite and poor performance are main symptoms
    - Colic and other signs are rarely seen
    - Up to 50% of foals may be afflicted
    - Gastric perforation and hemorrhage are occasional life-threatening sequela
    - Helicobacter infection has not been proven but may be a factor
  - Proximal Enteritis
    - Extension of a small intestinal disease discusses below

**Enteritis (small intestinal)**
Small intestinal enteritis frequently causes diarrhea in neonatal foals.
Adults will seldom display diarrhea due to the fluid absorbing capacity of the large colon. Adults are more likely to show colic like signs as main sign of small intestinal enteritis.

**Bacterial enteritis**
- Gram-negative coliform bacteria, *E. coli* and *Salmonella* spp.
- Gram positive anaerobic bacteria, *Clostridium perfringens*

**Proximal enteritis**
- Enteritis of Duodenum and Jejunum
- Frequently extends proximally into the stomach
  - Ileus in the small intestine allows gas and ingesta to flow both proximally and distally
  - Marked fluid and gas accumulation distends intestines and stomach causing marked pain
- Usually severe colic symptoms
- Can mimic volvulus or intussusception of small intestine
- Often associated with presence of *Clostridium perfringens*

**Viral enteritis,**
- In foals and calves these viruses result in diarrhea
  - Rotavirus, most common virus
  - Coronavirus
  - Adenovirus
  - Parvovirus
- Viral enteritis causes atrophy (necrosis of enterocytes) of the intestinal villi, referred to as “Villous Atrophy”
  - Decreased intestinal surface area results in decreased fluid and nutrient absorption capacity
  - Malabsorption results in fluid and nutrient loosing diarrhea

All of the above small intestinal infectious processes may also cause disease of the colon

- **Colitis**
  - *Salmonella, E. coli,* see above
  - *Ehrlichia risticii (Potomac Horse Fever),* will be discussed later
- **Colitis X**
  - *Clostridium perfringens* and other clostridia
- **Parasitism** (large and small strongyles)

- **Malabsorption**
  - Parasitism, due to lesions in the intestinal epithelium
  - Viral infection, due to villous atrophy
  - Granulomatous enteritis
    - *Mycobacterium avium var. paratuberculosis*
    - *Other Mycobacteria*
  - Enteric lymphosarcoma, invading the intestinal mucosa
  - Complications secondary to intestinal resection

**GI disorders that are associated with colic**
Colic is really a symptom of disease and not a disease entity
Colic - Abdominal pain that is severe enough to elicit symptoms and signs
Many GI diseases can cause colic in horses, including disorders listed above.

- **Impaction / Constipation**
  - Fibrous Impaction
    - Secondary to initiating causes
      - Dehydration
        - Most common in the winter
        - Horses naturally do not drink as much in the winter
        - Water sources may freeze
Insufficient water supply for other reasons
- Coarse feeds
- Bad teeth and poor mastication
- Coarse dry forage
- Altered gut motility due to other digestive disease
  - Parasitism
  - Sand accumulation in colon
  - Gastric ulcers
- Most common locations
  - Pelvic flexure
  - Stomach, Secondary to ileus, foreign material, parasites
  - Ileum and Ileo-cecal orifice
  - Cecum, primary neurogenic problem
  - Small Colon impaction (constipation)
- Symptoms
  - Mild colic symptoms early
  - Depression, weakness, anorexia later
  - Scant mucoid feces
- Treatment
  - Rehydration therapy
    - Balanced IV fluids 10 to 25 liters initially
    - Oral fluids
  - Mineral oil or other intestinal lubricants
  - Bran in diet
  - Pain medication
- Sand Impaction
  - Horses eating short grasses on sandy soil
  - Eating hay off of the ground on sandy soil
  - Sand accumulates in ventral portions of the cecum and colon
  - If sand accumulates rapidly in the colon, partial or even full obstruction can occur
  - Symptoms
    - Mild and intermittent colic symptoms
    - Depression, weakness, anorexia if obstruction is complete
    - Scant mucoid feces
  - Treatment
    - Rehydration therapy is occasionally needed
    - Mineral oil or other intestinal lubricants
    - Bran in diet
    - Psyllium in diet
    - Pain medication
  - Prevention:
    - Do not allow animals to graze grass shorter than 3” on sandy soil
    - Never feed hay or grain on sandy ground, even in a feed tub or hay rack, horses love to spill their feed
    - Feed on a concrete pad, rubber mats, dry manure pack, anything other than sandy ground
    - Feed psyllium in grain mix if sand continues to be a problem
- Retained Meconium
  - An impaction of the fetal feces at the rectum
  - Occurs in about 5% of foals born
  - The first feces in a newborn foal is called meconium
  - Consists of semi-digested material ingested and produced by the GI tract during fetal life
    - Amniotic fluid and cells
    - Sloughed epithelial cells
    - Bile and pancreatic secretions
Mucous, etc.

- Foals typically do not defecate during fetal life
- Meconium fills the rectum and small colon
- Cause of meconium impaction:
  - Neurologic immaturity
  - Related to late initiation of suckling
- Signs:
  - Straining to defecate
  - Sometime in a suckling foal a scant diarrhea like fluid is expressed around the impaction and mis-diagnosed as foal scours
- Rare occurrence in calves
- First defecation should occur within 2 hours of birth usually after the first suckling
- Treatment simply consist of an enema if caught early
- Many horsemen administer an enema to every foal born prophylacticly (Fleet, 4.5oz enema)

- **Physical injury to bowels**
  Twisting or other anatomic malposition of the bowels will result in:
  - Obstruction of the bowel lumen
  - Vascular disruption of the bowel
    - Occlusion of the venous return,
    - Venous pooling and ischemia
    - Rarely, occlusion of the arterial supply
  Typical injuries include
  - Intestinal incarceration, strangulation
    - Pedunculated lipoma
    - Inguinal hernia
    - Umbilical hernia
  - Volvulus
    - A twist in a bowel around it's mesenteric suspension
  - Torsion
    - A longitudinal twist in a section of bowel
  - Intussusception
    - Telescoping of a section of bowel inside adjacent bowel

- **Spasmodic colic**
  Parasites
  Circulatory disturbances
  - Strongylus vulgaris causes Verminous Arteritis (VA)
    - Aneurisms
    - Abscesses
    - Thrombosis
  - VA & Thromboembolic ischemia of intestines
  Moldy feed occasionally

- **Tympany**
  Gas accumulation in a section of bowel
  Causes
  - Altered gut motility or ileus
  - Peritonitis, causing ileus
  - Obstruction
    - Volvulus/Torsion i.e. large colon
    - Intussusception, ileoceleal, jejunal
    - Strangulation, abdominal lipoma, gastroplenic ligament defects
  Most common locations
  - Cecum
- Large colon
- Stomach
- Small intestines

**Equine Colic Treatment**
Treatment of colic implies pain management
Beyond pain treatment, treat underlying cause
Prevent injury to horse and handler
Refer if indicated
Surgical management
Medical management
- Hand walking, prevent horse from rolling and injuring itself or others
- Fluid and electrolyte therapy
  - Intravenous
  - Per Os
- Intestinal lubricants
  - Mineral oil
  - D.S.S.
  - Carboxymethylcellulose
- Intestinal protectants
  - Activated charcoal
  - Bismuth subsalicylate
  - Kaolin / pectin
- Analgesics (can mask signs)
  - NSAIDS, best all around pain control with few side effect in colic cases. Added benefit of reversal of endotoxin effects.
    - Banamine
    - Ketofen
  - Narcotic agonists
    - Pentazocine, (Talwin), (never with impaction)
    - Butorphanol, (Torbugesic), (never with impaction)
    - Alpha-2 adrenoreceptor agonists
    - Xylazine, (Rompun), (never with impaction)
    - Detomidine, (Dormosedan), (never with impaction)
  - Tranquilizer
    - Acepromazine as a vasodilator

**Therapy geared toward specific problems**
Antiserum for bacterial infection
- Endotoxin antiserum
- Enterotoxin antiserum
Antimicrobial treatments
- Antibiotics for specific infections
- Metronidazole (Flagyl) for gram positive anaerobic bacterial enterocolitis
Enema for meconium retention
Antacids for gastric ulcers
- Sucralfate coats stomach ling (specifically ulcerated areas) protecting against the activity of acid and enzymes
- H2 blockers to suppress acid production
Plasma or blood transfusion if patient is hypoproteinemic or anemic from parasitism, Protein loosing enteropathy, malabsorption or chronic blood loss
Plasma to treat low blood immunoglobulin levels
Gastric decompression and emptying
- Stomach tube

**Equine Colic Preventative measures**
Routine dental care
Deworming
Good feeding and watering methods
- Feed off the ground
- Use only good quality forages
- Make no sudden feed changes
- Adhere to regular feeding schedule
If sand is a problem
- Psyllium or bran in the feed
Regular exercise
Minimize stress
Endotoxin vaccine

Diarrheal Diseases

- **Neonatal Diarrhea (Scours) calves and foals**
Factors which contribute neonatal diarrhea
- Pathogen burden in environment
- Colostrum consumption (quantity)
- Colostrum quality
- Maternal immunity to specific pathogens
- Low blood immunoglobulin IgG levels due to “Failure of Passive Transfer” (FPT)
  - Low blood IgG level result from inadequate consumption or poor quality of colostrum
  - Several tests are available to assay IgG levels:
    - Radial immunodiffusion (RAD)
    - Zinc sulfate turbidity
    - CITE IgG (probably the best quick screening test as it can be done on the farm quickly)
    - A crude estimate of antibody transfer can be made by simply doing a total plasma protein with a refractometer
      - <=5gm/dl is indicative of FPT, while >=7gm/dl is normal
  - Treatment of FPT include administration of donor colostrum to neonates less than 24 hours of age and administration of blood plasma (1 to 3 liters) to older neonates
Must establish a diagnosis if possible
- Enables prevention against specific pathogens
- Determine antibiotic sensitivity
Methods of making a diagnosis
- Fecal or rectal cultures, sensitivity
- Fecal virology, usually electron microscopy (EM)
- Necropsy
  - Allows sampling for virology and bacteriology
  - Gross and histopathology findings are helpful
  - Sampling liver for evidence of malnutrition
- Fecal floatation – coccidiosis
- Microbiology – careful interpretation is essential
  - Culture and sensitivity testing
Etiology - variable
Bacterial
- E. coli is the most common pathogen
  - E. coli and other coliform bacteria must come in close contact or attach to intestinal epithelium. This allows the endotoxin on the surface of the bacterium to affect the enterocytes.
  - E. coli with K99 pili antigen are particularly pathogenic
The K99 pili antigen allows the bacterium to tightly attach to enterocytes, facilitating the effects of the bacterial endotoxins on enterocytes

Endotoxin effects include hypersecretion of fluid and electrolytes from enterocytes, cell membrane disruption, cell death

Endotoxins may increase capillary and small vessel permeability, causing fluid loss

Invasion of mucosa, submucosa and vasculature can result in systemic septicemia and endotoxemia

Hypersecretion of fluid and capillary leakage results in diarrhea in foals

Occurs typically in neonates under 1 week of age

Associated with “Failure of passive transfer of immunity”

Endotoxemia

Septicemia

- Salmonella spp. and other coliform bacteria behave similarly to E. coli
  - Salmonella is a potential zoonotic disease
  - Clinically similar to E. coli in most respects
  - May cause a more bloody diarrhea than E. coli

