Why a Necropsy

- Surveillance
- Post calves with HX representative of clinical signs: Resp, CNS, Dig etc.
- Those lesions that don’t fit need to be explained
- Diagnostics
- Change in management: handling, feeding, facilities, treatment protocols

No Loose Parts Necropsy

http://GPVEC.UNL.EDU
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The Movie

Overview

- Necropsy Step by Step
- Gross pathology review / show & tell
- Questions/Discussion

Why a Necropsy

- Early-mid feeding period
  - More often for diagnostic purposes
- Later in feeding period
  - More often surveillance
Steps

1. Neck incision
2. Thoracic cavity
3. Abdominal cavity
4. Pelvic cavity
5. Superficial lymph node system

Necropsy Doc
Collecting the Brain

Steps
**Considerations**

- Contamination
- Do I need the brain completely in tact?
- Be sure to try to go low on the back of the head – near occipital condyles (Flex atlantoccipital articulation) to expose cerebellum

**Alternatives: saw, cleaver**

- More controlled

**Steps**

- This calf just died during the night. Why does the report say the intestines were autolyzed?
  - Autolysis begins within minutes after death.
  - Demonstration of how soon autolysis takes place

**Autolysis Demonstration**

- Euthanatized calf
- Intestines placed in Styrofoam cooler with no cold packs.
- Segments collected at hours 0, 1, 2, 4, 12, 15, 24
- Each segment opened lengthwise and fixed in formalin
**Intestine Fixation**
- Segments ~ two inches long
- Open lengthwise
- Don’t crush by handling with fingers
  - Cradle samples in hands
  - Handle tissues with forceps.
- If not done.....
  - Specimens will look like about the 12 hour sample even when from a euthanized calf

**Lung Fixation**
- If palpating lungs, don’t collect specimens for fixation from same area.
- Palpation creates crush artifacts such as atelectasis
- Use sharp knife or scissors
  - Cut sections ~ ¼ or ½ inch thick

**Respiratory System**

**Honker Syndrome**
- Marked hemorrhage in trachealis muscle
- Minimal inflammation
- Occasional vasculitis
  - *Histophilus somni*?
- Probably due to altered pressures secondary to inspiratory dyspnea
- Trauma to vessels, leakages of serum proteins progressing to hemorrhage
Histophilus somni

- Initially, embolic pneumonia

Mycoplasma bovis

- Very ubiquitous
- Frequently identified with multiple miliary abscesses
- Rarely identified as a sole infectious agent in feedlot respiratory disease cases
- Experimentally produced disease reported
- Enzootic pneumonia of calves, similar to mycoplasmal pneumonia in swine
- Peribronchial and perbronchiolar lymphoid hyperplasia experimental
- Reagents to study pathogenesis not readily available

Summary of UNL-VDC Cases Where *M. bovis* was Identified

- 242 samples
  - Lung
  - Joint swabs
  - Joint fluid
  - Nasal swabs
  - Lymph node
Is *M. bovis* a Primary Pathogen

- 242 samples
  - 4 lungs *M. bovis* only pathogen
  - 4 joints *M. bovis* only pathogen
- Pathogenesis
  - Infection via respiratory system, so needs to go systemic to cause arthritis
  - How many cases where arthritis is first clinical sign?
Viral Agents

- Bovine herpesvirus – 1 (IBR)
- Bovine viral diarrhea virus
- Bovine respiratory syncytial virus
- Bovine Coronavirus
- Bovine Adenovirus

Summary of IHC Data from UNL-VDC

- 326 cases with IHC
  - Jul04 – Jan06
  - Presenting complaint = “respiratory disease”
- IHC tests
  - BVDV
  - BHV-1
  - BRSV

Summary of IHC Data from UNL-VDC

- BRSV – 8 / 95 positive (8.4%)
- BVDV – 37 / 337 positive (11%)
- Skin – 7 / 83 positive (3 of 7 had only skin submitted) (8.4%)
- IBR – 4 / 65 positive (6.2%)

Bovine Respiratory Syncytial Virus (BRSV)

- Cattle probably principal reservoirs, transmission by aerosols
- Some isolates can cause severe primary disease while others may predispose to secondary bacterial infection
- Replication in epithelial cells of respiratory tract, primary lesion is deep in lungs

BRSV

- Uncomplicated BRSV infection – “red hepatization” in cranioventral distribution
- Transient infection, limiting detection of virus in nasal secretions and tissues to a few days after initial onset of clinical signs.
- Immunohistochemistry and PCR most reliable for detection
- Virus isolation rarely successful
BRSV Microscopic Lesions

- Bronchial and bronchiolar epithelial necrosis, often leading to secondary bacterial infection
- Syncytial (multinucleated) cells in bronchioles in early stages of disease.
- Intracytoplasmic eosinophilic inclusions in airway epithelial cells rarely observed
- Loss of cilia in airways

Bovine Viral Diarrhea Virus (BVDV)

