Diseases of the alimentary tract

For AAU CVMA 4th year DVM students

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Digestive system

- Its primary functions are Prehension, Mastication, Ingestion, Digestion and Absorption of nutrients

- Equine teeth are

✓ constantly erupting throughout the animal's life

✓ equine jaws move in a lateral motion, simultaneously grinding the food and wearing down the teeth.

Terminologies

- **Occlusal surface**

✓ The surface of the tooth in contact with the food (and other teeth).

✓ This is large in size in cheek teeth and thin in incisors.

- **Apical**
- refers to the area of tooth farthest away from the occlusal surface, i.e. the area where the roots later develop and is the opposite of occlusal

Coronal: direction toward the occlusal surface

Terminologies…

- **Lingual surface**

✓ Tongue-side (medial aspect)

- **palatal**

refers to the same aspect (lingual) of the upper cheek teeth.

- **Buccal surface**

✓ Cheek-side (lateral aspect)

- **labial**

✓ surface toward lips of canine and incisors

Terminologies…

- **Mesial**
- refer to the surfaces of teeth that face toward an imaginary line between the central incisors

- **Distal**
- refer to the surfaces of teeth that face away from an imaginary line between the central incisors

- **Proximal:** surface contacting next tooth in sequence in the same arcade.

3 pairs of incisors (central, lateral and corner)
1 pair of canines (usually only in males)
3 pairs of premolars on upper and lower jaws (occasionally a vestigial small 1st premolar ‘wolf tooth’)
3 pairs of molars on each jaw

Types of teeth

- Adult mammals have four types of teeth, termed incisors, canines, premolars (PM) and molars (M), in a rostro-caudal order

- **Incisor**
- are specialized for the prehension and cutting of food
- help in estimating the age of equids

- the eruption dates and the changes in appearance of the occlusal surfaces are the main criteria to estimate age.

Types of teeth

- **Canine**
- Male horses normally have four permanent canine teeth
- erupt at 4–6 years of age in the interdental space
- The lower canines are more rostrally positioned than the upper and thus there is no occlusal contact between them.
- Canines do not continually erupt like cheek teeth and thus long reserve crowns can be present in older horses

Types of teeth

- **premolars**
- **First premolar (‘wolf tooth’)**
- One or both of the upper first premolar and less commonly, the lower can be present as the **small, vestigial** ‘wolf teeth.’
- normally lie immediately in front of the second premolar.
- sometimes displaced rostrally or rostrolaterally
- may be angulated (i.e. not vertical in relation to the hard palate).
- Usually erupt at 6–12 months of age and they do not have a deciduous precursor

Types of teeth…

- **The cheek teeth**
- are large quadrilaterals, nearly rectangular in cross-section except the 2nd PM and last molar which are approximately triangular
- The maxillary cheek teeth are wider in the buccolingual direction than the mandibulars.
- Dental overgrowths can often be prominent
- The maxillary arcades are 23% wider than the mandibular arcades.
- When the mouth is closed and the incisors are aligned, the maxillary arcades laterally overlap the mandibulars

Numbering

- **Triadan system of dental nomenclature**
- arcades are given a first number and teeth are given an additional number.
- Ex: 208, the number ‘2’ or ‘200’ refers to the arcade in which the tooth resides, and the ‘8’ refers to the tooth itself.
- Arcades are numbered beginning with number 1 at the horse’s upper right.
- The sequence is continued in a clockwise direction when observing the horse from the front.
- The upper left is number 2, lower left is number 3, and lower right is number 4.
-
- Teeth are numbered 1 through 11 on each arcade beginning with the central incisor as tooth number 1 and continuing distally in each arcade to the last molar which is numbered 11

Age estimation

- The deciduous incisors are smaller and whiter than the permanent teeth.

- The approximate age of eruption of the deciduous incisors in horse:

- Central—6 days
- Lateral—6 weeks
- Corner—6 months

Permanent dentition		
Central	2 1/2yr	
Lateral	3 1/2yr	
Corner	4 1/2yr	
Canine	4–5 yr	

Galvayne’s groove on the labial surface of the upper third incisor

appear at the gingival margin at 10 years of age.
At about 15 years of age, the groove’s margin is at the midpoint of the occlusal crown.
By 20 years, it reaches the occlusal border.
By 25 the apical margin of the groove is at the crown’s midpoint, and
By 30 the groove disappears.

The disappearance of infundibulum

- The infundibulum is a cone-shaped ring of enamel extending apically from the occlusal surface.
- It is filled to a variable degree with cementum, and is referred to by the common name of the ‘cup’.
- disappearance from the lower
- 1s = 6 years;
- 2s= 7 years;
- 3s = 8 years.
- There is much individual variation

	Age of deciduous teeth eruption		Age of permanent teeth eruption	
	Horse	Donkey	Horse	Donkey
Central incisor	0-1 week	0-2 weeks	2.5 years	3-3.5 years
Middle incisor	4-6 weeks	8 weeks	3.5 years	4 years
Corner incisor	6-9 months	12 months	4.5 years	5-5.5 years

Dental examination

- The dental examination and dental procedures must be performed in an area safe for the practitioner, the assistant, and the horse.

- The area must be free of objects
-
- Ensure that children and pets are not in the area
-
- Safety should be kept in the front of the mind of the attending veterinarian at all times.

Dental examination

- constant attention to the mood of the horse being examined is imperative.
- Learn to watch for signs of fear, anger, or frustration and to recognize softening and relaxation.
- If the horse does not relax or at least tolerate your initial examination, sedate the horse for the safety of yourself, your assistant, and the horse.
- Sedation is more humane for the horse and more reassuring for the owner than physical restraint.

Dental examination

- Dental abnormalities are underrecognized but common problem in equine

- Safety should be kept in the front of the mind of the attending veterinarian at all times

- Once the veterinarian has a sense for the safety of

✓ the patient,

✓ its surroundings, and

✓ handlers, the physical examination can continue.

Dental examination

- A typical examination starts with a brief history
- carry out a thorough clinical examination to assess all body systems
- After performing the basic physical examination, a specific and detailed evaluation of the head and mouth is carried out.

- **External evaluation includes**

✓ The range of motion of the jaw is viewed and its grinding sound and vibration are evaluated.

✓ the cheek overlying sharp enamel points of the buccal aspect of the maxillary cheek teeth

Dental examination

- ✓ alignment of the incisors and their manner of occlusion,
- ✓ shape, symmetry and obvious abnormalities of the head .
- ✓ any lumps, enlargements, or depressions in the region of the teeth.
- ✓ Nasal passages are evaluated for discharge and any abnormal odors are noted.
- ✓ pain elicited on palpation or movement.

Clinical signs associated with dental diseases

- Losing weight in spite of good appet
- Failure to gain weight
- Dribbling grain
- Obvious chewing abnormalities
- Accumulating wads of grass or hay between the buccal gingiva and the cheek teeth (quidding)
- Soaking hay in water before eating it

Clinical signs…

- Drooling
- Foul breath
- Excessive whole grain particles in feces
- Roughage particles longer than 1/4 inch in feces
- Discharge from nostrils
- Fistulous discharge from the jaw or face
- Swelling on the lower jaw or face

Sharp enamel points

– buccal (lateral) and
– lingual (medial) overgrowths

These are a very common abnormality associated with equine teeth worldwide

The hard points of enamel on the buccal surface of the upper (maxillary) teeth and

the lingual side of the lower (mandibular) cheek teeth become very sharp often lacerating the cheeks and tongue.

Sharp enamel points…

- tight nosebands and head-collars contribute to ulcers and calluses in the mouth

- Sharp enamel can
- affecting the ease with which equids can **grind food** (and hence affecting digestion capabilities and thus the nutritional value of feed),

- produce painful sores and ulceration of the mucosa lining the cheeks and tongue === >eating painful ==== > weight loss.

Rostral and caudal overgrowths of cheek teeth (hooks)

- Rostral hooks (106 and 206) and caudal hooks (311 and 411) – these beak-like occlusal overgrowths develop as a result of incomplete occlusal contact

- Hooks 106 and 206 may cause oral pain, bit pressure points and buccal surface calluses or ulcers.

- Hooks on the rostral aspect 06 are easily seen and rasped in a good dental examination;

Hooks

- hooks on the caudal aspect of the mandibular cheek tooth (11) are often missed and it is these which can cause the most pain
- Large hooks on 311 and 411 may cause penetration of the palatine mucosa, ulcer formation and in rare cases, laceration of the palatine artery.
- **floating**
- reduction of sharp edges and enamel points.
- The lingual edges of lower cheek teeth and buccal edges of upper cheek teeth are rounded
- For large dental overgrowths, gradually reduce over a number of examinations rather than in one go.
- a patient with severely excessive reduction of buccal cusps.