- Clostridium perfringens
  - Causes enterocolitis (small and large intestines)
  - Can occur at any time during lactation but usually occurs between 1 and 3 months of age (peak lactation)
  - Occurs in calves nursing cows with excessive milk
  - Can occur in older animals also, associated with “overeating”, sheep and cattle in feedlots vulnerable
  - Bacteria proliferate in a carbohydrate rich environment
  - Other bacteria and viruses predispose
  - Cl. perfringens may be normal inhabitant of gut, but proliferates and causes disease when there is excess consumption of starch and sugars causing excessive volatile fatty acid accumulation in the gut lumen and decreased oxygen content of ingesta. Other GI disorders and antibiotic therapy may also predispose to Cl. perfringens overgrowth by upsetting normal gut flora.
  - Enterotoxin (actually an exotoxin) are released from bacteria causing necrosis of intestinal epithelium, vascular damage, intestinal hemorrhage, ileus and other toxic effects.
  - Gas accumulation in bowel from bacterial fermentation
  - Enterotoxin in the peripheral circulation cause circulatory collapse, shock and death, referred to as Enterotoxemia
  - The neonate may die before diarrhea is even evident
  - Treatment difficult as cases are frequently peracute
  - Exotoxin release is hastened by the use of oral bactericidal antibiotics.
    Antibiotic treatment may be of limited value in treating enterocolitis. Metronidazole (Flagyl) may be of benefit in treatment. Also probiotic therapy and supportive therapy are beneficial.
  - Prevention includes vaccinating the dam and neonates with Cl. perfringens C&D toxoid and administration of antitoxin to vulnerable animals

Viruses

- Infect enterocytes
- Induce villus tip atrophy due to enterocytes loss (death of cell)
- Villus atrophy causes a decrease in intestinal surface area and marked decrease in fluid and nutrient absorptive capacity (malabsorption)
- Malabsorption results in fluid and nutrient loosing diarrhea

- Rotavirus
  - Most widespread virus that causes diarrhea
  - Disease may be mild to severe
  - Epidemics of rotavirus are common

- Coronavirus,
More serious damage to intestines than rotavirus
Less common
Can affect adults also

- BVD
  - Typically calves over 30 days of age
  - Congenitally infected calves may not show diarrhea but die
- Diagnosis of these viruses usually requires electron microscopy
  - It is essential to obtain fecal sample for EM from a neonate that has just started scouring within the last 12 to 24 hours.

Misc. protozoan organisms
- Cryptosporidia parvum
  - A protozoan parasite similar to coccidiosis
  - Affects calves over 2 weeks of age
  - Causes malabsorptive diarrhea
- Zoonotic risk
- Giardia - Calves over 2 weeks of age

Pathogenesis
- Dehydration
  - If severe enough, dehydration results in
  - Hypovolemia
  - Azotemia
- Acidosis
  - Primarily lactic acidosis secondary to hypovolemia
  - Metabolic acidosis possible in moribund neonate
- Hypoglycemia
- Intestinal mucosa damage and blood loss
- Septicemia
- Endotoxin shock secondary to septicemia
- Enterotoxin from Cl. perfringens causes intestinal necrosis and circulatory collapse

Assessment of the neonate
- Volume and consistency of diarrheic stool
  - The more watery the stool is, the faster the patient will deteriorate
- Dehydration
  - Skin elasticity test for dehydration
  - Eye ball position in orbit (sunken?)
- Evaluation of base deficit (acidosis)
  - Blood gas analysis – or –
  - Physical evaluation
    - Loss of suckle reflex but ambulatory (mild acidosis)
    - Sternal recumbency and unwilling to rise (moderate acidosis)
    - Lateral recumbency and inability to rise (serious acidosis)
- Circulation – Shock and Hypothermia
  - Subjective evaluation of oral temperature
    - Oral temperature inversely proportional to degree of shock
    - Cold mouth generally means a calf has lost ability to maintain body temperature (developing shock and probably acidosis), especially serious when temperature is below freezing
- Evaluation of blood glucose for hypoglycemia / hyperglycemia

Treatment
- Oral electrolytes
  - Non-alkalinizing oral electrolytes in early treatment
    - Fluids that do not contain sodium bicarbonate
    - Sodium bicarbonate will neutralize gastric acid and interfere with milk curd formation (necessary for normal milk digestion)
For calves with diarrhea but minimal dehydration and no signs of acidosis
OK to leave calf or foal on dam
- Sodium bicarbonate (alkalinizing) oral electrolyte solutions for more severely affected calves
  - 3-5% dehydration or mildly acidotic
  - Best to not allow calves to nurse while on these solutions
- IV electrolytes
  - 0.9% NaCl
  - 1.3% Na Bicarbonate to reverse acidosis
  - Avoid lactate in oral or IV solutions for calves with lactic acidosis
  - Don't use lactated ringers solution
- IV or oral Dextrose to treat hypoglycemia
- Other treatments
  - Oral colostral supplements
  - Antitoxins (Clostridial)
  - Kao Pectate, activated charcoal and other absorbptive / protective compounds are beneficial
  - Antibiotics
  - NSAID to treat endotoxemia

Prevention
- Reducing exposure to pathogens
  - Clean calving facilities
- Increasing herd resistance to disease
  - Vaccination of dam with Rotavirus, Coronavirus, E. coli K99 antigen, and Cl. Perfringens type C, vaccine - ScourGaurd 3K C (Pfizer)
    - Vaccinate dam with 2 doses with second dose 2 to 4 weeks pre-calving, annual booster 2 to 4 weeks pre-calving
    - Improves colostrum antibodies against the following:
      - Rotavirus and Coronavirus
      - E. Coli K99 antigen
      - Cl. perfringens bacteria and toxin
  - Assure adequate colostrum consumption
  - Reduce stress; Provide bedding for newborn calves. Housing in inclement weather.
  - Vaccinations and treatments of the neonate at birth
    - Use “Calf Guard” oral MLV Rotavirus and Coronavirus vaccine within 2 hours of birth
    - Use E. coli K99 antibody preparations if necessary
    - Vaccinate for Cl. perfringens type C&D
    - Use Cl. perfringens antitoxin if necessary
    - Use preventative antibiotics only as a last resort

**Potomac Horse Fever (Equine monocytic ehrlichiosis, Equine Ehrlichia Colitis)**

**Etiology**
- The pathogen that causes PHF was formerly known as Ehrlichia risticii
- The revised name is Neorickettsia risticii

**Transmission**
- Transmission studies using N risticii infected caddis flies have reproduced the clinical disease. The most likely route of exposure is the consumption, while grazing, of aquatic insects or snails that are infested with a metacercarial (larval) stage of a trematode (fluke) that is, in turn, infected with the N risticii bacterium.
- Sporadic incidence and the fact that infected clinical patients do not transmit the disease to stable-mates leads one to conclude that the infected fluke larva is an obligate intermediate host. More research is needed to really make that conclusion though. Other methods of transmission are possible.

**Pathogenesis:**
Infects monocytes, macrophages & glandular epithelium
· Obligate intracellular bacterium
· Acute colitis, typhlitis
· Inflammation, fluid loss into the lumen of the intestine and thus diarrhea

Symptoms
· Profuse watery diarrhea
· Depression
· Anorexia
· Fever
· Ileus
· Hypovolemic shock
· Colic and laminitis are common
· Signs are clinically difficult to distinguish from Salmonella

Diagnosis
· IFA
· Response to therapy can be diagnostic

Treatment
· Supportive care
· IV fluids
· Oral fluids
· Antibiotics, Oxytetracycline

Prevention
· Vaccinate
· Reduce tick load
· Regular parasite control

Equine Parasites
· **Large strongyles**
  Most harmful internal parasite of horses

Primary species
· S. vulgaris
· S. edentatus
· S. equinus

Direct life cycle
· Eggs hatch and free living larval stages live on pasture
· Require moisture and moderate temperatures to develop
· Third stage infective larva are picked up by foraging animals

6-7 month pre-patent period (1 cycle per year)

Larvae in abdominal tissue, organs and vasculature
· Causes of “Verminous Arteritis”
· Anterior mesenteric artery and other arteries
· Weakened arteries – aneurysms
· Secondary embolisms

Adults in cecum and large colon
· Blood sucking
· Chronic damage to mucosa can cause ulcerations

Signs;
· The majority of symptoms are a result of S. spp. adult infestation
· Mucosal damage results in:
  ñ Fluctuating fever
  ñ Leukocytosis
  ñ Colitis as a result of heavy adult infestation
· Blood consumption by adult parasites causes
  ñ Anemia
  ñ Hypoproteinemia
Can cause swelling of the legs and distended abdomen (ascites).

- Depression
- Inappetance
- Unthriftness

- Colic

  - Larval infestation can damage major arteries leading to colic in later life due to Verminous Arteritis:
    - Mesenteric arterial obstruction
    - Intestinal ischemia

Decreasing incidence due to use and efficacy of most modern paste wormers

- **Small strongyles**

  In the large colon and cecum:
  Cyathostomes is the family name
  Triodontophorus, Poteriostomum, Cyathostomum, and other species

  Direct Life cycle
  - Eggs hatch and free living larval stages live on pasture
  - Require moisture and moderate temperatures to develop
  - Third stage infective larva are picked up by foraging animals

  2 or 3 month to 2 or more years pre-patent period
  - Depending on length of time larva remain encysted in colon

  Signs
  - Dark feces
  - Anemia
  - Anorexia
  - Colitis
    - Diarrhea
    - Foul odor
    - Irritation from infective 3rd stage larval invasion
    - 4th stage larva encysted in the cecum and colon
    - Further damage from 4th stage larval emergence

  Incidence of small strongyles seems to be increasing
  - In spite of the widespread use of modern paste wormers
  - Poor efficacy of most wormers, used at routine dose, on larval stages
  - Loss of competition from Large Strongyles?

  Treatment of encysted larva requires multi-day therapy with fenbendazole or other broad spectrum wormers

- **Ascarids**

  Parascaris equorum
  Large roundworms
  Most common in young horses

  Pathology:
  - Parasite inhabits the small intestine (the most nutrient rich portion of the bowels)
  - Heavy burden of adult worms results in loss of large amount of high quality small intestinal nutrients to the parasites. This result in a malnutrition state
  - Parasites causes irritation of intestinal wall
  - Dead worms being expelled after de-worming can cause obstruction of the small intestine at the ileo-cecal valve
    - Usually when infestation is severe in foals
    - May be best, if severe infestation is suspected, to use ½ dose of a moderately effective anthelmintic such as Ivermectin and then repeat treatment with a full dose 2 weeks later

  Direct life cycle
  - Embryonated eggs are infective
  - Infection can be transmitted in a dry-lot or stall setting
• Larva migrate in liver and lungs of foals
• Larva enter the respiratory tree, are coughed up and then swallowed. They enter the small intestine and become adults

Signs
• Diarrhea
• Rough hair coat
• “Pot belly”
• Coughing from final stages of “Visceral Larval Migrans”
  ý The larval migration can contribute to respiratory infections
  ý “Summer Cough”

• **Pinworm**
  • **Oxyuris equi**
    In large and small colons

Signs
• Restlessness
• Irregular feeding
• Loss of condition
• Tail rubbing
• Dull hair coat

Adults feed on intestinal contents
Adults lay eggs outside of the rectum on the perineal area
• Egg bundles cause pruritis of the anus
• Like ascarids the eggs are infective
• Transmission can occur in a dry setting

Larvae feed on mucosa

• **Threadworm**
  • **Strongyloides westeri**
    Inhabit the small intestine of young horses

Adult horses may carry somatic larva yet never develop a patent infection, due to immunity directed at the adult parasite
Young horses (under one year of age) carry the patent infection and are responsible for maintenance of the infestation in a herd
Pregnant mares at parturition may pass somatic larva through the milk to new-born suckling foals, thus passing on the infection
It is almost as if the mares act as an intermediate host for this parasite
Foals will rid themselves of the adult infestation by one year of age

Signs
• Loss of weight
• Diarrhea in suckling foals
• Cause irritation of intestinal mucosa
• Enteritis

Most modern anthelmintics, particularly ivermectin are effective in treating adult parasite infestations. Treatment of mares with ivermectin at parturition may be effective in preventing transmission to foals.

