Bovine neurological disease

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WCVM

Bovine neuro cases are hard…….

- Often lacking specific history
- Classic neurological exam not really possible
- Limited diagnostic tests readily available
- There are some important disease you don’t want to miss

Presenting signs

- Blindness
- Recumbency
- Circling/head tilt
- (change in behaviour)

Neurological examination

- Mental status
- Cranial nerves
- Gait and posture
- Spinal reflexes
- Pain perception

Supportive diagnostic tests

- CBC
- Chemistry
- CSF
- Lead

Another useful diagnostic tool

- Time

- Rate of disease progression and response to treatment can be very helpful

- E.g.
- Rabies rapid progression to death (less than 3-5 days)
- BSE very, very slow progression (months)

2 common identifiers

- Blindness

- Circling/head tilt

Blindness

- Lead
- Polio
- Vitamin A
- Pituitary abscess

Lead

- Mainly batteries and paint
  - Also lubricants, roofing, lead shot

- Studies indicate 4-12% of the herd may be affected but asymptomatic despite levels >0.35ppm (safe consumption 0.05ppm)

Lead poisoning in Cattle

- Common

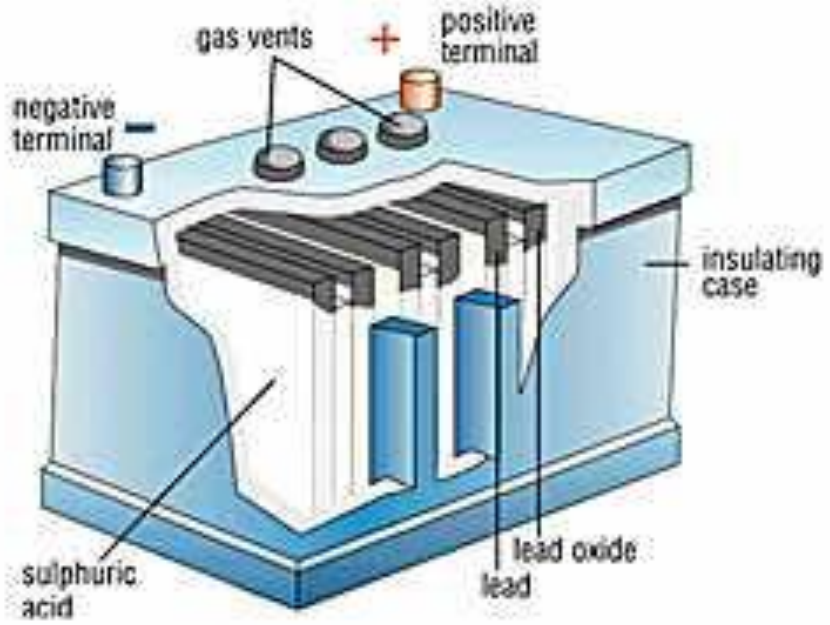
- Signalment
  - Any animal, any age (calves may be slightly more common)
  - Any bizarre behaviour patterns

Lead Poisoning
Distant Examination

- Ataxia (drunkenness)
- Weakness (recumbency)
- Convulsions
- Aimless wandering
- Headpressing
- Opisthotonus
- Teeth grinding
- Bellowing
- Hyperasthesia

Sources of lead

- Car batteries



Sources of lead

- Lead paint
  - White and green

Other sources of lead

- Crank case oil

- In all cases the owner will deny that there is any possible that the animals could have been exposed to lead

General Physical Examination

- Disruption of normal GI function
  - Decreased rumination
  - Mild bloat
  - Tenesmus
  - Constipation or diarrhea
- Blindness

Pathophysiology

- Acute vs Chronic exposure

- Acute is much more common
  - 400-600mg/kg in calves (2oz)
  - 600-800mg/kg in adults (1/2 lb)

Confirming Diagnosis

- Blood lead levels
  - Must use heparinized blood sample
  - Blood levels can vary widely in 24 hours
    - Normal 0.05-0.25ppm (death at about 1ppm)

  - High powered radiographs of the reticulum may indicate elemental lead

Confirming Diagnosis

- Post-mortem examination
  - Lead in reticulum and rumen
  - Check lead levels in;
    - Kidney
    - Liver

To treat or not to treat

1. Remove access to lead
2. Chelation therapy
    1. Ca-EDTA to promote excretion
3. Thiamine
4. Rumenotomy

Problems of the treated cow

- When is it safe to eat?