Diastema

- Incisors

✓ cause food impaction b/n the incisors

✓ Continued retention causes sever gingivitis == > gingival recession and even incisor loosening

- **Treatment**
- Widening the occlusal aspect and brush the back and

front of the teeth with a toothbrush twice weekly

Diastema

- Diastema of the cheek teeth

- **Etiology and pathogenesis**
- Congenial or acquired
- ✓ Inadequate rostro-caudal angulation of the cheek teeth
- ✓ Dental buds with normal angulation developing too far apart
- ✓ Disparity between the jaw size and the size of the cheek teeth at eruption === > overcrowding of dental buds== > teeth displacement == > secondary diastema b/n normal and displaced check teeth

Diastema

- Senile diastema commonly develop b/n cheek teeth in geriatric horse due to narrow reserve crown teeth
- Secondary to acquired check teeth displacements (medial, lateral)
- Tooth loss
- Cheek teeth ‘shifting’ in a rostro-caudal direction with dental overgrowth (overgrowth and caudal displacement of 11, overgrowth and rostral displacement of the 6
- The presence of supernumerary cheek teeth

Diastema

Pathogenesis

Diastema== > impaction of long fibers of food b/n CT and periodontal space == > food pocketing == > stretching and inflammation of the periodontal ligaments == > sever pain

- Secondary anaerobic bacteria infection exacerbates periodontal disease
- Destruction of the periodontal fibers may delay dental eruption in affected teeth and may result in the development of wave-mouth

Diastema..

- **Clinical signs**
- Diastema can sometimes be asymptomatic when it is very wide (>1cm) or with very small diastema where the feed is trapped b/n the teeth but does not reach the gingiva.
- Halitosis
- Quidding
- Buccal food packing
- Difficulty in chewing long rather than short (<5mm) fibers
- Chew very slowly and not make normal loud noises during mastication

Diastema..

- Preferentially chew on one side of their mouth
- Hold their head in abnormal positions when eating
- Weight loss
- Prone to large colon impaction
- Oesophageal obstruction (occasional)
- Undigested grains or long strands of forage

Diastema..

- **Treatment**
- **Dietary modification**

✓ Eliminating food containing long fibers (hay/silage etc) and substituting with short fibers (5mm) food

- Reduction of exaggerated transverse ridges that develop opposite the diastemata if present

- Reduction of the occlusal surface crown height on either side of the diastema by 2 to 5 mm and similarly reducing the height of the opposing teeth at he same site

Diastema..

•Tooth extraction (single diastema, diastema secondary to displacement

•widening the diastema reduces or eliminates the entrapment of food and allows any entrapped food to more easily exist

- If diastemata have developed secondary to dental overgrowth, removal of the causative overgrowths and removal of impacted food
- cleaning out the periodontal pockets of all impacted food and then irrigating them fully.

Diastema..

- The periodontal pockets are then filled with plastic impression material and possibly with a layer of antibiotis.

- Euthanasia if widespread diastema with deep food pocketing or if sever osteomyelitis

Esophageal obstruction

- **Etiology**
- Obstruction or “choke” is the most common of the esophageal disorders
- Common **predisposing factors** include feed changes, poor dentition, trauma, inadequate water intake ,foreign bodies, and ingestion of dry, coarse feed items (dry grains, ears of corn and pelleted feeds).

- **Clinical findings**
- Dysphagia, coughing, nasal regurgitation of food material, water and saliva from the mouth and nostrils, repeated attempts at ingestion, extension of the head and neck to facilitate swallowing, painful swallowing, head-tossing and other signs of agitation and frustration and cervical swelling.

Diagnosis

- clinical signs
- attempt to pass a nasogastric tube
- Palpation of the neck

❖ Crepitation and diffuse cervical enlargement may denote cellulitis resulting from perforation of the esophageal wall.

❖ Thoracic auscultation and ultrasound should be performed to ascertain and monitor the development of aspiration pneumonia.

❖ Endoscopy and radiography are helpful for definitive diagnosis

Treatment

- Prompt therapy is vital to successful management of esophageal impactions
- Access to food and water should be prevented
- Nasal intubation with gentle warm water lavage under xylazine sedation is successful in relieving most simple impactions.
- External massage may increase the efficacy of lavage.
- If this is unsuccessful, the animal should be muzzled to prevent food or water intake and administered balanced, isotonic IV fluids.

Treatment…

- After rehydration of the patient, sedation and lavage should be repeated.

❖ Excessively aggressive lavage can result in severe aspiration or esophageal damage and/or perforation.

❖ Attempting to remove the impaction by applying pressure with a nasogastric tube often lodges the impaction in the narrower, more distal esophagus or causes perforation of the esophagus

Treatment…

- Management following relief;
- up 24 -48hrs = withhold feed
- 1–7 days frequent interval, small volume, soft, moist feed (Eg ; grass)
- Feeding of hay and other dry feeds **should not be resumed** until it is felt that the esophagus has healed and returned to normal function.
- Fresh water should be available at all times and electrolyte abnormalities corrected with oral or IV solutions

Treatment…

- Broad-spectrum antimicrobial therapy should be instituted for at least 7 days for aspiration pneumonia.
- Long-term antimicrobial therapy is necessary in cases in which significant lung damage due to aspiration has occurred.
- Any underlying problems (e.g. management and dentition) should be corrected
- surgical intervention: Impactions that do not respond to conservative therapy should be definitively identified, localized and relieved by surgery

chronic weight loss

- Etiologies
- numerous and varied
- gastrointestinal diseases are among the most common causes of chronic

- Obtain a thorough history about the animal's husbandry, nutrition, workload and any previous health problems.

- Conduct a clinical examination to identify the underlying cause(s) and treat appropriately.

Debility…

- ENERGY INTAKE < ENERGY OUTPUT = WEIGHT LOSS AND DEBILITY

- When this imbalance is prolonged, body reserves of fat and muscle are used to provide energy, resulting in a thin, weak animal.

Debility…

- Causes of reduced energy intake

❖ Insufficient or poor quality food provided (most common)

❖ Inability to take in food properly, eg. overgrown teeth, sore mouth

❖ Inability to digest food properly, eg. dehydration, diarrhoea, worms

❖ Inability to metabolise food properly, eg. liver disease

Debility…

- Causes of excessive energy output

❖Overwork (most common)

❖Concurrent disease, pain or fever

❖Cold environment leading to excessive loss of body heat

❖ Lameness

Principles of management of thin equids

- Identify underlying cause
- Often both health and management issues are involved so it is important to identify all issues
- a blood sample should be taken to look for signs of anaemia and blood parasites.
- Discuss improvements which could be made to the energy content of the animal's diet, using locally available feed, to ensure adequate quality and quantity.
- Emphasise that reducing the animal's workload is essential if it is to regain condition.

Diarrhea

- Diarrhea is a common, and sometimes fatal, clinical problem of adult horses and foals.

- A number of specific causes for acute and chronic diarrhea have been identified

- **History and physical examination**
- acute or chronic, dietary changes, deworming program, involvement of single versus multiple animals, exposure to sand

Diarrhea

- the use of medications (especially antibiotics and NSAID), Other concurrent diseases, stress, possible exposure to toxins, weight loss, water consumption, and salt availability also may be significant.

- Clinical findings
- BCS
- the presence of any edema should be noted.
- fever, dehydration, or signs of endotoxemia
- Visible abdominal distention

Diarrhoea…

- Cause may not always be identified.
- Treatment of clinical signs should be implemented without delay.
- treatment should aim to reduce systemic and intestinal inflammation (NSAIDs) and then to promote intestinal mucosal repair
- Response to treatment should be monitored.

Diarrhoea…

- **Fluid therapy in diarrhoea**
- In mild to moderate cases of diarrhoea, fluid therapy via a nasogastric tube is considered an appropriate method of fluid therapy.

✓ it is readily available
✓ practical,
✓ cheap
✓ avoids the risk of thrombophlebitis associated with venous catheterisation
❖ Mucosal inflammation, often found in cases of diarrhoea, can compromise mucosal integrity and function, which in turn can affect the absorption of water and electrolytes.