• **Stomach worm**
  • **Habronema spp. and Draschia megastomias**
    Developing larva and adult parasites inhabit the stomach
Habronema larva may also infest skin lesions
Infested skin lesions are referred to as “Summer Sores”
Also called “Cutaneous Habronemiasis”

Indirect life cycle
• House flys and Stable Flies are the intermediate hosts
• Flys ingest the stomach worm eggs from feces
• Larval development occurs in the fly or in maggots
  ᵃ Flys deposit infective larva around the face
  ᵃ Larva migrate into the mouth and then to the stomach
  ᵃ Larva in the stomach develop into adults and re-establish patent infections
  ᵃ Flys may also deposit infective larva into abrasions or cuts
  ᵃ Larva in dermal lesions cause Summer Sores
  ᵃ Larva in a Summer Sore do not mature into adults
• Transmission can occur in dry-lots or stable settings

Signs
• GI
  ᵃ Gastritis and mucosal thickening are the result of Habronema activity in the stomach
  ᵃ Tumor like, granulomatous lesions, in the stomach wall, are the result of adult Draschia parasites activity. The adults live inside these lesions.
• Dermatologic
  ᵃ Granulomatous, non-healing skin ulcers

Treatment
• All modern anthelmintics are effective against GI infestation.
• Summer sores respond best to local debridement and topical organophosphate treatment.
• Fly control is also important for control of stomach worm.

• Tapeworm
• *Anoplocephala spp.*

  In small intestine and cecum

  Life cycle:
  • Typical tapeworm indirect life cycle, except for an insect intermediate host
  • A free living oribatid mite acts as the intermediate host
  • Mites containing infective larval cysts are consumed while grazing
  • Annual life cycle

Signs
• Unthriftiness
• Acute obstructive colic
• Causes obstruction and ulceration of ileocecal valve and enteritis

Treatment
• Best to treat in the spring so as not to infect pastures, retreat in the fall
• Fenbendazole or pyrantel pamoate at 2x dose is effective
• Niclosamide and praziquantel are also effective
  ᵃ Not approved for use in horses
  ᵃ Expensive
  ᵃ Narrow spectrum, might as well use a broad spectrum wormer

• Bots
• *Larva of the Gasterophilus spp. Fly*

  Life cycle
  • The fly lays eggs in the late summer
    ᵃ Attached to hair on legs - *G. intestinalis*
    ᵃ Attached to hair on jaw - *G. nasalis*
    ᵃ Hatch in response to warm moisture (licking)
    ᵃ Hatched larva migrate in gums and esophagus
  • Larva spend the winter in the stomach of horses
    ᵃ Larva erupt and attach to stomach mucosa
    ᵃ Larva release from gastric mucosa in late spring
  • Larva passed in feces to pupate on the ground and form adults
• Adults are free ranging appear to cover large area

Signs
• Usually sub-clinical
• Digestive upset
• Oral irritation, periodontal ulcers
• Cause inflammation, perforation of stomach

Treatment
• Tube or paste worming with boticide
  • Oral organophosphate boticides
    Trichlorfon
    Dichlorvos
  • Ivermectin
• Best to treat after hard freeze kills adult flies
• Removal of bot eggs from legs
  • Warm water wash
  • Curry combs

• Equine Parasite Control Beyond Anthelmintics

Manure management procedures
• Don’t spread manure on pastures where horses are grazing
• Compost manure to kill parasite eggs
• Prevent contamination of food and water with manure
• Remove manure from stalls daily and strip all bedding frequently
• Clean and disinfect stall floors, walls, feeders and waterers as needed.

Other management procedures
• Avoid low wet pastures if possible
• Use rotational grazing if possible
• Harrow pastures
  • Immediately after grazing, while grass is shortest
  • Breaks up manure and exposes larva to sun shine

• Strategic Anthelmintic Use
• Deworm all horses at the same time
• Worm horses prior to moving them to a clean pasture
• Separate older horses from weaned foals
• Isolate all new horses
  • De-Worm immediately upon arrival
  • De-worm a second time with a larvacidal regimen 2 to 3 weeks after the first de-worming and before turn-out with the resident herd
  • Do not intermingle them with resident herd until all de-worming is complete

4. Conformation and Lameness, 03-12-12
Conformation: the proportional and symmetrical shape of the body

• Conformation defects
  Many are merely blemishes or unsightly
  May or may not predispose to lameness
  Defects in the legs are most common
  • Greatest likelihood of causing unsoundness
  Defects in spinal conformation
  • Less common but occasionally detrimental
  • Excessive Lordosis
  • Kyphosis
  • Scoliosis
  • Individual vertebra deformities
Conformation Front Limbs

- **Normal forelimbs**
  Center of hoof lies directly below a vertical line from point of shoulder

Conformation Defects Front Limbs

- **Base Narrow**
  Center of hoof lies inside a vertical line from point of shoulder

- **Base Wide**
  Center of hoof lies outside a vertical line from point of shoulder

- **Toe In**
  Pigeon toed, toe points toward midline
  Toe in defects may result from un-corrected varus defect as a foal
  Base Wide Toe In
  Base Narrow Toe In

- **Toe Out**
  Splay foot, toe points away from midline
  Toe out defects may result from un-corrected valgus defect as a foal
  Base Wide Toe Out
  Base Narrow Toe Out, this conformation is most likely to result in interference between contralateral limbs

Conformation - Foot Path

- **Straight Toe**
  Straight foot path

- **Toe out**
  Winging
  Foot swings toward midline
  Causes interference between opposite legs

- **Toe In**
  Paddling
  Foot swings away from midline

Conformation, Rear Limbs

- **Normal Rear limb (lateral view)**
  Posterior side of cannon is directly below posterior point of tuber ischii (butt)
  Proper angulation in tibia, hock, fetlock, pastern and hoof

- **Normal Rear limb (AP view)**
  Center of cannon and hoof directly below posterior point of ischium

Conformation, Rear Limb Defects

- **Standing Under Behind or Camped Under (lateral view)**
  Posterior side of cannon is anterior to vertical line from posterior point of ischium
  Can be the natural conformation or
  Can be induced by pain in front feet
  - Causes horse to assume this conformation
  - Laminitis of front feet is most common condition

- **Camped Behind (lateral view)**
Posterior side of cannon is posterior to vertical line from posterior point of ischium (butt)

- **Base Narrow (A-P view)**
  Center of cannon and hoof inside of vertical line from posterior point of ischium (butt)

- **Cow Hocked (A-P view)**
  Medial deviation of hock joint and lateral deviation of the metatarsal so that the hock lies inside of vertical line from posterior point of ischium, while hoof lies on or outside of the vertical line
  May result from valgus deformity in hock as a foal

- **Post Legged (lateral view)**
  Decreased angle (from vertical) of the tibia (gaskin), such that the stifle and hock are too straight and upright.
  Increased concussive trauma to hock and other structures
  Associated with too straight or upright pastern

- **Sickle Hock (lateral view)**
  Increased angle in the hock
  Increased tension and strain on Achilles tendon, posterior flexor tendons and ligaments of the hock

**Structure and Conformation of the hoof**

- **Ventral structures of the normal hoof**
  Heel
  Bulbs
  Quarter
  Toe
  Frog
  Collateral Sulci
  Central sulcus
  Sole
  Bar
  Hoof wall
  Angle of wall
  White line

- **Hoof and pastern angle**
  A - normal, 47 degrees front, 52 degrees rear (+/- 2°)
  B - too small an angle (from Horizontal), shallow slope
  C - Too large an angle from horizontal, steep slope

  Relationship to soundness
  - Too small an angle (from Horizontal), shallow slope
    - Places excess strain on suspensory ligaments
    - Strain on flexor tendons (esp. superficial flexor tendon)
  - Too large an angle from horizontal, steep slope
    - Increased concussive force to joints in the lower limb
    - Increased incidence of traumatic osteoarthritis in the joints of the lower leg (i.e. Ringbone, Osselets, Etc.)

**Correctable angular and flexor limb defect seen initially in foals**

- **Calf- Kneed**
  Posterior deviation at carpus
  Due to carpal joint laxity
• **Buck-kneed and contracted tendons**
  Anterior flexor deviation at carpus
  Due to tension in flexor tendons and suspensory ligaments
  Sometimes in large fetuses the long bones grow faster than the tendons causing the relative “contraction” of the tendons.
  It is also thought to sometimes be related to uterine positioning.

**Medial and lateral angular limb deformity (front or rear legs)**
• **Valgus Angular limb deformity (front or rear legs)**
  Knock kneed or cow hocked defect
  May appear as a “medial deviation”, BUT in fact it is actually a lateral deviation of the leg distal to the area of the angular defect. The angular defect usually originates in the carpus or tarsus areas but can also occur at the fetlock or any other leg area.
  Frequently associated with lateral toe rotation (toe out)
  Correctable with restricted exercise, splints or surgery

• **Varus angular limb deformity (front or rear legs)**
  Bow-legged defect
  May appear as a “lateral deviation”, BUT in fact it is actually a medial deviation of the leg distal to the area of the angular defect. Once again the angular defect usually originates at the carpus or tarsus but can occur at any other leg area.
  Frequently associated with medial toe rotation (toe in)

• **Common causes of valgus or varus deformities include;**
  Incomplete ossification in a joint causing joint laxity
  Abnormal growth at a long bone growth plate

• **These defects are often correctable with:**
  Restricted exercise, for joint laxity and contracted tendons
  Splints, for joint laxity and contracted tendons

• **Surgical correction of angular limb deformities**
  Useful for deformities that originate in a growth plate or where conservative treatment has failed
  • Temporary growth plate stapling or bridging on the convex side; suppresses growth on the stapled side allowing the other side to “catch up” in length
  • Periosteal stripping at the growth plate on the concave side; stimulates new bone growth and lengthening

**Lameness**
**Definitions:**
• **Lameness:**
  A departure from normal stance or gait. A structural or functional disorder in one or more limbs. Manifested in the standing position or during progression.

• **Sound:**
  Exhibiting normal health; free of injury or disease; having the capacity to perform the function intended for that animal.

• **Unsound:**
  Not sound; not healthy; characterized by injury or disease that interferes with normal, desirable function of that animal.

• **Blemish:**
  An injury affecting appearance but not function

• **Peracute:**
  Very rapid onset
• **Acute:**
  Recent, sudden onset

• **Subacute:**
  Active or progressive disease process

• **Chronic:**
  Long duration, lingering

• **Convalescent:**
  Recovering

• **Sprain:**
  Trauma to a joint
  Injury to the joint capsule, ligaments and associated soft tissues.
  Pain, swelling and palpable heat in area

• **Strain:**
  Stretching injury to tendon or muscular
  Overstretching of a muscle or tendon
  Due to excessive tension or over-use.

• **Granulation Tissue:**
  Tissue formed in repair of soft tissue wounds
  Fibrous connective tissue
  Vascular tissue

• **Proud Flesh:**
  Excessive proliferation of granulation tissue when wounds heal via second intention
  Common on lower limbs of horses, where the skin is tight and tension stretches the skin, re-opening granulating wounds
  Treatment consists of surgical removal of excess granulation tissue and immobilization of the skin or even the limb
  When proud flesh will not heal in spite of treatment it may be the result of Habronema spp infestation (summer sore)

• **Synovial Fluid**
  Serum transudate with decreased serum proteins
  Lubricates and nourishes cartilaginous surfaces
  Proteins are primarily glycoprotein polymers
  - Hyaluronin, Lubricin and others
  - These act as lubricants and cushion the articular surfaces
  Normal synovial fluid analysis
  - Protein levels <2.0 g/dl (plasma protein is 5 to 7 g/dl)
  - Nucleated cells <300 cells / µl
  - High viscosity - a drop will form a 3 to 5 cm string
  - Mucin clot test positive
    - A tight mucous clot will form when acetic acid is added
    - Vinegar
  - Culture and sensitivity

• **AAEP System of Grading Lameness**
  Grade 0: Lameness not perceptible
  Grade 1: Lameness difficult to observe; not consistently apparent regardless of circumstances.
  Grade 2: Lameness is difficult to observe at a walk or while trotting in a straight line, consistently apparent under certain circumstances.
  Grade 3: Lameness is consistently observable at a trot under all circumstances.
  Grade 4: Lameness is obvious; marked head nodding, hitching or shortened stride.
  Grade 5: Minimal weight bearing in motion and/or at rest, inability to move, “three-legged lame”. This type of lameness is often associated with fractures, subsolar abscesses, severe tendonitis, and septic arthritis.