- As it relates to respiratory disease, BVDV infection primarily interferes with the immune response to bacteria and other viruses
- Bacterial clearance from the lung is reduced due to effect on alveolar macrophages
- BVDV inhibits lymphocyte function and there is a reduction in B and T lymphocyte numbers
- Vasculitis results in leakage of serum proteins
BVDV
- Lesions
- Pneumonic lesions are generally those of bacterial pneumonia
- Sometimes seen in conjunction with BRSV
- BVDV infection alone may cause only mild pneumonia
- Immunohistochemistry, PCR, and virus isolation for detection in the lung

Infectious Bovine Rhinotracheitis (IBR)
- Pathogenesis
  - Virus replicates in respiratory epithelium causing necrosis of epithelial cells then transient viremia

IBR
- Gross lesions – generally upper respiratory disease with ulceration and crusts of the nares and nasal mucosa, fibrinous tracheitis
- Necrosis of nasal and tracheal epithelium with occasional eosinophilic intranuclear inclusion bodies
- Can sometimes detect viral antigen deep in the lung by means of immunohistochemistry
- Viral antigens or virus are best detected by immunohistochemistry and virus isolation
- PCR may become more widely used
Bovine Coronavirus

- Not commonly reported in surveys of bovine respiratory disease
- Same virus that causes enteric disease and so transmission is the same as with enteric disease; fecal – oral or oronasal
- Lesions consist of mild interstitial pneumonia and may be complicated by secondary bacterial infections or undetected in bacterial pneumonia
- Detection by virus isolation from nasal swabs or from lung using a specialized cell line not commonly maintained in many diagnostic labs or by fluorescent antibody test of lung or preparations made from nasal swabs
Bovine Adenovirus

- Only sporadically seen
- Detection usually by histopathologic observation of “typical” basophilic intranuclear inclusion bodies
- Lesions include necrotic bronchitis, bronchiolitis, interstitial pneumonia

Malignant Catarrhal Fever

- Ovine herpesvirus-2;
  - species rhadinovirus, subfamily Gammaherpesvirinae, family Herpesviridae. Latin root rhadino = fragile, relating to the lability of the virus.
  - Related to Kaposi’s sarcoma-associated herpesvirus (HHV-8)

Atypical Interstitial Pneumonia (AIP)

- Toxic metabolic intermediates affecting bronchiolar and alveolar epithelial cells
- Bronchiolar and alveolar epithelial necrosis
- 3-methyl indole
  - Metabolite of tryptophan
- 4-ipomeanol
  - Product of moldy sweet potatoes
Dictyocaulus viviparus

Liver
- Molded to the right half of diaphragm
- Attachments/ligaments
  - Vena cava to diaphragm
  - Area Nuda (dorsal medial diaphragm to right lobe
  - Right triangular ligament – dorsal margin right lobe to dorsal abdominal wall
  - Left triangular ligament – esophageal impression to diaphragm
  - Coronary ligament
  - Hepatorenal ligament – caudate lobe to rt kidney
A. pyogenes

Liver Flukes

- Fasciola hepatica – “Pipe stem” liver
- Fasciolides magna – Large fluke often black tracts in the liver due to digestion of blood
- Mainly SE & NW United States

Fasciola hepatica

Fasciolides magna

http://cat.vet.upenn.edu/pensa/index.html
**Fasciolides magna - histo**

**Gastrointestinal Tract**

**Congested Intestines**

**Forestomachs and Abomasum**
- Normal Structure
- Traumatic reticuloperitonitis
- Bloat
- Ruminal acidosis / Grain overload
- Abomasitis / Perforation

**Papillae unguiculoformae**
**Bloat**

- Frothy bloat
- Free gas bloat
- Physical entrapment

http://www.agric.gov.ab.ca/app21/rtw/index.jsp
Ruminal Acidosis / Rumenitis

Mycotic Rumenitis

- Secondary to prior damage to epithelium or disruption of normal flora
- Zygomycetes
  - Mucor
  - Rhizopus
  - Absidia
- Candida
Abomasal Perforation

- BVDV infection
- Salmonella sp.
- *Clostridium perfringens* (young calves)
- Hardware - rare
Stress Ulcer

Infectious Causes of Enteric Disease

- Salmonella
- Bovine viral diarrhea virus
- Eimeria bovis
- Ostertagia spp.
- Coronavirus

Salmonellosis

- Salmonella typhimurium
- Salmonella dublin
- Salmonella agona
Bovine Viral Diarrhea

- Affects Immune System
- Lymphopenia
- T cells and B cells
- Lymphoid depletion – lymph nodes, thymus, Peyer’s Patches, spleen
- Exacerbates infection with other agents
- Vasculitis
- Ulcerative lesions in GIT

Oral Ulcers

Gingivitis
Peyer’s Patches

- Stand back and look at the small intestines
- Easily identified on serosal surface
- Peyer’s patch is diffuse in the ileum
- No need to visualize it to collect
Ulcerated Jejunal Peyer’s Patch

Ulcerated Ileal Peyer’s Patch

Mucosal disease

Normal PP
Jejunal Peyer's Patch

Ileal Peyer's Patch - IHC

Hemorrhagic form of BVDV

Hemorrhagic Jejunal Peyer's Patch
Amprolium
Decoquinate
Sulfonamides
Ionophores

Ostertagia

Miscellaneous

Hypoderma lineatum
Malignant Catahrral Fever

- Crusts on muzzles
- Corneal opacity
- Hypopion
- Tracheolaryngeal erosions, catarrhal exudate
- Esophageal ulcers
- White foci on capsular & cut surfaces of kidneys

[Images of affected cattle and close-up views of eyes and organs]
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Thromboembolic Meningoencephalitis

- *Histophilus somni*
**Listeriosis**

- Often associated with history of silage from a new pit
- Growth continues at refrigerator temperature
- Less common – due to better silage handling?