Polioencephalomalacia

- Aka:
  - PEM
  - Polio
  - Cerebrocorticalnecrosis – CCN
- This disease should really be split in two
  - Thiamine deficiency
  - Sulfate toxicity

Polio
Distant examination

- Blindness
- Ataxia (drunkenness)
- Tremors
- Recumbency
- Convulsions
- Opisthotonus (star gazing)

Polio - Pathophysiology

- Thiamine is normally made in rumen
- Needed for pentose phosphate pathway of glycolysis
  - Main energy producing pathway in the brain

Thiamine deficiency

- Thiamine antagonists
  - Amprolium – used for coccidiosis

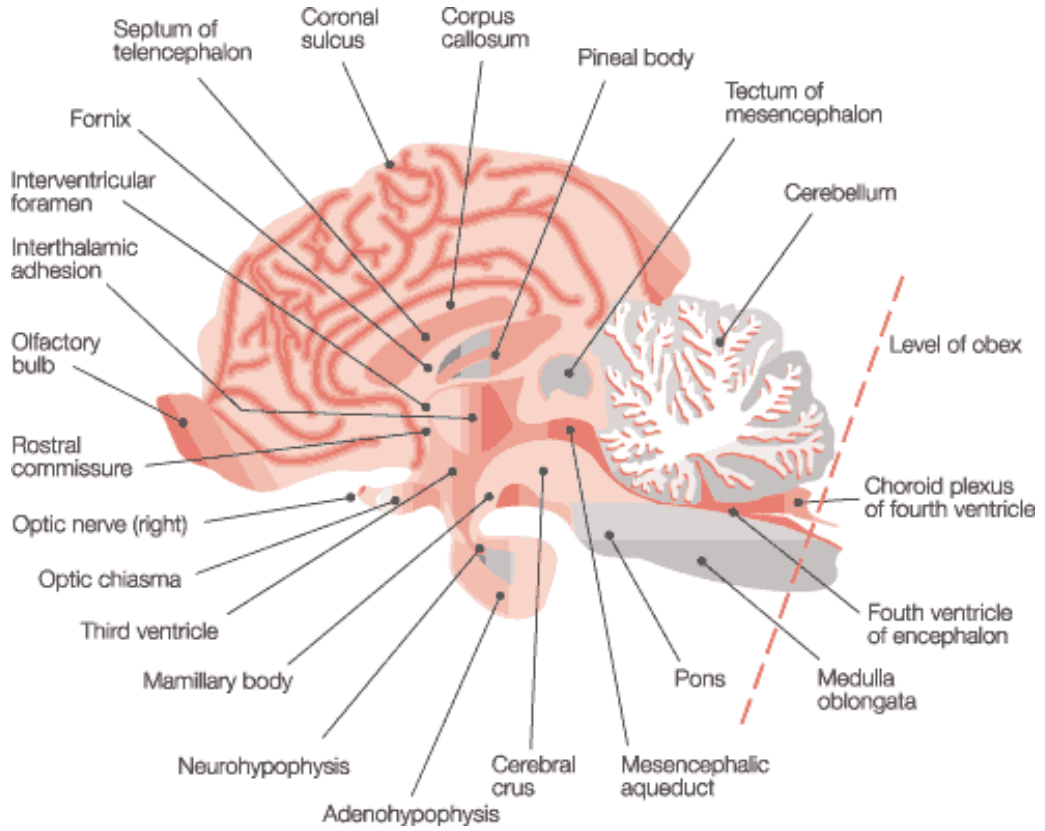
- Rumen thiaminases
  - Produced by bacterial fermentation
    - *Bacillus thiamainolyticus*
    - *Clostridium sporogenes*
  - Ingested pre-formed
    - Bracken fern

Diagnosing Polio

- Signs are essentially similar to lead poisoning
- GI motility is normally OK
- Pupillary light reflex is maintained