• **A locomotion score in dairy cattle, is very similar to an equine lameness grade.**
1. Normal: The cow walks and stands with a flat back. She walks normally.
2. Slightly abnormal gait: The cow stands with a flat back but arches her back while walking. She walks normally.
3. Moderately lame: The cow stands with an arched back and walks with an arched back. She short-steps while walking.
4. Lame: The cow stands with an arched back and walks with an arched back and walks with decreased weight bearing on one limb (limps).
5. Severely lame: The cow stands and walks with an arched back and refuses to bear weight on a limb.

Causes of lameness:
- **Trauma**
  - Fractures
  - Contusion
  - Lacerations with injury to joints, ligaments and tendons
- **Congenital or acquired neonatal anomalies**
  - Hoof and bone deformities
    - Club foot, congenital or acquired
    - Angular limb deformities, see above
  - Arthrogryposis
    - Fetal paresis/paralysis results in faulty development and of joints in one or more legs
    - Genetic, toxic plants, idiopathic causes
  - Spinal deformities
    - Cervical Vertebral Malformations (CVM or Wobbler Syndrome)
      - Cervical Vertebral Malformation (CVM) result in ataxia and is referred to Wobbler Syndrome
      - Subluxation and vertebral canal stenosis result in neurologic deficit
    - Spinal Bifida and deformed sacral-pelvic s, congenital defect seen in cattle
    - Lordosis, Kyphosis, Scoliosis
- **Poor conformation**
  - Concussive joint injury (straight legged conformation)
  - Tendonitis due hyperextension (low slope on pastern)
  - Repetitive trauma due to interference
- **Circulatory disorders**
  - Aortoiliac Thrombosis (saddle thrombus)
    - Thrombosis at the bifurcation (really a quadrifurcation) of the aorta to the 4 iliac arteries
    - Possible causes include degenerative arterial disease and parasitism
    - Signs relate to decreased blood flow; primarily weakness and ataxia in the hind legs when worked
  - Exertional rhabdomyolysis (exertional myopathy)
    - Myositis and myolysis as a result of exertion after a period of several days rest
    - Also called “Monday morning disease”, “Tying Up”
    - Severe pain, sweating, stilted gait or recumbency
    - Musculature of back, hind quarters, shoulders
    - Cause unclear;
      - Aberrant glycogen storage & use
Excess glycogen storage allows prolonged anaerobic metabolism of muscle cells causing local lactic acidosis, occurring if horse is not properly “warmed-up”

Acidotic condition causes arteri

- Circulatory dysfunction in muscles
- Electrolyte imbalance
- Genetics

**Diagnosis**
- Clinical signs;
  - Stiff stilted gait
  - Reluctance to move
  - Recumbency
- Elevated CPK (>1000 IU/L), LDH and AST
- Myoglobinuria

**Treatment**
- Tranquilization (Acepromazine is a mild vaso-dilator

Navicular disease
- Degenerative disease of the distal phalangeal sesamoid bone (navicular bone)
- Navicular bone lies posterior to the P2 – P3 joint (coffin joint)
- Frequently bilateral front limb lameness
- Multiple syndromes not a single entity
- Erosion of articular cartilage if frequently present (DJD)
- Osteopathy of sesamoid
  - Can be due to thrombosis of veins draining the bone
  - Ischemic necrosis of bone

**Diagnosis,**
- Hoof tester
- AP & DV radiographs (pack hoof or water bath)
- Digital nerve block

**Treatment**
- NSAID
- Corrective shoeing
  - Raise heal 2 to 4 degrees
  - Elevated heal on shoe with rolled or rocker toe
- Anticoagulants (warfarin)
- Beta adrenergic vasodilator Isoxuprine
- Digital neurectomy for chronic cases

Laminitis, see below under hoof disorders

**Metabolic disorders**
Hypocalcemia, tetany and/or muscle weakness

Hyperkalemic Periodic Paresis, in Quarter Horses
- Descendants of a QH stallion named “Impressive”
- An autosomal dominant gene results in an inability to control blood K level.
- When blood K is too elevated, it causes Persistent depolarization of muscle cells, resulting in muscle fasciculations at first followed by profound muscle weakness (paresis)
- Treatment; IV Dextrose and Na Bicarbonate or IV Calcium

**Nutritional deficiencies leading to osteodystropies**
Nutritional secondary hyperparathyroidism (Low Ca:P ratio)
• Animals kept on a high phosphorous and deficient calcium diet
• Ca depletion of bone in order to maintain normal blood Ca level
• Demineralization of bone results in bone thickening, pathologic fractures, pain and various lamenesses

Osteochondrosis
• An important problem in all domestic species
• Affect juvenile animals that are genetically capable of rapid growth and large mature height and weight. Affect animals on a high plain of nutrition.
• Disturbance of cellular differentiation in growth cartilage associated with bone development
• Decreased ossification at the osteo-condral interface
• Disturbance in vascularization of new bone
• Causes are complex and still unclear
  ų Mineral deficiency or imbalance in mineral nutrition involving: Ca, Zn, Cu, Mn, and CA:P ratio (<1.5:1)
  ų All meat diet in carnivores
  ų Feeding a high energy, concentrate (grain) ration which is high in phosphorous and deficient in calcium and/or trace minerals
• 3 syndromes:
  ų Osteochondritis Dessicans
    Disturbed ossification and formation of subchondral bone
    Detachment of surface cartilage from subchondral bone
    Exposure of subchondral bone
    Detached cartilage and bone fragment in joint causing further damage
    May resolve itself, require surgery to remove joint mouse, or lead to degenerative joint disease
  ų Subchondral bone cysts
    Decreased ossification of subchondral bone
    Etiology similar to OCD but without detachment of fragments
  ų Physitis or epiphysitis
    Delayed ossification of bone at growth plate of long bones
    Marked widening of the physeal cartilage
    Thickening of the long bone near the physis to compensate for weakness in the area
    Gross appearance of a thickened joint and adjacent area

• **Joint injury and disease**
  Luxations and sub-luxations
  • Causes
    ų Trauma
    ų Tearing a ligament
    ų Avulsion fractures at attachment of ligaments
    ų Advanced DJD with weakening of periarticular structures
  • Diagnosis:
    ų Obvious deformity of joint, marked lameness (Luxation)
    ų Palpation of laxity in joint, variable lameness (sub-luxation)
    ų Radiography
    ų Blood in synovial fluid analysis
  • Treatment
    ų Casting, surgery, etc

Degenerative Joint Disease, arthritis
• DJD is a common abbreviation or acronym
• Causes
  ų Chronic, repetitive injury to a joint, from concussive forces, interference,
Intra-articular fractures
Ligament damage, sub-luxation, etc.

- Degeneration of joint result in:
  - Thinning of articular cartilage
  - Erosion of articular cartilage
  - Exposure of subchondral bone
  - Synovial & periarticular inflammation
  - Osteophyte formation; at margins of the joint capsule initially and later the osteophytes may invade the joint

- Some common examples of DJD include:
  - **Osselets**: Traumatic arthritis of the fetlock joint
    - Common in young horses starting training
    - Can result from hyperextension of the fetlock joint with bruising trauma or fracture of the extensor process of P1
    - Can also be a result of concussive trauma from too steep a slope in the hoof and pastern
  - **Ringbone**: DJD of interphalangeal joints
    - Exostosis or new bone growth on P1, P2 or P3
    - High ringbone, DJD of P1 - P2 (pastern) joint
    - Low ringbone, DJD of P2 – P3 (coffin) joint
    - Usually is the result of concussive trauma; from riding too hard (trotting or loping on paved surfaces and perhaps too steep a slope in the hoof and pastern
  - **Bone Spavin**: DJD of intertarsal and tarsal metatarsal joints (hock)
    - Can result from bad conformation of the hock joint such as sickle hock or post legged.
    - Can also be a result of concussive trauma from too steep a slope in the hoof and pastern

Septic Synovitis and Arthritis

- Causes
  - Navel ill
    - Septic arthritis secondary to neonatal naval infection
    - Septic arthritis due to navel infection is sometimes called “joint ill”.
    - Common bacteria include E. coli and other gram negative coliform bacteria
    - Pasteurella spp.
    - Gram positive cocci
  - Trauma from laceration or puncture injury with introduction of infection
  - Iatrogenic from intra-articular procedures and injection
  - Infectious disease agents
    - Mycoplasma spp.
    - Brucella abortus
    - Borrelia burgdorferi, a spirochete causing, Lyme Disease

- Diagnosis:
  - Enlarged, painful joint, bursa or tendon sheath
  - Synovial effusion
  - Grade 5 lameness
  - Radiography, gas in peri-articular tissues may evident
  - Synovial fluid analysis & culture

- Treatment:
  - Eliminate the organism
  - Remove harmful material in the joint,
flush joint
install a drain tube

Bog Spavin
- Synovial and effusion of the tibio-tarsal joint
- Frequently associated with osteochondrosis
- Can also be caused by sprains, synovitis or other trauma
- Often asymptomatic, with no lameness, when examined
- Treatment unrewarding

Windpuffs
- Fetlock, Hock, Knee
- Swelling around joint capsule
- Swelling in tendon sheath or bursa
- Result from a sprain, OCD, synovitis or trauma
- Often asymptomatic and only a blemish rather than a lameness

- **Neurologic disorders**
  Viral encephalitis and myelitis, equine encephalitis viruses, rhinopneumonitis virus
  Equine Protozoal Myeloencephalitis, Sarcocystis neurona, a parasite with the opossum as the definitive host and source of infection
  Otitis media, otitis interna

- **Disorders of the Sole and frog**
- **Sole Bruise**
  Probably the most common cause of limping (or walking tenderly)
  Trauma from stones and other objects
  Cleaning with a hoof knife will reveal blood staining, bruising and darkening of sole
  Treatment is shoeing with pads
  Cattle can suffer from this condition also, especially large bulls

- **Subsolar abscess**
  Infected puncture wounds on sole
  Treatment
  - Open wound and establish drainage
  - Lavage
  - Antibiotics
  - Tetanus toxoid, etc.