**Organophosphate/Carbamate Toxicosis**

- Clinical Signs
  - Salivation
  - Lacrimation
  - Urination
  - Defecation
  - Death
Diagnosis of OP Toxicosis

- Brain acetylcholine esterase levels
  - Expressed as percent depression of normal eg. 45% of normal
- Demonstration of OP or carbamate in rumen contents, liver, blood, feed
- Some carbmates break down rapidly in tissues

Polioencephalomalacia (PEM)

PEM Causes

- Generally ruminants resistant due to ruminal microflora production of thiamin
- Plants high in thiaminases
  - Bracken fern, Horse tail, Kochia
- High concentrate diets – flora that produce thiaminases
- High sulfur in water sources and plants
  - Thiamin analog produced in rumen with excess sulfur
- Less commonly
  - Lead poisoning
  - Water deprevation
Musculoskeletal

- Injection site lesions
- Injuries
- Infectious arthritis
- Infectious myositis
- Ionophore Toxicosis
- Laminitis
- Toe abscesses

Injection Site Lesions
Injuries

- Fractures
- Torn ligaments and tendons
- Nerve Damage
  - radial nerve
  - chute injury?
- Abraded toes
- Injection sites

Infectious Causes of Arthritis

- *Histophilus somni*
  - with meningoencephalitis (TEME) and pneumonia
- *Mycoplasma bovis*
- *Arcanobacter pyogenes*
  - often secondary infection or opportunistic infection, ascending infection
Otitis Media / Interna

- *Mycoplasma bovis* most common
- Others
  - *Mannheimia haemolytica, Pasteurella multocida, Histophilus somni*
- Diagnosis usually on clinical signs
  - Head tilt
  - Drooping ear(s)
  - Exudate draining from ear canal
Bacterial Myocarditis

- *Histophilus somni*
- *Salmonella sp.*
- *Staphylococcus sp.*
- *Streptococcus sp.*
Blackleg
- Clostridium chauvi
- Clostridium novii
- Clostridium sortellii

Ionophore Toxicosis
- Feed mixing errors
**Diagnosis of Ionophore Toxicosis**
- History
- Histologic lesions
- Demonstration of ionophore in rumen contents
- Difficult to determine what level in rumen is considered significant
- Dilution, distribution, duration etc.
- Concentration in ration

**Ionophore Toxicosis**
- Monensin
- Lasalocid
- Mechanism
  - Ionophore-induced Na⁺ influx
  - Admits toxic levels of extracellular Ca²⁺ to the cytoplasm of cells with excitable membranes

**Ionophore Toxicosis**
- Clinical Signs
  - Heart Failure
  - Sudden Death
  - Respiratory Distress

**Infectious causes of “Foot Rot”**
- *Fusobacterium necrophorum*
- *Bacteriodes spp.*
  - Infection in soft tissues
  - Interdigital space
  - Often associated with wet conditions
Laminitis
- Inflammation of laminae in the claw
- Usually due to ruminal acidosis
- Result of release of vasoactive substances
- Reduced or interrupted blood supply
- Inflammation and permanent damage in blood vessels
- Two - three weeks after pneumonia

Laminitis
- Acidosis
- Trauma – high impact foot strike, crushing corium
- Injury to blood vessels in foot
- Vasoactive substances
- Formation of vascular shunts as a result of vasculitis
- Altered blood supply to laminae

Laminitis
- Ruminal Acidosis
  - “dyspnea” - metabolic acidosis
  - mistaken for pneumonia
Laminitis
- Separation of hoof from laminae
- Rotation of distal phalanx (coffin bone)
- Abnormal growth of hoof - “snow ski”
- Double sole
- Growth interruption ridges

Abridged Toes - Toe Abscesses
- Rough surfaces
  - Trucks with too little sand
  - Rough concrete
  - Processing areas
  - Alleys
  - “Pushing” cattle too hard

Abridged Toe
- Seemingly unpredictable
  - But can be increased incidence within groups
- Predisposing factors
  - Prior episodes of laminitis?
  - Wet surfaces - slippery
  - Soft feet - wet environment
  - Hoof color - hoof hardness? white softer?

Toe Abscesses
- Trauma - impact
- Secondary to toe abrasions
- Ascending infections
  - Through compromised white line
  - Environmental bacteria

Toe Abscesses - Mechanism
- Toe abrasions - weakens the white line
- White line separation
- Opens hoof to infection
Treatment

- Early recognition!
- Establish drainage
- Antimicrobials - as per feedlot veterinarian
- Dry environment, if possible