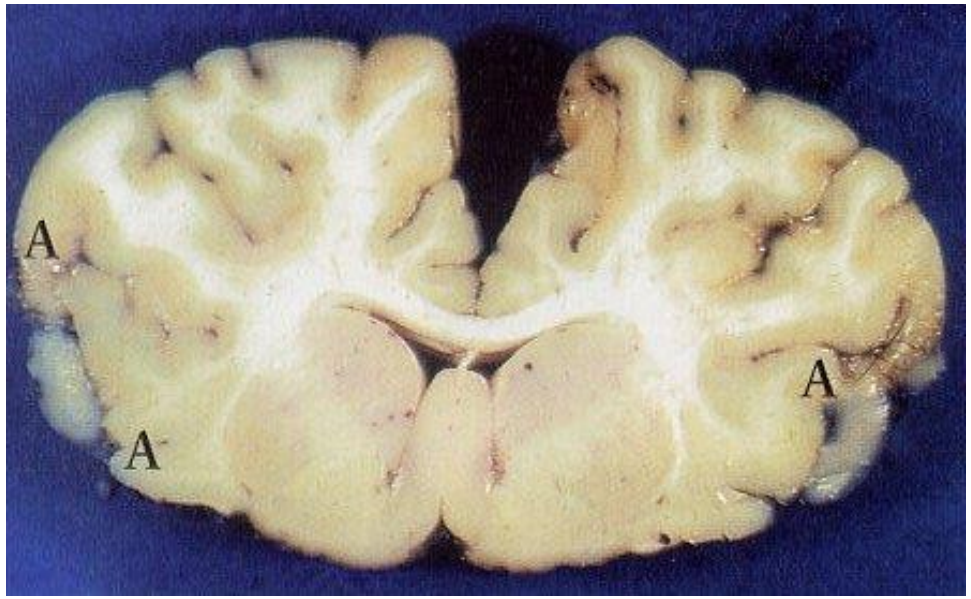
Blindness in polio

- Pupillary light reflex
  - Simple reflex in the brain stem
- Menace response
  - Learned response using higher brain function



Finalizing the diagnosis

- No simple lab tests
  - Erythrocyte transketolase
- PM examination of the brain
  - Cerebral edema
  - Mild yellow discolouration
- Fluoresces under UV light



Treatment of Polio

- Give thiamine (lots of it)!
- 10mg/kg IV then 10mg/kg IM every 3 hours for 5 treatments
- Prognosis good if caught early

Sulfate toxicity

- Syndrome cannot be distinguished clinically or at PM from thiamine deficiency

Sources of sulfate

- Water
  - Slough water may be particularly bad especially in drought years
  - Easily tested
  - The exact cut off is not clear
    - 2000-7000ppm is the region

Sources of sulfate

- Feed
  - Hay and grain grown in high sulfate areas will contain more sulfate
  - Certain plants store sulfate
    - Kochia
    - Canadian thistle

Sources of sulfate

- Just when you think you have it straight

- Copper and Molybdenum interfere with sulfate absorption and may be protective

How sulfate is toxic

- Not entirely clear

- Best theory
  - Sulfate is converted to H_2S in the rumen. This is eructated and inhaled, absorbed by the lung and transported to the brain in the blood, where it exerts a toxic effect

Sulfate toxicity

- No definitive clinical diagnosis
- Thiamine treatment does not work

Vitamin A deficiency

- Actually more than one syndrome to consider
  - Adult cattle on scrub pastures
  - Feedlot steers
  - Neonatal calves

Presenting signs

- Night blindness – rarely recognized
- Blindness – esp feedlot steers
- Weak animals dying shortly after birth
- Reproductive problems
- Poor hair coat
- Paralysis
- Convulsions

History and epidemiology

- Vitamin A is easily produced from β-carotene
- Carotenes are freely available in any green coloured feed

Syndromes recognized

- Neonates
  - Vitamin A crosses the placenta poorly
  - Calves are heavily dependent on colostrum

- Pastured animals
  - Takes months for adults to develop signs due to good liver stores

Syndromes recognized

- Feedlot animals 6-12 months age

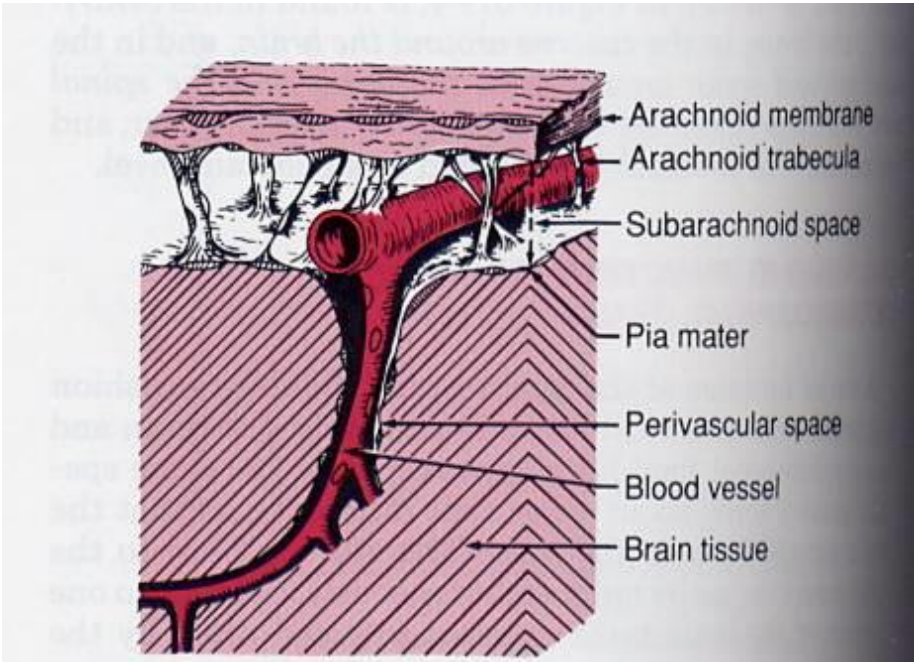
- Toxicity of chlorinated napthalenes
  - Block production of vitamin A