Diarrhea

- ***Acute diarrhoea***
- ***Causes***
- Salmonellosis
- Clostridiosis
- Antibiotic-associated diarrhea
- NSAIDs toxicity
- equine monocytic ehrlichiosis

Diarrhea

- **Chronic Diarrhea**
- Chronic salmonellosis
- Parasitism (strongylosis, cyathostomosis
- NSAIDs
- Neoplasms
- Sand…

Salmonellosis

- can occur sporadically or as outbreaks in stables, barns and veterinary hospitals
- Equids of all ages can be affected although younger animals seem more susceptible.
- Stress is often indicated as an underlying factor in outbreaks e.g. transportation, GI disease, changes in feeding patterns, high temperatures and antibiotic therapy.

Salmonellosis

- **Etiology**
- Salmonella spp. of bacteria.

- Saimonella typhimurium is the isolate most commonly associated with equine diarrhea

- **Clinical findings**.

- The enteric form in the adult may be

✓ asymptomatic,
✓ mild,
✓ acute severe, or
✓ chronic.

Salmonellosis

- **Mild infections**

✓ Soft faeces with intermittent bouts of diarrhoea

✓ persistent weight loss

✓ May have fever or poor appetite.

Salmonellosis

Acute severe infections

- ✓ fever and depression is seen during the first 24-48 hours.
- ✓ mild to severe abdominal pain (the condition can be confused with a surgical colic).
- ✓ Diarrhea begins sometime after the initial signs but may take 2-4 days to develop.
- ✓ Diarrhea is projectile, foul smelling, and persistent.
- ✓ Expression of diarrhea often is accompanied by improvement in the other clinical signs.
- ✓ Horses usually continue to eat, but in the case of anorexic animals, the prognosis for survival is poor.

Salmonellosis...

- **chronic**
- The diarrhea may persist for 3-4 weeks, at which time horses will have experienced significant weight loss.
- reappearing in times of stress
- Ventral edema caused by hypoproteinemia also may be a finding.
- Laminitis is a frequent sequela to salmonellosis.
- **A peracute form**
- affected horses die within 6-1 2 hours.

Salmonellosis...

- **Diagnosis**
- The diagnosis is based on clinical findings supported by laboratory confirmation.
- **Loboratory tests**
- a neutropenia with a left shift and varying degrees of cellular morphologic changes (toxicity).
- The albumin fraction of the TP is low,
- the total protein may be elevated or normal due to dehydration
- The PCV is elevated due to dehydration,
- a metabolic acidosis with electrolyte losses through the feces.

Salmonellosis...

- **Fecal culture**
- may be unrewarding because of the dilution effect of diarrhea and the adherent nature of the bacteria to the intestinal mucosa.
- A rectal mucosal biopsy may enhance the likelihood of culturing the organism.

- **Differential diagnoses**

✓ intestinal clostridiosis,

✓ antibiotic-induced diarrhea,

✓ small intestinal obstruction.

Salmonellosis…

- **Treatment**

❖ primary consideration is fluid replacement therapy, with large volumes of alkalinizing fluids.

❖ Antibiotics should only be proposed in severe cases of septicaemia and for immune-compromised animals with careful selection of a narrow spectrum bactericidal agent

✓ If antibiotics are used in the enteric form of the disease, those with gram-negative specificity are recommended (e.g., gentamicin, amikacin, trimethoprim-sulfa combinations).

Salmonellosis...

- Flunixin meglumine is recommended for its anti-inflammatory effect.
- Bismuth subsalicylate is recommended as an intestinal protectant and anti- prostaglandin.
- Plasma transfusions may be necessary in hypoproteinemic horses.
- Heparin may be used in cases of coagulopathies [e.g., disseminated intravascular coagulation (DIC)] associated with the disease.

Salmonellosis…

- **Prevention**
- animals with diarrhea should be isolated in a separate barn if possible.
- Care- takers should wear dedicated and protective clothing.
- A foot bath should be used at the entrance to the facility.
- Manure from cases should be handled and disposed of in a secure and separate way.
- Salmonella species are **zoonotic.**

Intestinal clostridiosis

- Clostridiosis is an important cause of acute enterocolitis in foals and adult horses
- **Cause**

- *Clostridium difficile*
- *C. perfringens*

Intestinal clostridiosis

- Clinical Signs
- Equine intestinal clostridiosis is clinically similar to other forms of acute enterocolitis
- the clinical course is usually acute,
- peracute colitis with rapid death may occur.
- Occasionally, a milder, more prolonged clinical course occurs.
- fever, anorexia, and depression before the onset of gastrointestinal signs,
- more commonly no prodromal signs are apparent.
- Signs of endotoxemia and shock may accompany acute signs of colic and severe, dehydrating diarrhea.

Intestinal clostridiosis

- **Diagnosis**
- Laboratory tests
- C. perfringens counts may be performed on the feces.

- **Therapeutic**
- Massive fluid therapy is essential for any hope of success.
- Antibiotics are of little value, but penicillins may be employed as a logical choice for antibacterial therapy.

Antibiotic-associated enteritis

- Nearly all antibiotics used in horses have been reported to cause diarrhoea
- Tetracycline is the antibiotic most often incriminated
- lincomycin, tylosin, and high doses of penicillin and erythromycin also have been associated with the disease.
- There also have been reports of diarrhea after the use of trimethoprim-sulfa drugs.

Antibiotic

- **Clinical findings.**
- Diarrhoea starts 2–6 days after antibiotics are first administered.
- Signs may subside rapidly when the antibiotics are discontinued.

- **Treatment**
- Discontinue the antibiotic use,
- Give dry forage diet and supportive care

GI tumours

- This should be considered in older equids with chronic, non-progressive diarrhoea which does not respond to treatment.

- The most common form is intestinal lymphosarcoma, although mesenteric lipomas are also common in older animals.

GI tumours

- **Clinical signs**
- Persistent weight loss
- Diarrhoea
- colic, and oedema of the ventral abdomen and limbs as, over time, the protein-losing enteropathy leads to hypoproteinaemia.
- Contrary to other causes of chronic diarrhoea, the animal may or may not have a good appetite or fever.

- **Diagnosis**
- Clinical signs and a lack of response to therapy.
- A rectal examination may reveal enlarged mesenteric lymph nodes, or the tumour itself, and a haemogram or abdominal paracentesis may show increased numbers of lymphocytes.

GI tumours

- **Treatment**
- None available which is curative.

- Supportive therapy (rest, good quality feed, and pain relief if required) is indicated until the animal is too debilitated to work.

- euthanasia

GRAIN OVERLOAD

- Etiology

- The consumption of readily fermentable carbohydrates causes colitis.
- Some starches are poorly digested in the small intestine of the horse.

Grain overload

- The highly fermentable starch === > fermented in the cecum and large colon by Lactobacillus === > form lactic acid in place of volatile fatty acids === > reduces the pH and increases the osmolality of colonic contents=== > damaging the mucosal cells & Death of the Gram-negative bacteria === > release of endotoxins === > inflammation and absorption the toxins across the damaged mucosa === >endotoxemia.

Grain overload

- Changes in the concentration of volatile fatty acids in the colonic contents alter colonic motility.

- The osmotic changes, mucosal necrosis and inflammation, endotoxemia and altered motility result in diarrhea.

Grain overload

- **Clinical findings**
- vary with the quantity of fermentable feed ingested.
- The most common signs to be noted are

✓ variable degrees of abdominal pain and diarrhea.

- In more severe cases,

✓ hyperemic and/or cyanotic mucous membranes,

✓ fever or subnormal temperature resulting from shock,

✓ sweating and

✓ tachycardia develop.

Grain overload

- Laboratory findings include
- leukopenia resulting from endotoxemia,
- increased PCV and total plasma protein due to dehydration,
- metabolic acidosis resulting from shock and hypoperfusion, and renal or prerenal azotemia.
- Laminitis is a common complication.

Grain overload

- **Diagnosis**
- history of exposure to quantities of concentrate, lush pasture or legume to which the horse is not accustomed

- physical examination and laboratory findings and

Grain overload

- **Treatment and prognosis**
- Therapy is most rewarding when administered before the onset of clinical signs.

- Mineral oil (1-2L/250kg BW) is administered to decrease fermentation and to facilitate evacuation of colonic contents.

- Fluid therapy and other supportive therapies are recommended

Grain overload

- Frog supports should be applied due to the high incidence of laminitis.

- The prognosis is guarded to poor due to the common occurrence of laminitis in cases of grain overload.