- **Gravel**
  Infection of the white line
  Dissecting infection travels up the hoof wall
  Abscess opens at coronary band
  Caused by stone bruising or puncture
  Treatment same as sole abscess
  - Establish drainage
  - Lavage.
  - Antibiotics
  - Tetanus toxoid

- **Thrush**
  Bacterial infection in the sulci of the frog
  Cause:
  - Moist environment
  - Dirty, manure packed feet
  Treatment:
- Move to dry environment
- Removal of necrotic frog and sole
- Topical Kopertox (copper napthalate)
- Antibiotics are indicated if animal is lame due to a deep or extensive infection

**Foot Rot** *(Interdigital Pododermatitis and Phlegmon)*
Bacterial infection in the interdigital cleft in cattle, other ruminants and swine

**Cause:**
- Trauma to the interdigital skin, abrasion, etc., which becomes infected
- Dirty, wet environment
- Fusobacterium necrophorus is the most common pathogen
- Infection can be superficial (dermatitis)
- Or the infection can gain access to deep fascial layers (phlegmon)

**Treatment:**
- Move to dry environment
- Removal of necrotic skin and establish drainage
- Topical Kopertox (copper napthalate)
- Antibiotics

**Hoof Wall Injury and Disease**

**Hoof cracks**
Develop at any point on the hoof
Cracks will frequently migrate upward into the coronet

**Predisposing factors**
- White feet
- Uneven trimming of hoof wall or untrimmed hoof
- Use of an unshod horse on rough terrain

**Treatment:**
- Treatment aimed at preventing crack from migrating into coronary band, resulting in a permanent growth defect of hoof
- Score or drill proximal end of crack (prevents migration)
- Debride and smooth crack using dremmel tool
- Debride down to sensitive lamina
- Use wire to reinforce crack
- Pack debrided crack with acrylic cement (Technovit or other)
- Apply shoes with clips to support crack (prevents spreading of hoof)

**Laminitis**

**Acute Laminitis**
Inflammation of the sensitive lamina of the hoof wall
Laminitis can affect all species of hoofed animals
Separation of the hoof wall from the sensitive lamina

**Typical Stance in horses**
- Camped under behind
- Holding front leg out in front
- Stance will remove weight from the front feet

**Common causes:**
- Overeating, “grain founder”, “grass founder”
  - Overeating or eating excess concentrate feed
  - Bacterial proliferation and starch fermentation in cecum and colon
  - Lactic acid production, acidemia, diarrhea
  - Endotoxin release from gut
  - Inflammatory mediators in general circulation
- Pneumonia, metritis and other infectious diseases
- Endotoxemia & circulating inflammatory mediators
- Black Walnut toxin in wood shavings bedding
Pathogenesis – UNCLEAR and varies with cause

- Early increase in blood flow in all cases
- **OLD THEORY**
  - Foot edema and increase pressure inside hoof
  - Later; blood flow decrease
  - Ischemic necrosis of the lamina
- **NEW THEORY**
  - Matrix Metaloproteinase (MMP) enzyme activity increases due to inflammatory mediators
  - MMP enzyme results in lamellar breakdown
- Actual pathogenesis probably incorporates old and new
- Separation of the hoof wall lamina from the sensitive dermal lamina of the third phalanx
- Rotation of the coffin bone due to separation of lamina layers
- Loss of support for coffin bone leads to pressure on sole
- Coffin bone will actually rest directly on sole (dropped sole)
- Perforation of sole and exposure of the coffin bone is possible

**Chronic laminitis**
Laminitis leads to decreased hoof wall growth due to the separation of the hoof wall (epidermis) from the sensitive lamina (dermis)

- If the toe and quarter areas of the hoof are affected by separation while the heal remains relatively intact, the toe and quarter hoof wall growth is dramatically decreased, while the heal hoof wall growth remains fairly normal. This results in “differential growth” of the hoof wall.
- The degree of hoof wall deformity due to differential growth depends on severity that different areas of the hoof wall are affected
- Results in laminitis rings on hoof wall
- Leads to “slipper foot”
  - Decreased hoof wall growth on anterior hoof relative to heal
  - Dropped sole anteriorly

**Treatment of Acute Laminitis**

- Emergency medical management
  - Clear the intestinal tract, mineral oil
  - Treat primary inciting disease
  - Ice or cold water soak of affected feet
    - Intermittently for 24 hours if possible
  - NSAI DS (phenylbutazone, flunixin, etc.)
- Eliminate grain, lush pasture and legume hay
- Soft deep bedding in a small stall

**Treatment of Chronic laminitis**

- Corrective trimming, shoeing
  - Heart Bar Shoe moves weight bearing to rear of foot / frog
- Hoof wall resection
  - Allows for resolution of severely rotated third phalanx

**Sidebone**
Ossification of the collateral cartilages in the heal area of the horses hoof
Frequently see mild swelling with no signs of lameness
Some ossification occurs in many horses with aging
Fractured collateral cartilage is a possible sequela – severe lameness

**Other entities causing lameness**

**Splints**
Disruption or ossification of the interosseous ligament
Usually medial
Swelling in area of second metacarpal bone
Must be differentiated from splint bone fractures
Trauma from interference
  · Winging of opposite limb
Lameness goes away with time
May leave a permanent blemish

· **Bucked shins**
  Periostitis and stress fracture of the cranial aspect of the cannon bone
  Very painful
  Heals in time

· **Sweeny**
  Result from trauma to the suprascapular nerve
  Common in draft horses
  Use of poorly fitted draft collars
  Atrophy of the supraspinatus and the infraspinatus muscles

**INFECTIOUS DISEASES**

**Viral Diseases (Respiratory and other viral diseases)**

**Influenza Virus Infections**

· **Equine Influenza**
  Caused by an influenza virus in the Orthomyxovirus family. It is thought that equine infl
  Diagnosis:
    · Virus isolation
    · Paired serology (SN)
  Transmission
    · Spread via inhalation of infectious virus
    · Spread on environmental surfaces
    · Young horses in crowded conditions are most susceptible
  Pathogenesis:
    · Short incubation period--1-3 days
    · Respiratory inflammation
      · Erythema and edema in epithelium
      · Focal erosions of respiratory epithelium
    · Can lead to pneumonia, pericarditis and pleuritis
  Signs:
    · High fever
    · Depression
    · Anorexia
    · Coughing
    · Serous nasal discharge
    · Swollen lymph nodes
    · Weakness and stiffness
    · Leukopenia and other sequela
  Treatment is supportive in nature
    · NSAID
    · Prophylactic antibiotics
  Prevention
• Routine immunization
  ý Immune responses are weak and short
  ý Start all foals at 3-6 mo of age
  ý Initial series: 2 or 3 IM injections, 3-4 weeks apart
  ý Booster according to risk; 2 - 4 times per year
• Management
  ý Isolate new horses
  ý Adequate ventilation
  ý Good disinfection of equipment

• Bovine Parainfluenza 3 (PI 3)
  Etiology
  • Parainfluenza Virus-3
  • Viral infection that is uncomplicated by bacterial infection is usually mild
  • Morbidity up to 100%
  • Low mortality (less than 5%)  
    ý Mortality associated with secondary bacterial pneumonia
    ý A severe form of the disease can result in death
  • Calves 3 to 10 months of age most susceptible
  Pathogenesis
  • Affects all levels of the respiratory tree
  • Interstitial pneumonia with some emphysema
  • Alveolar collapse (atelectasis)
  Symptoms ###
  • Usually mild to sub clinical infections
  • Initiator to secondary bacterial infections, pneumonia, bronchopneumonia
  • Cough
  • Serous nasal and lacrimal discharge
  • Lung consolidation
  • Bronchiolitis
  Diagnosis ###
  • Immunoassay
  • Fluorescent antibody
  • Paired Serum
  Treatment
  • Supportive
  • Broad spectrum antibiotics to prevent secondary infections
  • NSAID (Banamine IV)
  Prevention
  • Vaccination
    ý 1st vaccination at 2 to 3 months
    ý Modified live vaccine is safe and effective
    ý Intranasal vaccine available (combination with IBR)
  • Isolation of new cattle
    ý Especially from calves
    ý Prevent overcrowding

• Bovine Respiratory Syncytial Virus (BRSV)
  Etiology ###
  • A pneumovirus in the Influenza family
  Pathogenesis
  • Causes a characteristic interstitial pneumonia with pulmonary edema and emphysema
  • Can be fatal due to dyspnea and hypoxia but is frequently a mild disease
  • Becomes severe with secondary bacterial pneumonia
Symptoms ##
- Fever, Depression, Anorexia
- Cough
- Nasal discharge
- Dyspnea
- Emphysema
- Secondary bacterial pneumonia

Diagnosis ##
- Immunoassay
- Fluorescent antibody
- Virus isolation
- Paired Serology

Treatment
- Supportive
- Broad spectrum antibiotics to prevent secondary infections
- NSAID (Banamine IV)
- If BRSV is confirmed corticosteroid therapy may be of benefit

Prevention
- Vaccination
  - 1st vaccination at 2 to 3 months
  - Modified live vaccine is safe and effective
- Isolation of new cattle
  - Especially from calves
  - Prevent overcrowding

Herpes virus Infection

- Equine Herpes Virus Infections
  - Equine Herpes Virus-1 (EHV-1) Rhinopneumonitis respiratory disease,
  - EHV-1 abortion
  - EHV-1 neurologic disease (myeloencephalopathy), and
  - EHV-4 Rhinopneumonitis respiratory disease

Etiology:
- A variable species of herpes virus that can affect all species of equines
- The two varieties that affect domestic horses are:
  - EHV 1 Equine Rhinopneumonitis, Herpes viral abortion, Herpes viral myelo-encephalitis
  - EHV 4

Transmission
- Airborne
- Oral and respiratory contact

Pathogenesis:
- Highly contagious
  - Carrier animals exist
- Inhalation, ingestion of contaminated material
- EHV 1 causes:
  - Abortions
  - Upper respiratory infection
  - Encephalomyelopathy
  - High mortality in newborn
- EHV 4 causes:
  - Upper respiratory infections
  - Occasional abortions

Symptoms
- 2-10 day incubation
- Unapparent infection is common
• Young horses more likely to show signs
• Rhinitis, fever, conjunctivitis, coughing
• Short illness- 2-5 days
• Persistent cough
• Secondary pneumonia
• Abortion
• Outbreak of late term abortions
  y Can follow respiratory phase by up to 4 months
  y Foals can be stillborn or weak at birth
  Severe respiratory distress and depression
  Usually die within a few hours of birth
• Neurological signs
  y Ataxia
  y Spinal cord signs

Treatment
• Supportive care
• Antibiotics
• NSAID
• Fluids and other symptomatic treatments

Prevention
• Preventive immunization
  y No protection against neurological form of disease
  y Immune responses are weak and short (Killed Virus Vaccine)
  y Start all foals at 3-6 mo of age
  y Initial series: 3 IM injections, 3-4 weeks apart
  y Booster according to risk; 2 - 4 times per year
• Vaccinate pregnant mares at 5, 7, and 9 months of gestation
  y Use inactivated virus EHV-1 or combination vaccine
  y Pneumabort ® Ft. Dodge is vaccine of choice for mares
  y Assists in prevention of EHV-1 abortions

• Infectious Bovine Rhinotracheitis (IBR)

Etiology
• Bovine Herpes Virus h-1
• Sometimes called “Red Nose”

Pathogenesis
• An Epitheliotropic virus
• Can infect many epithelial tissues
• Rapid incubation, 4-6 days
• Typical herpes like blisters, pustules and subsequent erosions on affected epithelium
• There can be few local lesions to extensive involvement of multiple locales
• Can cause several syndromes
  y Upper respiratory infections
  y Meningoencephalitis, occasionally in young calves
  y Abortion
  y Venereally transmitted disease
    Infectious Pustular Vulvovaginitis (IPV)
    Infectious Pustular Balanoposthitis (IPB)

Transmission
• Airborne
• Oral and respiratory contact
• Venereal

Symptoms
• Respiratory disease
  y Oral, nasal, tracheal and bronchial pustules and erosions
Dyspnea and open mouth breathing
GI lesions can occur
Viral infection by itself is mild
Recovery in 4-5 days
Bacterial infection (pneumonia) is the main complicating factor

Genital Infection; IPV and IPB
Infectious Pustular Vulvovaginitis (IPV)
Vestibular and Vaginal epithelium become infected
Infectious Pustular Balanoposthitis (IPB)
Glans penis and prepuce epithelium become infected
2-5 mm blisters, pustules and subsequent erosions on genital epithelium
2 to 3 week duration
Transmissible during coitus
Virgin animals can acquire infection via oral (or respiratory) to genital transmission
If bacterial infection does not ensue, fertility is unaffected

Abortion at any stage of gestation
Abortion occurs 20 to 90 days after any forms of the disease
Abortion can occur after vaccination of naïve, pregnant cows, w/ MLV