Pathophysiology of Vitamin A deficiency

- Vitamin A required for:
  - Maintenance of epithelial surfaces
    - loss of secretory capacity
    - replacement with a keratinized epithelium esp respiratory and UGT, cornea (xerophthalimia)
  - Thickened arachnoid results in decreased CSF absorption
  - Production of retinol – night blindness

Neurological signs

- Increased CSF pressure due to:
  - Thickened arachnoid
  - Failure to remodel bone as the animal grows
    - Also puts pressure on cranial nerves esp II



Diagnosis

- Blindness
  - Careful optho exam should reveal papilledema
- Convulsions and paralysis
- Measure CSF pressures with a direct manometer
- Response to therapy
- Measure plasma Vitamin A levels

Diagnosis (2)

- Liver biopsy

- Diet calculations
  - Look at the diet for green coloured feeds
  - Signs typically seen with grains, straw, poor quality hay

Treatment

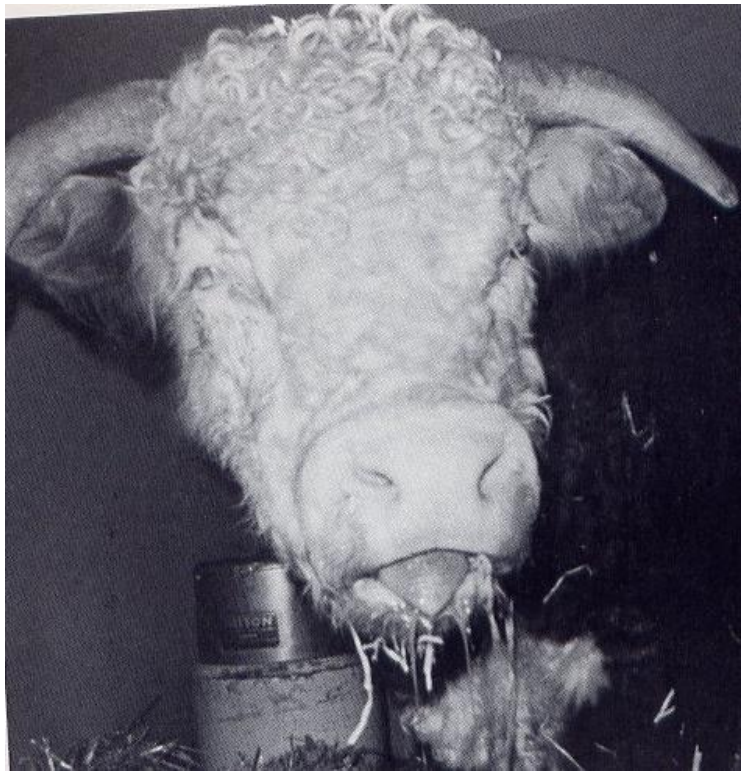
- Injectable Vitamin A

- Prognosis is good for calves with improvement with 48 hours

Pituitary Abscess

- Bacteria can easily localize in the region of the pituitary

- Open mouth
- Protruding tongue
- Blindness
- Decreased milk production



Pituitary abscess

- Treatment
  - Prolonged antibiotics
  - Prognosis
    - poor

Circling

- Listeria
- Middle ear disease

Listeria

- Classic presentation
  - Head tilt
  - Circling
  - Unilateral Facial Paralysis

Listeria

- *Listeria moncytogenes*
- Saphrophyte
- Mainly found in poor quality silage

Listeria

- Bacteria enters at gingiva
- ascends to the brain stem through CN-V
- Micro-abscess formation
  - CN VII and VIII