Colic

- Abdominal pain
- Is a common problem in equine practice
- most respond to analgesic therapy alone
- In more serious cases, early detection and referral for intensive therapy or surgical intervention can dramatically improve prognosis and outcome.
- **Etiology**

➢ intestinal (true colic)

➢ non-intestinal (False colic)

COLIC…

- **False colic**

✓ Myophaty

✓ Azoturia

✓ Tying up

✓ Laminitis

✓ Uterine torsion

✓ Sever bleeding ….

COLIC

- **True colic**

✓ **Large colon Impaction**

- Usually occurs at pelvic flexure

✓ large colon displacement

- Reno-splenic entrapment

✓ Spasmodic colic

✓ Large colon volvulus

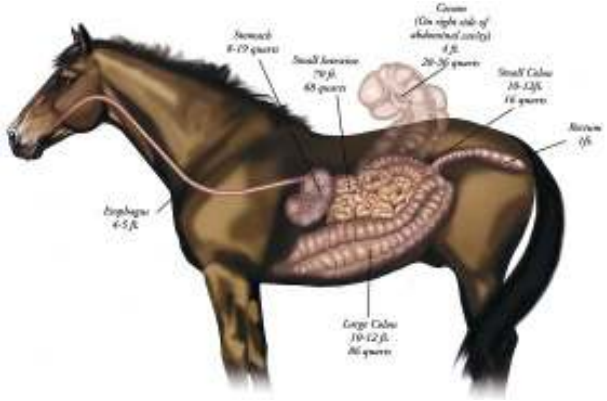
✓ Lipoma

✓ Strangulation

✓ Enteritis

✓ Peritonitis

✓ Verminous arteritis



Impaction

- Intestines become blocked and feed cannot pass

- **Cause**
- Undigested feed staff
- Foreign materials
- Sand
- Worms

❖ Most common impaction site

✓ Pelvic flexure

❖ Impaction usually responds to treatment

Colic examination

- A thorough history with emphasis on the husbandry, previous episodes of illness, duration, severity of present condition
- general appearance and attitude
- abdominal pain minimally responsive to analgesics more often have a lesion requiring surgery
- Equine with intermittent pain have a better chance of responding to medical therapy.

Colic examination …

- TPR

✓ reassess intermittently to evaluate the progression of the disease.

- **Temperature**
- The temperature is usually normal

- elevated temp. (overexertion, enteritis or colitis)

- *subnormal* temp. (hypovolemic or endotoxic shock)

Colic examination …

- **Heart rate**
- Elevated pulse indicates pain, dehydration or enterotoxaemia.
- A rising heart rate is an adverse sign.
- A heart rate remaining high after analgesia is also an adverse sign.

- **Respiratory rate;** a high rate also indicates pain

Colic examination …

- mucous membrane color, moistness and capillary refill time skin turgor and temperature of extremities should be assessed as markers of circulating volume and hydration status.

- Tachycardia, poor pulse pressure, cool extremities and dry mucous membranes with a prolonged capillary refill time reflect significant volume contraction and the need for IV fluid therapy.

Colic examination …

- **Evaluation of the gastrointestinal tract**

✓ auscultation and percussion of the abdomen,

✓ nasogastric decompression,

✓ rectal examination,

✓ abdominal radiography,

✓ abdominal ultrasonography

✓ abdominocentesis.

Auscultation

- It is important to listen for several minutes to allow a full cycle of contractions of the bowel to occur.
- Increased abdominal sounds may be heard in horses with impending colitis or enteritis.
- It may indicate spasmodic colic
- **An absence of gastrointestinal sounds**
- Seen in impaction colic may be caused by administration of a sedative,

Ascultation

- Absence of gut sounds, particularly after analgesia, is an adverse sign. It may imply that surgery is needed
- The presence of gut sounds that then disappear is an adverse sign.
- Auscultation of the thorax is important to rule out thoracic disease in horses that are exhibiting signs of colic.
- **Rectal examination**
- can offer important information in colic case.
- Assess fecal out put and characteristics of the feces = no or little fecal output in impaction colic

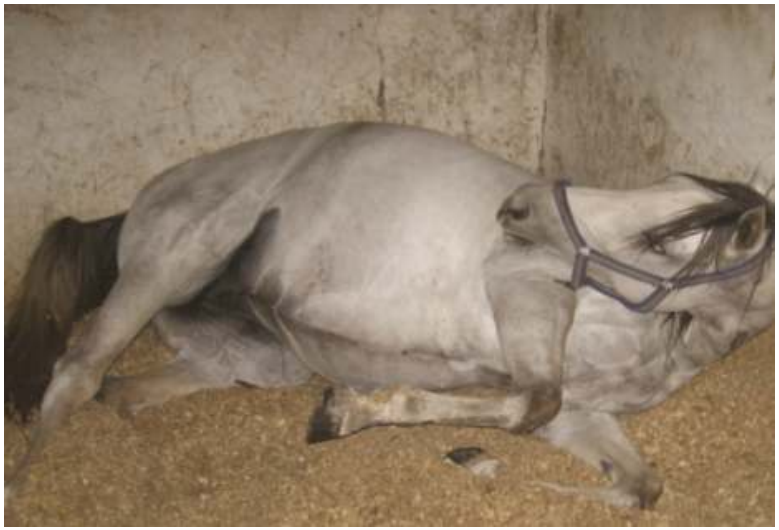
Rectal examination

- Abnormalities noted on abdominal palpation per rectum can include
- ✓ distended or edematous small intestine,
- ✓ small intestinal impactions,
- ✓ large colon distention, impaction or edema,
- ✓ displacements of the large colon,
- ✓ space-occupying masses (abscesses, neoplasia)
- ✓ abnormalities of the urogenital tract (uterine torsion or hemorrhage into the broad ligament),
- ✓ inguinal herniation, and
- ✓ free abdominal gas associated with visceral rupture.

- **Nasogastric Intubation**
- Nasogastric intubation should be performed on all equine presented for colic.

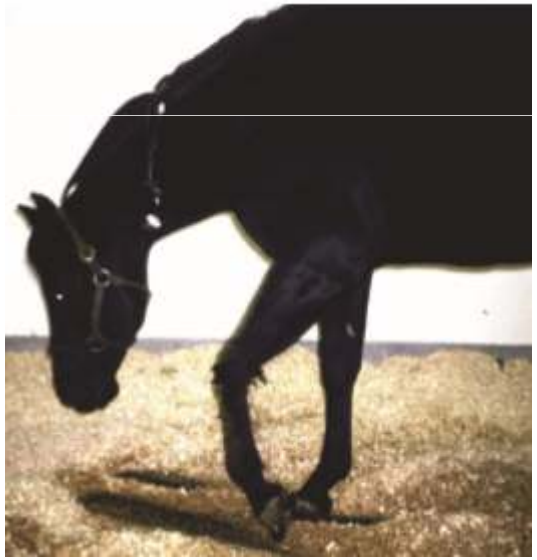
- Assess gastric reflex (>2lit in horse is significant)
- When there is gastric dilation it may prevent gastric rupture and save the life of the horse.
- Do not add more if there is GR
- **Abdominocentesis** should be performed in most cases of colic, particularly referral cases and those that are persistent or recurrent.