Diagnosis
Clinical signs primarily
Virus Isolation (VI)
Tracheal wash, nasal swab or Necropsy material
Virus Fluorescent Antibody (FA)
Identifies presence of virus in exudate or tissues
Serology
Serum Neutralization (SN)
Kinetic Enzyme Linked Immuno-Sorbent Assay (K – ELISA)
Paired samples are required
Obtain samples 2 weeks apart
A rising titer is diagnostic of infection or recent vaccination

Treatment
Supportive
Broad spectrum antibiotics to prevent secondary infections
NSAID (Banamine IV)

Prevention
Vaccination
1st vaccination at 2 to 3 months repeat in 2-3 weeks or prior to weaning
Modified live vaccine is safe and effective on calves
Do not use MLV vaccine on pregnant cows or on calves nursing pregnant cows
Intranasal vaccine available (combined with PI3, TSV-2, Pfizer (The intranasal vaccines contain temperature sensitive live viral strains that are safe to use on pregnant cows)
Isolation of new cattle
Vaccinate new cattle upon arrival and repeat in 2-3 weeks
Do not co-mingle with other new arrivals
Prevent overcrowding

Togaviridae Virus Infections
(A group of RNA containing viruses)

Equine Viral Arteritis
Etiology
Caused by arterivirus in the Togaviridae family
Pathogenesis:
- Upper respiratory tract infection
- Abortion
- Arteritis

Transmission
- Ingestion of contaminated material
- Inhalation
- Virus can survive in environment 10 days
- Virus is shed in urine and semen
  - Infected stallions can be carriers for several years
  - Testicular and epididymis infection
  - Venereal transmission
- Transplacental transmission

Symptoms
- Incubation of 1-6 days
- Fever
- Vascular damage
- Ocular
  - Lacrimation
  - Conjunctivitis
- Pulmonary edema w/ pneumonic symptoms
- Limb edema and abdominal edema (ascites)
- Abortion
- Course of disease is 3-8 days in surviving animals

Diagnosis
- Paired Serology (Serum Neutralization)
- Virus Isolation

Treatment
- Supportive treatment
- Antibiotics
- Anti-inflammatory drugs
- Diuretics; when peripheral edema or severe respiratory problems are present

Prevention
- Vaccination
  - A modified live vaccine offers some protection
- Test animals
  - Remove any stallion from service if he tests positive for the virus

• **BVD (Bovine Viral Diarrhea)**

Etiology
- Pestivirus (family Togaviridae)
- Related to hog cholera and border virus of sheep
- Related to Equine Viral Arteritis
- Several strains
- Cytopathic and non-cytopathic variants within strains
- BVD virus easily mutates into new variants
  - i.e. from non-cytopathic to a cytopathic variant

Pathogenesis
- Epitheliotropic
  - Affects several epithelial tissues
    - Respiratory tree
    - GI epithelium
    - Epidermis of mouth, pharynx, larynx
    - Skin of the nose, horns and feet corium
Lesions can resemble Foot and Mouth Disease if severe
- Causes necrosis of mucosal lymphoid tissues
  - Peyers patches in small intestines, ileum
  - Pharyngeal lymphoid tissue (Tonsillar tissue)
  - Immunosuppression, (Lymphoid depletion?)
- During the febrile, viremic stage of the disease many other tissues and the fetus of pregnant cows can become infected
- CNS tissue of fetuses in very vulnerable

Transmission
- Horizontal
  - Airborne – respiratory
  - Fecal contamination
  - Fomites
  - Animal – animal contact
  - Persistently Infected (PI) calves are responsible for maintenance of infection in a herd
    and will shed virus into environment and infect herdmates
  - PI calves were infected as a fetus (vertically)
    Act as a virus carrier for life

Transmission
- Vertical
  - Trans-placental
    - Fetal death, abortion  (infection of fetus in 1st trimester)
    - Congenital abnormalities (infection of fetus in 1st trimester)
    - Congenital disease  (infection of fetus in 3rd trimester)
    - Persistent infection  (infection at 4 Mo. Gestation)

Symptoms and signs
- Respiratory distress
- Diarrhea
- Fever and depression
- Oral, pharyngeal & esophageal lesions, excessive salivation
- Nasal and tracheal lesions, bloody & purulent nasal discharge
- Bronchitis and Pneumonitis, coughing

Unique syndromes
- Mucosal disease is a severe, peracute, fatal form of BVD
  - Occurs when a non-immune or a non-immunocompetent animal acquires a cytopathic BVD virus.
  - Usually occurs in persistently infected calves
- Neurological disease in neonatal calves
  - Due to cerebellar dysgenesis or agenesis
- Congenital defects
  - Cerebellar agenesis or dysgenesis
  - Cranial, spinal and limb deformities
  - Ocular deformities
  - Alopecia
- Persistently infected (PI) carriers
  - Occurs with transplacental transmission at 3 to 4 months gestation. This coincides with a period where the fetal immune system is developing it’s “recognition of self”
  - Fetus develops immunotolerance to any infecting BVD virus and if the virus is non-cytopathic the fetus may survives to term
  - PI calves have no immunity (antibodies or cellular response) to the BVD strain it is infected with
  - PI calves may be normal at birth
PI calves usually are “poor doers”, due to activity of the virus and immunosuppression

PI calves may develop Mucosal Disease” when infected by a cytopathic BVD virus

BVD Vaccination of PI calves may precipitate Mucosal Disease

Diagnosis of PI calves

1) Identification of virus:
   Positive Viral FA or
   Immunohistochemistry of biopsy sample or
   Virus isolation on blood, exudate or tissue sample
2) Negative serological (SN) test.
   No antibodies!
   In spite of viral presence!

Diagnosis

- Clinical signs
- Virus Isolation
  - Esophageal swab
  - Nasal swab
  - Blood (EDTA or heparin tube)
  - Fecal sample
  - Necropsy material, fetus
- Virus Fluorescent Antibody (FA)
  - Identifies presence of virus in exudate or tissues
- Serology
  - Serum Neutralization (SN)
  - Paired samples are required (as with IBR)
  - Obtain samples 2 weeks apart
    A rising titer is diagnostic of infection or recent vaccination

Treatment

- Supportive
  - IV & oral electrolytes when severe GI symptoms occur
- Broad spectrum antibiotics to prevent secondary infections
- NSAID (Banamine IV)

Prevention

- Vaccination
  - 1st vaccination at 2 to 3 months repeat in 2-3 weeks or prior to weaning
  - Modified live vaccine is safe and effective on calves
  - Do not use MLV vaccine on pregnant cows or on calves nursing pregnant cows
  - Annual vaccination prior to breeding with MLV vaccine to eliminate PI calves
- Isolation of new cattle
  - Vaccinate new cattle upon arrival and repeat in 2-3 weeks
  - Do not co-mingle with other new arrivals
  - Prevent overcrowding
- Test for BVD and cull all persistently infected cattle
  - Skin biopsy (ear notch) is a good, general-purpose test to identify animals that have the virus present. (most will be PI)
  - Further testing can be done, if desired, to confirm PI status

Ulcerative diseases

Could be confused with BVD, IBR

Differential diagnosis for oral ulcers

- BVD
- IBR
- Bluetongue (orbivirus)
- Vesicular Stomatitis (rhabdovirus)
- Foot and Mouth (enterovirus), exotic, not in US.
Swine Vesicular Disease (enterovirus), exotic not in US
Rinderpest (myxovirus), exotic not in US
Papular Stomatitis
Others

Bacterial Pneumonia

- **Strangles (equine distemper)**
  
  **Etiology**
  - Caused by Streptococcus equi
  
  **Transmission**
  - Highly contagious bacterial disease
  - Young horses more susceptible
  - Inhalation or ingestion of contaminated material
  - Horses can shed the bacteria for up to 4 weeks after getting the disease
  - Organism can last for long periods in environment
  
  **Pathogenesis**
  - Pharyngitis and laryngitis result from the initial acute infection
  - Incubation of 4-8 days
  - The bacteria invades the lymph nodes causing inflammation and then abscessation
    - Submaxillary, Mandibular nodes inflammation and abscessation causes unsightly draining lesions and act as massive source for S. equi organisms
    - Retropharyngeal node abscessation can cause obstruction of the air way (strangles)
    - Secondary guttural pouch empyema
    - Spread to other organs (“bastard strangles”)
  
  **Symptoms**
  - Anorexia and depression
  - Fever
  - Purulent nasal discharge-copious
  - Cough
  - Abscessation of lymph nodes in throat area
  - Purpura hemorrhagica – rare
  
  **Diagnosis**
  - Clinical symptoms
  - Culture from abscess drainage
  
  **Treatment**
  - Isolate
  - Antibiotics,
    - Controversial, thought to predispose to bastard strangles
    - Appears to be best approach to serious illness
  - Drainage of abscesses
  - Supportive care
  - Disinfect environment
  
  **Prevention**
  - Routine immunization with killed whole cell bacterin
    - 3 IM injections at 3-4 week intervals
    - Begin vaccinations at 2 months
    - Biannual vaccination of yearlings
    - Annual booster 2-4 weeks prior to foaling for mares
    - Annual or biannual boosters for other horses as needed
    - Reactions at injection site are common
  - Vaccination with avirulent live bacterial culture, intranasal
    - Pinnacle IN ® Ft. Dodge
    - 2 doses at 3-4 week intervals
- Begin vaccinations after weaning is probable the best approach
- ?? No recommendation from Ft. Dodge
  - Annual booster
  - Do not use in pregnant mares
  - Use caution in handling live bacterial cultures – local infection
  - Isolate infected animals

• **Pasteurella pneumonia**
  **Etiology**
  - Gram negative ovoid bacilli
  - Pasteurella hemolytica is the most common bacterial species that affects cattle in the “Bovine Respiratory Disease” (BRD) complex
  - Pasteurella hemolytica
    - “Shipping Fever” pneumonia
    - Pleuritis
    - Hemorrhagic septicemia
    - Weanling and feedlot cattle
  - Pasteurella multocida
    - Respiratory infection
    - Common wound contaminant
    - Opportunistic bacteria
    - Dairy cattle
    - Young suckling calves
    - Hogs
    - Sheep
  **Pathogenesis**
  - Pasteurella are frequently normal pharyngeal inhabitants
  - Viral infection and stress will predispose an animal to Pasteurella infection
  - Early pharyngitis, laryngitis
  - Bronchopneumonia and pleuritis develops as organisms reach the bronchioles and alveoli
  - Hemorrhagic septicemia result from Pasteurella septicemia and endotoxemia
  **Symptoms**
  - Fever
  - Cough
  - Muco-purulent nasal and ocular discharge
  - Dyspnea as pneumonia advances
  **Treatment**
  - This pneumonia can be hard to treat with high losses - **if not treated early**
  - Several antibiotics are available and effective for use in BRD complex
  - NSAID
  - Fluids and other supportive therapy
  - In the face of an outbreak or in high risk situations
    - It may be efficacious to treat all cattle in the herd with an effective antibiotic.
    - Prevent symptoms in animals incubating disease.
    - This type of procedure is called “Metaphylaxis”.
  **Prevention**
  - Several vaccines available
  - Typically 2 injections, 3 to 4 weeks apart, prior to or at weaning

• **Haemophilus somnus**
  **Etiology**
  - Gram negative ovoid bacilli
  **Pathogenesis**
"Haemophilus Septicemia of Cattle"
- Pneumonia and pleuritis
  - Difficult to differentiate from pasteurella clinically
- TEME (Thrombo Embolic Meningo Encephalitis)
  - From septicemic seeding of the CNS
- Septic synovitis and arthritis
- Vaginal / uterine infection may affect reproduction

Transmission
- Direct
- Aerial transmission from inapparent carriers

Symptoms
- Fever
- Depression
- Signs of septicemia
- Respiratory symptoms
  - Cough
  - Muco-purulent nasal and ocular discharge
  - Dyspnea
    - From pneumonia
    - From Pleuritis
- TEME
  - Depression, ataxia, weakness and recumbency
  - Other asymmetrical neurological signs