- Treatment
  - Prolonged antibiotics
    - Penicillin
    - Oxytetracycline

Middle ear disease

- Calf dz. assc. With weaning
- *Histophillus, mannheimia* and *mycoplasma* (esp. dairy calves)

- Dairy calves 2-8 wks age
- More common in the winter

- True incidence unknown as mild cases are often missed

- Middle ear disease can occur in catastrophic outbreaks with morbidity up to 80-100%
- Associated with cross contamination in bottle raised animals
- Associated with URT infections
- Colonization of the pharynx ascending the eustachian tube
  - Rupture of the tympanic membrane
  - Infection of inner ear and osteomyelitis and meningitis

Clinical signs

- Facial nerve paralysis and head tilt/circling

- Purulent aural discharge in many cases
- Head rubbing, head shaking – ear trauma

- Confirm diagnosis with a deep ear swab
  - Otoscope, imaging

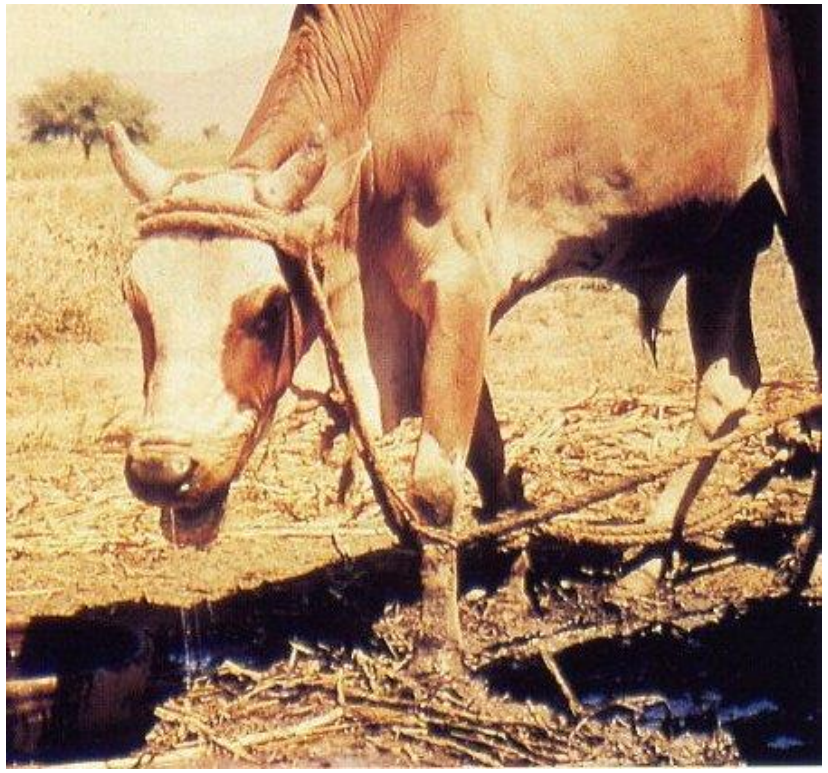
Treatment

- Antibiotic therapy
  - Limited evidence – best choices appear to be tulathramycin and enrofloxacin for at least 2 weeks

- Prognosis
  - Better in young calves with early treatment
  - May have persistent signs after treatment

Rabies

- Clinical signs
  - Salivation
  - Bellowing
  - Aggressiveness
  - Paresis/paralysis



Epidemiology

- Vector
  - Skunk
  - Raccoon
- Incubation period
  - 2 weeks – months
- Clinical course
  - 5 days

What to do if you suspect rabies

- Reportable disease
  - Contact Manitoba Ag
  - Contact public health if human exposure
- Isolate and observe animal
- After death or euthanasia
  - Get brain and give to Manitoba Ag.

BSE

- Signs
  - Nervousness
  - Aggression
  - Ataxia
- Slowly progressive to recumbency and death
- Much easier to diagnose in a dairy cow!

What does BSE look like?

Tetanus

- A disease of antiquity that is coming back!

- Many multivalent clostridial vaccines no longer contain tetanus
  - Combined with the use of banding for castration this a bad combination

- Signs
  - Muscle rigidity – often most noticeable in the hind limbs
    - Typically history of surgery, multiple animals affected, lack of vaccination Hx
- Treatment
  - none

Conclusion

- Neurological disease in the bovine is challenging
- There are only a few “real” differentials of consequence
  - Separating them out is not impossible

  - Using clinical signs
  - Diagnostic tests
  - Time
  - Response to treatment

Questions