Signs of colic

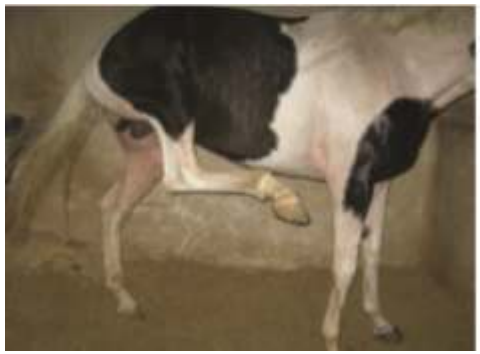


- looking at the flank region

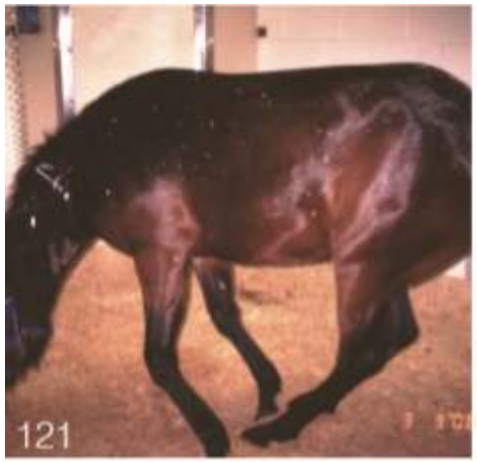
- Pawing

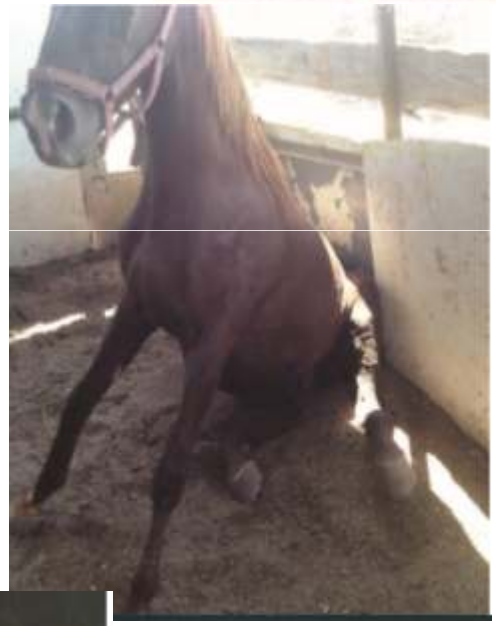


- kicking at their abdomen



- Frequent lying
- dog sitting position





- Sweatin

- Rolling

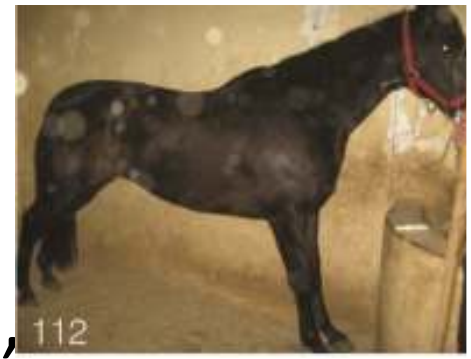
- Abdominal distention

- self inflicted trauma

- a “stretched out” posture

✓straining to defecate

✓Frequent small amounts of urination,



Treatment

- Medical (impaction, inflammation)
- Surgical (sever impaction, displaced colon)

✓ Condition worsens over time

✓ Careful with pain relief medication, may mask the condition

✓ The earlier the surgery the better

- ***Analgesia***
- Often the most immediate problem is control of abdominal pain.
- **Flunixin meglumine is most** commonly used for its analgesic and anti-endotoxin properties

Treatment

- Failure to relieve abdominal pain with flunixin meglumine suggests a more serious disease requiring surgery.
- NB: potential toxic side effects of NSAIDs in the hypovolemic patient
- Decompression of the stomach by nasogastric intubation *relieves pain and helps prevent gastric rupture.*
- Cecal tympany *due to obstruction of the large and/or small colon can* be relieved by cecal decompression.

Medical Treatment…

- *Fluid therapy*
- Severely dehydrated horses, especially those requiring surgical intervention, require rapid volume replacement with a balanced polyionic IV solution.

- **Lubrication** of the tract eases transport.
- Mineral oil 2–4L/450kg

- Mineral oil is not good at penetration of firmly impacted material such as sand and can be passed around the impaction.

- Its use is contraindicated in horses with gastrointestinal reflux

Medical Treatment…

- **Walking**

✓ can increase motility and decrease pain, but is best used in conjunction with other therapy

✓ Excessive walking can exhaust the horse and prevent ongoing treatment with other therapeutics.

- ***Nutrition***
- Horses with colic of undetermined origin should be held off feed until the primary problem is corrected.

Medical Treatment…

- Horses with large colon or cecal impactions may be allowed oral fluids but should not be fed until the impaction is passed.

- Gastrointestinal reflux indicates small intestinal ileus *and addition of* food or water will only add to the gastric distension and discomfort.

Medical Treatment…

- ***Prognosis and referral***
- Horses with progressive, non-responsive abdominal pain, cardiovascular deterioration (dehydration, tachycardia, poor perfusion), distended small intestine or significant volumes of gastric reflux *should be referred to a surgical facility* for further evaluation.

- simple medical colics usually resolve in 2–4 h, referral should also be considered in patients whose pain persists beyond this time interval.

- Early surgical intervention in cases with strangulation obstruction improves prognosis.

Colic prevention

- Feed at regular interval
- ✓ More frequent meals better
- ✓ Do not allow to eat a lot at a time
- ✓ Provide water frequently especially in hot climte
- Ensure quality of feed
- ✓ Avoid feeding mouldy hay, too much dry straw
- Exercise regularly
- Deworm
- Rasp teeth regularly
- Avoid sudden changes of diet

Colic in donkeys

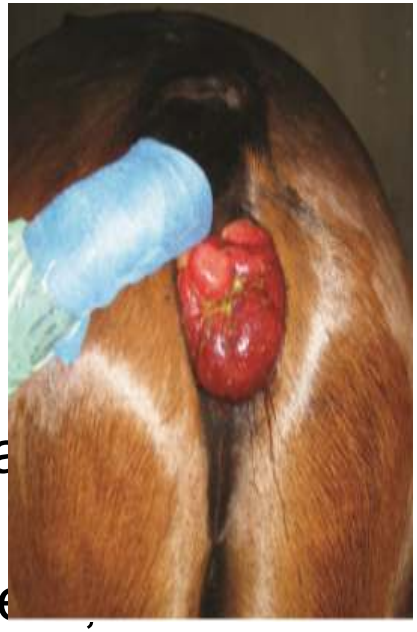
- Donkeys show few overt signs of pain
- Donkey with pain

✓ Stand with its head lowered

✓ Lie down or not respond as normal

❖ Pain has only a relatively minor effect on the donkey's HR. the rate is influenced much more by haemoconcentration, endotoxemia

Rectal prolapse



- **Cause**
- chronic diarrhoea associated with infection or pa
- foreign bodies or feed impaction due to poor tee dehydration, or following parturition
- overloading (the animal strains in the hindquarters to move the load)
- prolonged recumbency from other illnesses.
- the presence of Gasterophilus nasalis.

Rectal prolapse

- **Treatment**
- replacing the prolapse

- Treat the underlying cause (colic symptoms, pain and infection)

❖ Many cases of prolapse are recurring and, unless the underlying cause is corrected, the animal's welfare will be severely compromised with repeated attempts at replacement.

Rectal prolapse

- Provide analgesia and anti-inflammatory drugs.

- **How do you replace the prolapse?**
- adequately restrain the animal. (sedation and, ideally, epidural anaesthesia, particularly if the animal is still straining a lot or distressed)

- Tie back the tail and clean any faeces from the tail and perineal area.

- Wash the prolapse gently and remove as much necrotic tissue as possible.

Rectal prolapse

- Applying table sugar will help decrease any oedema and allow for easier replacement back into the rectum.

- Gently push the rectal tissue back into the rectum manually, avoiding excessive pressure which could tear the delicate rectal mucosa; use lubrication to aid the process.

Rectal prolapse

- Many rectal prolapses can be resolved through manual correction and do not require suturing. If this is not possible then a suture can be placed.
- Ensure that the animal can pass droppings and that sutures are removed.
- Infuse local anaesthetic into the area first with a 24G needle.
-
- Using a large needle and thick suture material make a ‘purse-string’ suture (a single, continuous suture pattern with needle insertion at 4–6 points in a full circle around the rectum).

Rectal prolapse

- Gently pull the two ends of the suture so that the rectum closes.

- Leave a space large enough to insert 2–3 fingers into the hole (so the rectum will not prolapse back through the hole, but that soft faeces and gas can still escape)

Rectal prolapse

- Pass a nasogastric tube and give liquid paraffin or mineral oil as this lubricates the faeces to allow for easy passage once the suture is removed.

- If parasites are suspected to be the underlying cause, do not give anthelmintics until the sutures are removed and faeces are passing normally, as this could cause recurrence of the prolapse or colic.

Rectal prolapse

- Important:

- If the prolapse has been replaced and held in place with sutures, ensure that the sutures are removed after 48 hours (72 at most).

- Leaving in sutures will ultimately lead to the death of the animal, which is far worse than the original prolapse

Rectal prolapse

- The animal will require several days of soft laxative feed, plenty of water, and a reduced workload for the next week to ensure the prolapse does not recur.

- Always remember to remove the sutures.

Rectal prolapse

- careful monitoring of the animal is required and analgesia and anti-inflammatory drugs administered.

- Prevention:
- The underlying cause must be found and treated or repeated prolapse may occur.

GIT parasites

- **The abundance of parasite larvae and eggs in the external environment**
- This varies according to ambient temperature and humidity so that there is variation with season and also geographical region.

- In temperate climates, the highest numbers of larvae on pasture usually occur in late summer or early autumn.