Treatment
- Several antibiotics are available and effective for use in BRD complex
- NSAID
- Fluids and other supportive therapy

Prevention
- Vaccine is available
- Typically 2 injections, 3 to 4 weeks apart, prior to or at weaning

Arboviruses Infections (arthropod borne viruses)

- **Equine Encephalomyelitis**
  
  **Etiology**
  - 3 species of Alphavirus in the family Togaviridae
  - Western Equine Encephalomyelitis, (WEE)
  - Eastern Equine Encephalomyelitis, (EEE)
  - Venezuelan Equine Encephalomyelitis, (VEE)
    - Severe epizootic occurred in Texas in 1971
    - VEE prevention is important in southern US

  **Transmission**
  - Birds are Primary hosts or Reservoirs for encephalomyelitis viruses
  - Horses and humans are secondary (dead end) hosts
  - Spread via mosquito (disease vector)
    - From birds to other birds
    - From birds to horses, humans (primary to secondary host)
    - From infected horses to other horses (rare)
    - Incidence is highest when the mosquito population is the highest

  **Pathogenesis and clinical signs**
  - The virus does not always penetrate the nervous system
  - Infection may be inapparent
  - Viremia may be accompanied by fever and other mild symptoms
• Mortality:  
  WEE = 20-50%  
  EEE = 40-90%  
  VEE = 40-90%  

Symptoms
• If the virus penetrates the nervous system; signs would include:
  - High fever
  - Depression
  - Hypersensitivity to stimuli
  - Incoordination and ataxia
  - Poor prehension of food
  - Compulsive behavior, head pressing, maniacal walking
  - Blindness
  - Seizures
  - Paralysis

Diagnosis:
• CF and SN tests  
• ELISA  
• Virus isolation, post-mortem

Treatment:  Supportive care
Prevention; Immunization
• Killed multivalent vaccine  
• EEE and WEE are most commonly included in vaccine products  
• Initial immunization:
  - Two IM injections 3 to 4 weeks apart  
  - Start foals from vaccinated mares at 3-4 mo. of age  
  - Start foals from unvaccinated mares at 2-3 mos. of age  
• Booster immunizations
  - Administer in the spring, prior to the ‘vector season’  
  - Two boosters per year in areas with year round mosquitoes
• In the Southern US: Florida, Texas use VEE vaccine also
  - Prevent re-introduction of VEE from Mexico & S.A.

• **West Nile Virus**

  Etiology
  • A Flaviridae  
  • An emerging disease in the US  
  • First discovered in US in August 1999 on Long Island, New York  
    - 29 cases in horses  
    - Numerous cases in birds  
    - Human fatalities have occurred  
    - 59 cases in 2000  
    - Has spread rapidly since ‘99  
    - Florida to the south in 2001  
    - Michigan to the west in 2001  
    - Hundreds of cases

Pathogenesis
• Similar to EEE or WEE  
• Clinical sign evident in less than 50% of infected horses  
• Approx. 40% mortality in horses showing clinical signs

Symptoms
• Indistinguishable from EEE or WEE

Vaccine has been developed
• Fort Dodge is manufacturer of the first vaccine  
• Efficacy is less than 100%

• **Equine Infectious Anemia**
• Swamp Fever (chronic form of the disease)

Etiology
- Caused by a Lentivirus
- RNA virus
- Related to immuno-deficiency viruses HIV, FIV
- As with immuno-deficiency viruses, EIA infection persists for life
- Persistently infected animals are a source of infection

Transmission
- Transmitted via blood from infected, viremic horses
- Horseflies and deerflies are main vectors
- Mosquitoes could also transmit virus
- Virus survives less than 4 hours in fly mouthparts
- Contaminated needles and instruments
- Vertical transmission
  - Transplacentally
  - Colostrum, milk?

Pathogenesis
- Virus primarily infects mononuclear cells
- Replication only in mononuclear cells
- Replication primarily in tissue macrophages (a mononuclear cell)
- Does not infect lymphocytes thus no immuno-suppression
- Strong but only partially effective immune response
  - Cellular immunity responsible for suppression of viremia
  - Ineffective humoral antibodies are part of pathogenesis
    - Antibody coated virus is still infective
    - Antibody-virus complex attaches to or infects many cell types
    - Antibodies help cause viral infection of non-mononuclear cells
    - Antibody-virus complex coated cells susceptible to immune (compliment) mediated lysis
      - RBCs, thrombocytes and renal glomerulus cells destroyed
      - Results in anemia, hemorrhage glomerulonephritis, etc.
- Virus mutates often
  - New strains are responsible for intermittent recurring viremia

Signs
- Mostly inapparent
- Acute infection and recurrent viremia
  - Fever
  - Depression
  - Anorexia
  - Mild anemia
  - Thrombocytopenia
- Chronic infection (Swamp Fever)
  - Anemia
  - Thrombocytopenia and hemorrhages
  - Hypoalbuminemia and edema
  - Fever
  - Weight loss
- Abortion if viremia occurs during pregnancy
- Mild glomerulonephritis may occur even in inapparent infection

Diagnosis and control
- EIA is a USDA and state DA reportable disease
- AGID test, “Coggins Test”, specific for EIA core antigen
  - Only USDA approved labs can perform official EIA test
  - Test required for interstate movement, sale and equestrian events
  - Required by most insurance companies
- EIA positive horses
Must be destroyed or:
- If not euthanized they must be quarantined for life
- Quarantine stipulates removal by at least 200 yards from all other equines
- EIA positive horses cannot be moved except to slaughter or an USDA approved research center
- EIA positive horses must be branded with an A and other ID numbers on the neck
- No vaccine available due to large number of antigenic variants and continued mutation

Other Bacterial Diseases

- **Tetanus:**
  - **Cause:** Clostridium tetani
    - Gram +
    - Spore forming
    - Anaerobic
    - It is usually introduced through wounds.
  - **Clinical signs**
    - Lameness
    - Stiffness
    - Inability to eat
    - Tetanic convulsions
  - **Treatment,** (difficult)
    - Antitoxin; neutralize toxin
    - Antibiotics (penicillin), eliminate organism
    - Diazepam, control neurologic symptoms
    - Supportive care
  - **Prevention**
    - Pre-exposure - immunization with tetanus toxoid
      - Two initial injections 4-8 weeks apart
      - Annual booster.
    - Post-exposure
      - Vaccinated animals:
        - Booster with tetanus toxoid
      - Unvaccinated animals:
        - Antitoxin 1500-3000 I.U. subcutaneously
        - Commence tetanus toxoid series

- **Brucellosis**
  - **Hosts**
    - Domestic animals
    - Wild animals
    - Humans
  - **Etiology**
    - Gram negative bacteria
    - Brucella abortus - cattle
    - Br. suis - swine
    - Br. melitensis - goats
    - Br. canis - dogs
    - Br. ovis - sheep
    - All species infectious for all domestic animal
    - Man is susceptible to all
  - **Resistance**
• Heat
  · Destroyed by 60°C for 10 minutes
  · Pasteurization
  · Sunlight, < 1 day
• Survives days to months in the environment

Transmission
• Spread may be vertical or horizontal
• Bacteria is present in most body fluids
  · Very heavy load of bacteria in uterine fluids at abortion
    Vaginal shedding several days before parturition or abortion, continues for one mo.
  · Milk from infected animals
  · Semen
• Ingestion of
  · Un-pasteurized milk
    Calves
    Humans
• Direct contact with infectious material via:
  · Conjunctiva (droplets from tail, membranes or vaginal discharge)
  · Abraded skin
  · Respiratory tract
  · Reproductive tract (venereal transmission – sheep)
• Public Health Aspects
  · Human susceptible to all brucella species
  · Disease in humans is called “Undulant fever”
  · Occupational disease (Veterinarians, packing house workers and others animal workers
  · Vaccine (strain 19 or RB51) can cause human disease
  · Disease in humans treated with tetracycline and streptomycin

Pathogenesis
• Following penetration – localizes in organs and adjacent lymph nodes
  · Mammary gland
  · Reproductive tract of females and males
  · Horse - ligamentum nuchae and navicular bursa
  · Fetus infected as early as 5th week of gestation
• Intracellular infection
  · Very difficult to treat w/ antibiotics
• Incubation period in cattle is 3 -12 weeks
• Orchitis – ram, bull and boar
  · Bulls play minor role in spread
• Mastitis
• Weak calves or pigs
• Arthritis - discospondylitis
• Horse
  · Fistula of withers
  · Poll evil
• Not an important cause of infertility in the bull
• Calves nursing infected cows get a transient infection.
• Females most susceptible during pregnancy
• Abortion,
  · 2-3 mo after contact
  · Any state of gestation
  · Retained placenta
  · Lowered conception - temporary sterility

Diagnosis
• Clinical
All aborting cattle should be tested
Rams, boars and bulls with orchitis should also be tested

- Brucella milk ring test (dairy cattle)
  - Hematoxylin stained antigen added to tube of milk
  - Incubate 37°C for 30 - 60 min.
  - Blue milk with white cream layer = negative test
  - White milk with blue cream layer = positive test
  - Not effective in low fat milk, colostrum
  - Can not test non-lactating animals
  - 3 negative tests per year to maintain negative herd status

- Tube, Card and Rapid plate agglutination tests
  - Serum diluted two-fold for plate or card test
  - Tube test uses serial dilutions

- Mercaptoethanol test
  - Mercaptoethanol inactivates IgM but leaves IgG

- Rivanol test - precipitation plate agglutination test
- Rivanol precipitates IgM in serum
- Buffered antigen, Card test
- RB51 Vaccinates can be differentiated from naturally infected animals
- No serological test to differentiate strain 19 vaccination and wild strain infections

Test animal identification
- Cattle markets (sale barns, packing plants)
  - Backtag - Market Cattle Testing program
  - Identification tag applied on all potentially test eligible cattle (6 mo. old for non-vaccinates, two yrs old or older for vaccinates) before co-mingling with other cattle at market.
  - Tag should be applied on cattle marketed through dealers, stockyards, order buyers or direct to packer
  - Tag identifies animal so it can be traced to original owner
  - ID remains with animal through slaughter inspection and blood collection
  - All slaughtered cattle are blood tested for brucella
- On farm cattle
  - Steel clip ear tag for all tested cattle

Brucella control programs
- Veterinarians must be accredited by USDA & State DA
- Certified free herd
  - Test entire herd at least once per year
  - New additions should be tested and should come from a certified free herd or area
  - All new additions should be isolated 30 days and retested
- Herd eradication
  - Test all animals
  - Reactors are slaughtered
  - Clean and disinfect premises
  - Retest at specified interval, 60 days, until no further reactors
  - Then follow procedure for a certified free herd

Treatment - None practical
- Reactors
  - Branded on left jaw, B
  - Reactor tag in left ear
  - Permit required to move to slaughter

Immunization
- Strain 19 vaccine- NO LONGER USED
- RB51 Vaccine used as of October 1997
- Vaccine causes no infertility in female
- Vaccine organism is pathogenic for humans
• Bulls should not be vaccinated
  • Residual titer
  • Infertility - orchitis
• Eligible Animals
  • Only heifer calves
    - between 4 and 12 months of age (beef)
    - between 4 and 10 months of age (dairy)
    - Best time is 4 to 6 months of age
    - Some states have stricter requirements
  • Adult vaccination for brucellosis is conducted by state or federal officials only

Vaccinate identification
• with a tattoo, either 3V1 (strain 19) or RV1 (RB51) in right ear, (R = RB51, V = Vaccination shield, 1 = year of vaccination
• R replaces the quarter for RB51 vaccine, for strain 19 the quarter of the year is used as first digit in the tattoo
• Orange steel clip tag with a unique ID number placed in the right ear, i.e. 84 VPI 1234 (84 = Colorado, VPI = identifies veterinarian 1234 = sequence #)