- Pasture larvae and eggs survive best in wet, mild conditions but the larvae die quickly in dry, hot weather.

GIT parasites

- Both eggs and larvae are fairly resilient to frosty conditions.

- Ascarid eggs (the infective stage) are particularly adapted to survive for prolonged periods of many months (even years) in the external environment.

- The numbers of pasture eggs and larvae are affected by the levels of worm egg output by grazing animals, and this is intrinsically related to the intensity of the adult worm burden

GIT parasites

- **The number of parasites of one species within an individual animal**

- This varies with
- the level of pasture contamination
- host immunity: ascarid infections
- individual propensity to infection: there is natural predisposition of certain individuals to parasite infection.

GIT parasites

- **Management** of the horses
- The likely exposure to parasite infection via contaminated pasture will be affected by
- the grazing practices and
- the parasite control program applied on the pasture, and also
- on any premises on which the animal(s) were kept previously

GIT parasites

- **Strongyles**
- The strongyles, synonym 'red worms’

- Live as adults in the LI

- Categorized as small and large strongyles

GIT parasites…

❖Large strongyles,

- The common large strongyle spp
- *Strongylus vulgaris,*
- *S. edentatus,*
- *S. equinus,*

GIT parasites

- **The essential features of the large strongyles**

✓ a direct life cycle

✓ migratory

✓ a pre-patent period of 6-10 months

✓ all stages are susceptible to modern anthelmintics.

GIT parasites…

- **Clinical Signs**
- appear slowly
- Anemia
- Emaciation
- poor coat and poor performance are frequently attributed to large strongyles

- S. vulgaris associated with colic, intermittent lameness in rear limbs,

GIT parasites…

- **Diagnosis:**

- History (deworming history)
- Clinical presentation (Colic, ill thrift and weight loss)
- Fecal examination (Epg), but larvae are required for specific identification.

- **Control:**
- Avoid overstocking and overgrazing.
- Practice pasture rotation and mixed grazing.
- Proper stacking of manure to heat will destroy many infective forms.

GIT parasites…

- The dry season is not favourable for the development and survival of the parasite

- Animals acquire infection from pasture during and immediately following wet seasons.

GIT parasites…

- ❖ Small strongyles/ cyathostomins
- The essential features include
- ✓ A direct LC
- ✓ non-migratory
- ✓ a pre-patent period of 6-20 weeks
- ✓ Over 50 spp have been reported
- ✓ the larvae are able to go into a dormant state & encyst in the epithelial cells of LI (for as long as 2-3 years)
- ✓ arrested larvae are poorly susceptible to modern anthelmintics

GIT parasites…

- **Acute verminous enteritis/ larval cyathostomosis**
- **Clinical signs**
- Abdominal pain, Anorexia, Depression and hyperlipaemia
-
- Sever diarrhoea (death due to toxic shock may occur before diarrhoea develops)

GIT parasites…

- Second form in horse

- Diarrhoea is not a feature
- Sign of ill thrift and weight loss
- Occurs in grazing season

GIT parasites…

- Parascaris equorum.

- **Essential features**
- a direct, migratory (gut-liver-lung-
- a pre-patent period of 3 months
- the adult stages live in small intes
- prolific egg producers
- adult and luminal larval stages are susceptible to modern anthelmintics
- migrating larval stages have low susceptibility to modern anthelmintics.

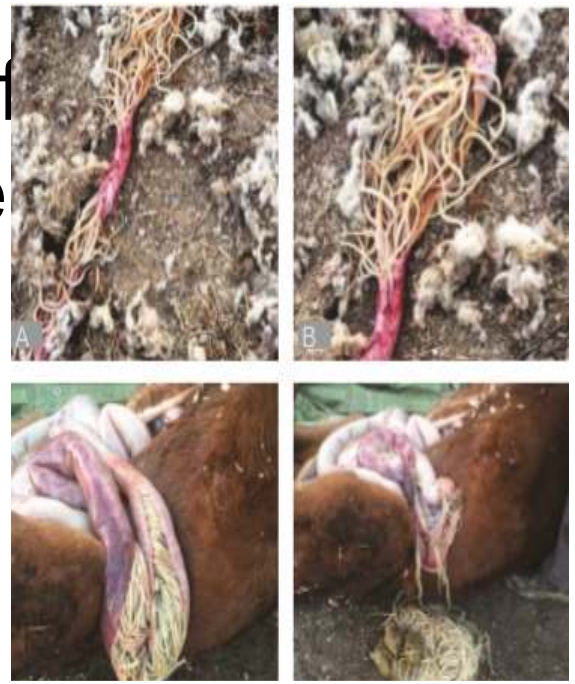
GIT parasites…

- **Clinical Signs**
- Young animals chiefly affected.
- Clinical signs are most frequently seen in animals 2-4 months of age.
- coughing and elevated temperature (Large numbers of larvae migrating through the lungs) .
- Adult worms are associated with a chronic catarrhal enteritis and diarrhea of varying intensity.
- General debility and weakness may occur.

GIT parasites…

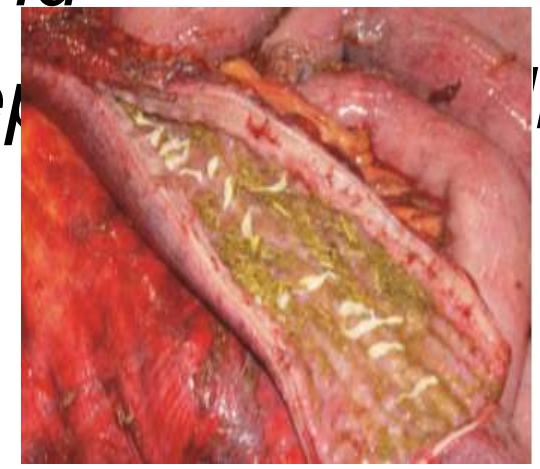
- In massive intestinal infections, **obstructive colic** with intussusception may occur.

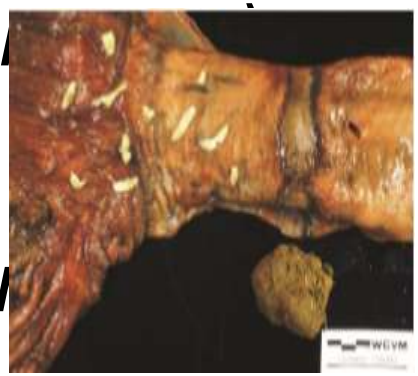
- Bile duct obstruction and perf
  small intestine with a fatal pe
  been reported.



GIT parasites…

- **Tapeworms**
- The three species of tapeworm that affect equine are
- *Anoplocephala perfoliata (co*
- *A. magna, and*
- *Paranoploce* *lla*





GIT parasites…

- Essential features
- an indirect life cycle with the oribatid mite as the intermediate host
- a pre-patent period of 6-10 weeks
- the adult stages are either cecal (A. perfoliata) or small intestinal or stomach (A. magna and P. mammillana);

GIT parasites…

- Incriminated as a cause of
- intestinal intussusception
- Caecal perforation leading to peritonitis
- Intestinal obstruction
- Colic

- The severity and depth of ulcerative lesions of the mucosa increase as the number of tapeworms attached in the area increases

GIT parasites…

- **Diagnosis**
- Fecal examination
- Serology

- **Treatment**
- Pyrantel
- Praziquantel

Diseases and disorders of the respiratory tract for AAU CVMA 4th year DVM Students

Reta Tesfaye (DVM, MTADM)

2020

Anatomy and physiology…

- **Nostrils**
- The nostrils has flexible margins
- The alar cartilages support the dorsal and lateral margins of the nostrils.
- The *alar fold divides the nostril* into a dorsal and ventral part.

- **Dorsal part**: leads to a blind-ending pouch known as the *false nostril or nasal diverticulum.*
-
- **The ventral part** of the nostril leads into the nasal cavity. It is the path for nasogastric tube & Endoscope introduction

Anatomy…

- **The nasal cavity**
- Separated into 2 by the nasal septum.
- It is covered with a highly vascular mucosa.
- The septum extends along the whole length of the hard palate so that each nasal cavity communicates with the pharynx via a separate opening (*choana).*

- ***Nasal turbinates***
- Each side of the horse's nasal cavity has two turbinates that divide the cavity into 3 air passages, the ventral, middle and dorsal meatuses.

Anatomy…

- The ventral meatus, is the primary path for an endoscope or stomach tube.
- The function of the turbinates is to warm, moisten and clean inhaled air before it reaches the trachea.
- **The paranasal sinuses**
- are extensive air-filled cavities within the skull.
- The sinuses communicate with the nasal cavity via relatively small openings
- The largest and clinically most important sinuses are the *caudal and rostral maxillary sinuses and* the *frontal sinus.*

The Pharynx

- The pharynx delivers air from the posterior nasal cavity to the larynx and is also the pathway that delivers food from the oral cavity to the esophagus.
- In equine, the oral cavity and pharynx are normally separated by the soft palate except during swallowing.
- For this reason, equids are obligate nasal breathers.

The Larynx

- The *larynx is the gatekeeper to the entrance of* the trachea.
- It prevents inhalation of food into the lower airway during swallowing
- It maintains a rigid, boxlike shape via a series of paired and unpaired cartilages that are moved relative to one another by striated laryngeal muscles.

The trachea

- The trachea is a tube transporting air from the larynx to the lungs.
- It can be palpated immediately beneath the skin on the ventral midline of the neck.
- divides into two smaller *bronchi within the thorax.*
- The middle layer of the trachea is composed of incomplete rings of cartilage joined together by sheets of elastic connective tissue.
- They prevent the trachea from collapsing and allow for adjustments in length when the neck is extended.

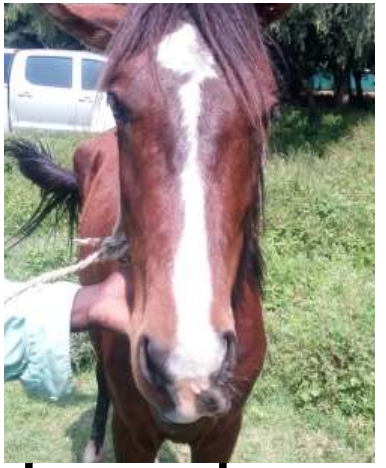
alveoli

- Gas exchange occurs in the alveolar ducts and alveoli, both of which are lined by an extensive pulmonary capillary network so that there is a huge vascular surface area for oxygen and carbon dioxide diffusion.

Clinical Examination of the Respiratory Tract

- **Signalment and History**
- Signalment and a thorough case history provide essential diagnostic information, because factors such as age, environment, diet, exercise activity, vaccination history, Previous &current medications, and transportation history can be important risk factors for particular respiratory diseases.

Clinical Examination...



- **Nostrils and nasal cavity**
- The nasal area is normally dry, except for a few drops of serous fluid, which consists mainly of tears.
- The nares should be assessed for symmetry;

✓ asymmetry indicates the presence of disorders such as wry nose, facial paralysis, or injuries.

- **Dilation**

✓ In dyspnea the nostrils will fully dilate during inspiration, and in severe dyspnea, the nostrils may remain permanently dilated at rest.

Clinical Examination...

- **Inability to dilate**

✓ unilateral or bilateral inability of the nostrils to dilate= nostril paralysis and consequently may have asymmetry of the muzzle

- **Inflamation**

✓ Inflammation of the nasal mucosa will lead to mucosal edema, erythema, and variable volumes of purulent to mucopurulent exudate.

- **Ulceration**

✓ Marked ulceration of the nasal mucosa is characteristic of diseases such as epizootic lymphadenitis in horses.

Clinical Examination...

- **Flow of air**

✓ Under normal condition the flow of air from each nostril is approximately equal .

✓ **A unilateral obstruction** in a nasal cavity, such as that caused by a large sinus cyst, will reduce or stop the airflow from the ipsilateral nostril.

- **Odor of breath**
- Malodorous breath can occur in cases of sinusitis (especially, but not exclusively, dental sinusitis), nasal mycosis, guttural pouch mycosis, and gangrenous pneumonia.

Clinical Examination...

- **Nasal discharge**
- Nasal discharges can be characterized as being

✓ **continuous or intermittent**,

✓ **scant or profuse**

✓ **malodorous.**

- **Unilateral nasal** discharge occurs in disorders of the nasal cavity and paranasal sinuses.

- A small proportion of horses with pulmonary disease inexplicably have unilateral or predominantly unilateral nasal discharge.

Clinical Examination...

- Lesions of the respiratory tract caudal to the nasal septum typically result in bilateral nasal discharge.

- **Bilateral nasal discharge**
- is usually indicative of pulmonary disease,
- The nature of any discharge can further be categorized as serous; mucoid; mucopurulent; hemorrhagic; and containing food or gastrointestinal contents.

Clinical Examination...

- Serous nasal discharge is observed in horses with viral respiratory infections and allergic rhinitis.

- Mucoid to purulent discharge indicates increasing evidence of a primary or secondary bacterial respiratory infection.

Clinical Examination...

- **dysphagia**

✓ Pharyngeal or esophageal dysphagia may lead to nasal discharge that contains food.

- This food and saliva will flow down both nasal cavities, especially when the head is lowered, usually within a minute or so after the ingestion of food or liquids.

✓ neuromuscular dysfunction (general or local)

✓ Acute pharyngitis

Clinical Examination...

- ***Epistaxis***
- Epistaxis denotes hemorrhage from the nose, but does not indicate the origin of the hemorrhage.
- Unilateral epistaxis suggests a lesion rostral to the nasopharynx, while bilateral epistaxis indicates a lesion caudal to the nasal cavities.
- Fresh blood may originate from the URT (guttural pouch mycosis, trauma) or LRT (EIPH).
- Brown (old hemorrhage), mucoid, malodorous discharge occurs in horses with necrotizing pneumonia and ruptured pulmonary abscess, both of which carry a poor prognosis.

Submandibular lymph node

- The submandibular lymph node groups are usually not palpable or are barely palpable in healthy horses.

- Even when these lymph nodes are enlarged, they are often not readily detected unless the dorsomedial aspect of the more caudal aspects of the mandible is specifically examined for their presence.

Submandibular lymph node

- ipsilateral Submandibular lymphadenopathy + unilateral nasal discharge = a unilateral upper respiratory tract disorder.
- Bilateral submandibular lymphadenopathy is

✓ occasionally the result of bilateral UR disorders (bilateral sinusitis or guttural pouch disorders)

✓ most commonly a response to generalized respiratory infections, such as equine influ herpesviruses 1 and 4, or strangles.

Examination of the paranasal sinuses

- Diseases of the paranasal sinuses often cause
- ✓ unilateral nasal discharge,
- ✓ submandibular lymph node enlargement, and
- ✓ epiphora.

- The thick lateral maxillary wall is not usually distorted in primary and dental sinusitis, unless gross sinus distension and bone softening occur.

paranasal sinuses...

- the thin medial walls are readily distorted towards the nasal septum, leading to unilateral nasal obstruction.
- Percussion of a sinus may be of value in assessing its contents. An exudate-filled sinus may lose the resonance of a normal air-filled sinus.
- A diseased sinus is not necessarily filled with exudate , affected horses usually resent percussion of an inflamed and presumably painful sinus.

paranasal sinuses...

- Assess the maxillary bones for pain, swelling, increased heat and softening of the bones.
- With low-grade sinusitis all clinical examinations are unreliable.
- Therefore, if sinusitis is suspected the diagnosis should be confirmed by radiography, endoscopy, or sinoscopy

Nasopharynx

- Pharyngeal dysphagia in the horse is manifested by

✓ food and saliva flowing down the nasal cavity, during or immediately after eating;

✓ coughing caused by food inhalation; and

✓ evidence of froth and masticated food in the horse's water bucket.

Guttural pouches

- The guttural pouches (GP) are positioned dorsal to the nasopharynx and medial to the mandible and parotid salivary glands.
- they cannot be visualized or palpated in the normal animals.
- endoscopy is the technique of choice for examining the guttural pouches.
- GP distension may be evident in case of guttural pouch tympany, and empyema or chondroids.

Trachea

- The cervical portion of the trachea is examined by inspection of the overlying skin to identify changes in shape or position, deformities, and scars.
- Palpation may detect pain, local swellings, deformities from a previous tracheostomy, and developmental defects such as dorsoventral or lateral collapse, and of the free borders of some tracheal rings.
- Mild digital pressure on the trachea may elicit coughing in horses with tracheitis.

Clinical Examination...

- **Abnormal Respiratory Sounds**
- listen for respiratory sounds which are audible without the aid of a stethoscope.
- Their presence usually indicates RT disease.
- Cough is a forced exhalation that clears mucus and foreign material from the airways.
- is a common sign of respiratory disease and a reflex pulmonary defense mechanism.

Clinical Examination...