• Tuberculosis
  • Etiology
    • Mycobacterium tuberculosis
    • Acid-fast organism
    • Types
      • Mammalian
        - var. bovis
        - var. hominus
      • Avian
        - Mycobacterium avium
        - M. avium var. paratuberculosis causes Johnes disease in cattle and granulomatous enteritis in other species (horses)
  • Bacterial Resistance
    • Lives in soil for 4 years
    • Destroyed by pasteurization
    • Disinfectants
      • Cresylic disinfectants
      • Sodium orthophenylphenate
  • Transmission
    • Humans
      • Close contact
      • Poor sanitation
      • Un-pasteurized dairy products
    • Bovine
      • Primarily inhalation
      • Contaminated feed and water
      • Close housing
      • Contact with infected herd
      • Consumption of milk from infected cow
    • Swine
      • Primarily orally
      • Contaminated food and water
      • Uncooked garbage
    • Avian - all species susceptible
      • Contact with infected birds
      • Offal - infected birds
contaminated yards and equipment

**Symptoms**
- Main lesion is a caseous abscess
- **Swine**
  - Primarily lymph node involvement of pharyngeal, cervical and mesentary nodes
  - Primarily digestive tract involvement indicates oral transmission
- **Cattle**
  - Emaciated with GI disease
  - Respiratory system (pulmonary)
  - Many inapparent infections in infected herds

**Prophylaxis**

**USDA Eradication program**
- Caudal Fold PPD tuberculin skin test, used for routine testing of cattle
- Comparative Cervical Test, retest of all suspects on the caudal fold TB test, clarify cross reaction of mammalian and avian strains to the PPD caudal fold TB test
- All Grade A dairy herds must be accredited free (complete herd test every 2 or 3 years)
- Pasteurization of milk prevents transmission from infected cows to humans
- All slaughter cattle are examined for lesions
- Cattle moving interstate from non-accredited areas are tested
- Neither treatment or immunization are practical for livestock

**Public Health Aspects**
- Humans subject to disease
- Incidence has decreased following eradication programs for cattle

- **Leptospirosis**
  - Cause: L. pomona & about 100 other serotypes
  - Shed in urine; contaminating water supply
  - Wildlife act as reservoirs
  - Low mortality in cattle

  **Symptoms**
  - Abortion of infected fetus is the main symptom
  - Anemia
  - Decreased milk production
  - Respiratory distress due to anemia
  - Nephritis
  - Encephalitis in sheep and goats

  **Treatment**
  - Streptomycin or tetracycline

  **Prevention**
  - Multivalent bacterins
  - Environmental controls of livestock water sources

- **Clostridial diseases**
  - **Blackleg**
    - Myositis caused by Cl. chauvoei
  - **Malignant Edema**;
    - Gaseous and edematous necrosis of muscle and connective tissues
    - Cl. septicum is the classical etiologic organism
    - Other Clostridial organisms cause identical disease
  - **Blacks Disease and Bacillary Hemoglobinuria**
    - Necrotic infarcts in the liver, associated with Fasciola (liver flukes)
    - Caused by Cl. novyi and Cl. hemolyticum
    - Clostridial toxins cause vascular damage and hemolysis

  **Treatment** is difficult, frequently unrewarding
- Penicillin
- With myositis due to Cl. chauvoei or Cl. septicum the infected tissue must be incised and debrided, to establish drainage and expose the area to oxygen. Prevention is very effective with multivalent clostridial bacterins.

**Bovine and food animal issues**

**Pneumonia and Stress in Livestock**

**Stress - predisposes animals to disease**
- Weaning
- Shipping long distances
- Taking away from familiar environment
- Crowding - feedlot
- Change of feed
- Change of people
- Rough handling techniques
- Dusty conditions
- Wet and cold conditions

**Immunosuppression**
- Stress
- BVD (Bovine Viral Diarrhea)

**Bovine Respiratory Disease Complex - BRD complex**
- IBR,
- BVD,
- PI3,
- BRSV,
- Pasteurella spp. and
- Haemophilus somnus
- Mycoplasma bovis

**Hematological disease caused by Mycoplasm.a.spp. (Hemoplasmas) of Veterinary Importance**

Species: Hemoplasma
- Dogs: 1. Mycoplasma haemocanis (formerly Haemobartonella canis)
  2. “Candidatus Mycoplasma haematoparvum”
- Cats: 1. Mycoplasma haemofelis (formerly Haemobartonella felis)
  - (Causes Feline Infectious Anemia)
  2. “Candidatus Mycoplasma haemominutum”
  3. “Candidatus Mycoplasma turicensis”
- Pigs: 1. Mycoplasma suis (formerly Eperythrozoon suis)
  2. Mycoplasma parvum (formerly Eperythrozoon parvum)
- Cattle: 1. Mycoplasma wenyonii (formerly Eperythrozoon wenyonii)
- Sheep and goats: Mycoplasma ovis (formerly Eperythrozoon ovis)
- Llamas and alpacas: “Candidatus Mycoplasma haemolamae”

**Food Animal Issues**
**Animal Identification**

Permanent identification; of all animals is essential even in small herds. This allows the owner to track an animal’s pedigree, production data, genetic statistics, breeding data and veterinary medical tracking of sick animals and many other data items. One of the most important reasons to permanently identify all food animals is to prevent a treated animal, or milk from a treated dairy animal from prematurely entering the human food market with a violative drug or vaccine residue. After an animal has been treated with a pharmaceutical or vaccines, and before the expiration of the prescribed withdrawal period for that treatment, a meat animal cannot be slaughtered nor can the milk from a treated dairy animal be marketed. Without adequate identification it is easy to loose track of, or mistakenly identity any animal.

Some of the standard method of animal Identification used in the United States and elsewhere include:

**Ear Tags:**
- There are many different types of ear tags
- Standard plastic ear tags, sometimes referred to as a bangle tag can be pre-numbered or they can be marked with an indelible marking pen with a within-herd ID number.
- USDA approved 15 digit, ADT tag (Animal Disease Traceability) ID tags (these are a bangle tags) or RFID (Radio Frequency ID) button ear tags can also be used. These tags use a 15 digit numeric ID # that identifies the country (the US is 840) and a unique number identifies the individual animal. The # looks like: 840 003 123 456 789. Whether the 15 digit tag is a bangle tag or a RFID button tag, the livestock owner cannot purchase and use these tags unless the ranch or farm premise is registered with the USDA and the state department of agriculture. In summary the national animal identification system (NAIS) and ADT rely on premise ID as well individual animal ID for traceability to work. These tags will be required for all animal sales of animal 18 months of age or over, starting in March, 2013.
- Obsolete RFID tags (Radio Frequency ID tags) with the first three digits of ‘982’ and ‘985’ are currently being accepted as official ID for sale animals; however, the USDA will not accept these tags in the future. Producers are asked to discontinue use of these tags as soon as possible.
- USDA 9 alphanumeric digit bright metal clip tag, usually used when an animal is tested for a disease by an accredited veterinarian. These tags are also used on dairy cattle for ‘Dairy Herd Improvement Association’ identification of dairy cattle, similar tags, only a bit smaller are designed for use on sheep, goats and swine.
- USDA brucellosis calfhood vaccination tag, is also 9 alphanumeric digit, orange clip tags
- Dairy Herd Improvement Association (DHIA) tags with 9 -digit American number

Tattoos are a permanent ID that are applied to the skin on the concave surface of the ear. They can be a good animal ID but require restraint of the animal in order to be read. A Tattoo can be hard to read due to fading or poor tattooing technique. A tattoo is often considered to be a good emergency, last resort, form of identification for a registered animal that has lost its ear tag.

Freeze branding an animal with the herd brand and individual animal ID can be an easy to read, permanent ID. A tattoo and a freeze brand can be used interchangeably for an animal that has lost its eartag.

**Beef Quality Assurance Program**

- **Purpose**
  - Supply only quality beef
  - Improve consumer perception of beef’s safety
  - Elimination of drug residues
- **Good records of all treated animals**
  - Record of treatment is the responsibility of the owner and the person administering the treatment
Compliance with label or prescription dosage
Proper route of administration (SQ, IM)
Compliance with pre-slaughter drug withdrawal times
Veterinarian must order prolonged withdrawal time if label directions have not been followed exactly

- **Prevention of muscle damage through proper drug administration**

  Cleanliness - use sterile techniques
  Proper restraint
  Proper injection procedures
  Proper implant placement
  Use appropriate size needle for route of administration
  Use sharp needles
  Try not to inject when animals are wet
  Proper restraint assures proper administration
  Proper injection procedures

  - Oral administration is preferable to injections if possible. Always use a balling gun of the proper size to administer oral pills or boluses to food animals. Unfortunately, there are only a few drugs available for food animals that are approved for oral administration. A few examples are:
    - Sulfamethazine boluses (also available as an IV solution)
    - Sulfadimethoxine boluses (also available as an IV solution)
    - Magnesium hydroxide - Antacid boluses
    - Corid brand of amprolium to treat coccidiosis, usually administered as a solution with a dosing syringe.
    - Anti-diarrhea agents and nutritional supplements

  - Intravenous - is the next most preferable method of drug administration. It has the advantage in food animals of not creating a tissue depot of drug or carrier vehicle. Some drugs, such as injectable sulfonamides and sodium iodide, must only be given IV to prevent tissue damage. Oxytetracycline is sometimes mixed with saline or dextrose solution to be administered IV as a dilute solution to prevent tissue damage or venous damage from the concentrated, undiluted solution. For other drugs, such as flunixin meglumine, an NSAID, commonly used in cattle, IV is the only approved route of administration. Other drugs, such as penicillin-G suspension or tilmicosin (a macrolide antibiotic) can be fatal if administered IV. Always follow label or circular directions when administering pharmaceuticals to food animals.
    - The IV route minimizes the risk of muscle damage
    - 14 to 18 ga. 1½ to 2 inch

  - Subcutaneous - is the preferred injection method for beef cattle
    - On neck, behind the shoulder or at base of the ear
    - Avoiding muscle reduces potential trim at slaughter
    - Tent the skin
    - 16 to 18 gauge ¾ inch needle
    - "B" bevel needles may be preferable to the "A" bevel

  - Intramuscular
    - Neck musculature is preferred site for beef cattle
      - It is less valuable cuts of beef
    - Avoid semitendinosus and semimembranosus muscles
    - Avoid gluteus muscles or any other primal meat areas.
    - Remember, even dairy cows will eventually be used for beef
    - 16 to 18 gauge 1 to 1½ inch needle

  - Anabolic implants - Proper placement
    - Subcutaneous on convex surface of ear, using only the proper implanter, which has been designed for the specific implant being used
• **See hand out**

• **Extra label drug use**
  A valid patient - client - veterinarian relationship exists
  A diagnosis is made by a veterinarian
  It must be determined that:
  • There are no approved drugs specifically labeled to treat the condition diagnosed
  • The approved label dosage is ineffective
  • The condition to be treated is a life or performance threatening medical emergency
  No illegal drugs may be used (illegal for use in cattle), for example:
  • Chloramphenicol
  • Diethyl Stilbestrol

Withdrawal times
  • A significantly extended period of time is assigned for the drug withdrawal, in order to assure that there is no chance of a violative meat or milk drug residue.
  • The veterinarian is the only one who can determine the withdrawal time for extra label use of a drug.
  • Veterinarian will refer to research reports, and information in an online database to help determine the withdrawal time for extralabel drug use. The ‘Food Animal Residue Avoidance Databank’ (FARAD) is a congressionally-mandated risk-management program that is supported by the United States Department of Agriculture (USDA). FARAD's primary mission is to prevent or mitigate illegal or harmful residues of drugs, pesticides, biotoxins and other chemical agents that may contaminate foods of animal origin. [http://www.farad.org/](http://www.farad.org/)

Only a veterinarian can order extra label drug use
The attending veterinarian assumes responsibility
The client has agreed to follow all instructions
The veterinarian is available for follow up
The client must keep written compliance records