- **Stimuli of Cough**
- Cough may be stimulated by

✓ airway smooth muscle contraction (bronchoconstriction),

✓ excessive mucus production,

✓ presence of inhaled particles in the airways,

✓ release of inflammatory mediators (infectious diseases),

Clinical Examination...

- ✓ exposure to cold or hot air,
- ✓ intramural or extramural pressure or tension on the airways (tumor, granuloma, abscess, or decreased pulmonary compliance caused by restrictive disease such as interstitial fibrosis or pleuritis),
- ✓ sloughing of airway epithelial cells, and enhanced epithelial permeability (pulmonary edema)

Abnormal respiratory sound

- **Wheezes and crackles**
- Wheezes and crackles may be heard at the nostrils of horses with severe RT disease.
- **Stridor**
- is a term used to describe a particularly loud monophonic inspiratory wheeze that may be heard considerable distances from the horse.
- It is indicative of an extrathoracic airway obstruction.
- **Grunts or groans**
- These loud expiratory sounds, produced by sudden laryngeal opening after a period of breath-holding against a closed glottis, are usually an indication of pain.

Clinical Examination of the Lower Respiratory Tract

- **Audiovisual inspection of breathing**
- The clinician should determine

✓ the rate,

✓ depth, and

✓ pattern of breathing, and

✓ listen for abnormal sounds associated with breathing.

- This is best performed by distant observation, with the horse in a quiet location and undisturbed, because anxiety can profoundly alter the breathing pattern and rate.

Clinical Examination...

- Breathing rate

SPP	Breath/Min
Horses	8-12
Ponies	15-20
Young Donkey	16-68
Adult Donkey	13-31
Mule	12-20

Abnormalities in the rate, depth, and pattern of breathing

- Abnormalities in the rate, depth, and pattern of breathing may be subdivided into five broad categories

- Rapid deep breathing

✓ Physiological: exercise and anxiety

✓ Pathological: lung disease, anemia, metabolic acidosis, pain

- Rapid shallow breathing:

✓ chest wall injury or pleuropneumonia.

✓ interstitial pneumonia and pulmonary fibrosis

Abnormalities in the rate, depth, and pattern of breathing

- Slow deep breathing

✓ severe airway obstruction

- Slow shallow breathing

✓ central nervous system depression, or

✓ A compensatory response to metabolic alkalosis

Rhinitis

- characterized clinically by

➢ Sneezing

➢ wheezing, and stertor during inspiration

➢ a nasal discharge that may be serous, mucoid, or purulent in consistency depending on the cause.

- Rhinitis in equine may be caused by

❖ viral infection

✓ Equine influenza virus, EHV-1, and EHV-4, Equine Rhinitis Viruses, Equine adenovirus

❖ bacterial Rhinitis is not common and usually occurs secondary to trauma or foreign body.

Rhinitis

- ❖ Fungal infection *(such as Aspergillus fumigatus, Cryptococcus neoformans or Rhinosporidium seeberi),* or

- ❖ Immune-mediated processes such as allergy.

- usually occurs in conjunction with inflammation of other parts of the respiratory tract.

Disorders of the Paranasal Sinuses

- **Causes**

✓ primary bacterial or mycotic infections
✓ Secondary to dental disease
✓ facial trauma,
✓ sinus cysts,
✓ progressive ethmoid hematoma or
✓ sinonasal neoplasia

Primary Sinusitis

- Primary Sinus Empyema (**Primary Sinusitis**)
- obstruction of the normal nasomaxillary drainage === > accumulation of mucus in the sinus ===== > infection

- Some cases occur following URT infections that cause inflammation, increase mucus production within the sinuses, and decrease drainage of secretions from the sinuses into the nasal cavity

- **Dental Sinusitis**
- Sinusitis commonly occurs with apical infections of the caudal maxillary cheek teeth.

Sinusitis...

- **Clinical signs**

❖ unilateral purulent nasal discharge,

✓ odorless/malodorous in primary sinusitis

✓ frequently malodorous when associated with dental secondary sinusitis and mycotic sinus infections

❖ Ipsilateral submandibular lymph node enlargement

❖ epiphora.

Less common signs include: facial swelling, exophthalmos, abnormal respiratory noises, head shaking, and Exercise intolerance

Treatment

- Long-term antimicrobial, often weeks of treatment, are required.
- ✓ Acute cases of primary sinusitis may respond to antimicrobial drug administration
- ✓ Chronic cases may only show a transient improvement to antibiotic treatment.

- Treatment by sinus irrigation
- Extensive flushing of the sinuses (If a trephine is available, and the attending veterinarian has the relevant experience)

Treatment …

- Sinusitis secondary to maxillary dental apical infections usually necessitates removal of the affected cheek tooth before resolution of the sinusitis will occur

✓ Definite diagnosis of dental involvement in sinusitis is essential before embarking on tooth removal.

- The treatment of superficial mycotic lesions with antimycotic drugs including nystatin by topical application directly or via an endoscope carries a good prognosis although recurrence is possible

Treatment…

- Surgical removal of large intrasinus fungal granulomas or plaques or of any underlying cause such as sequestra, cysts or progressive ethmoidal hematoma lesions, followed by sinus irrigation with a topical antifungal usually results in rapid resolution of the lesions.

Disorders of the guttural pouches...

1. Guttural pouch tympany

- involves the accumulation of a large quantity of air within a guttural pouch

2. Guttural pouch empyema

- Infection (URT bacterial and viral infections)=== > Failure of mucus and/or pus within the pouches to drain satisfactorily === > guttural pouch empyema.
- URT infections, especially those caused by *St. equi, and rupture of abscessed* retropharyngeal lymph nodes into the guttural pouch are the most commonly implicated causes of GPE

Disorders of the guttural pouches...

3. **Guttural pouch mycosis**

- is a fungal infection that invades the mucosa and, frequently, the closely associated various neurovascular structures.

- *Aspergillus spp, particularly Aspergillus fumigatus and A. nidulans and Candida spp.* are the most commonly incriminated organisms.

Disorders of the guttural pouches...

- **Clinical signs**
- dysfunctions of the surrounding neural structures—cranial nerves VII, IX, X, XI, and XII and the sympathetic trunk, or
- cause erosion of the vascular structures—the internal carotid, the external carotid, and the maxillary arteries.
- Interfere respiration (snoring noises, severe dyspnea**)**
- The clinical signs are potentially very serious

Treatment

- **Guttural pouch tympany** requires surgical correction.

- **Guttural pouch empyema**
- Treatment may entail medical and surgical modalities.

✓ Aggressive lavage of the guttural pouch with saline solutions and the administration of systemic antimicrobials is a first step in therapeutic

✓ Surgery may be necessary if medical therapy is unsuccessful.

Treatment

- **Guttural pouch mycosis**
- Both medical and surgical treatments for guttural pouch mycosis can be used.

- surgical occlusion of the affected vessels

- Topical antifungal medication is frequently used as an adjunct to arterial occlusion.

- Systemic antifungal treatment may also be effective, but the treatment is usually very expensive.

Strangles

- Strangles is a common, highly contagious bacterial infection of the URT caused by *Streptococcus equi:*
- **Etiology**
- *Streptococcus equi subspecies equi*

✓ Gram-positive, chain-forming cocci

✓ *This organism is not a normal inhabitant of the equine respiratory* tract, and

✓ it acts as a primary pathogen causing a highly contagious respiratory disease.

✓ The organism has a worldwide distribution.

Strangles…

- *Streptococcus equi* is not a hardy organism and survives for only short periods (less than 10 days) if desiccated or exposed to ultraviolet light.

- is readily inactivated by commonly used disinfectants including bleach, quaternary ammonium compounds, chlorhexidine, and Virkon, thus greatly facilitating environmental control.

Pathogenesis

- Entry of the bacteria via nasal or oral routes === > adhere to, and colonize, the epithelium in the URT (nasopharynx and pharyngeal tonsil) === > Rapid invasion through the epithelium into the lamina propria and lymphatics === > efficiently evades phagocytosis ===> efficient chemotaxis of neutrophils to the sites of infection === >

Pathogenesis…

- In most horses bacteria are confined to the lymph nodes draining the head (submandibular, parotid, retropharyngeal, and cervical lymph nodes).

- In a minority of cases *S. equi is released in* efferent lymph === > gains entry to the blood circulation via the thoracic duct==== > systemic dissemination of bacteria=== > metastatic abscessation.

- Bacterial antigens (either whole bacteria or bacterial surface proteins) in the circulation can trigger purpura hemorrhagica, an immunecomplex- mediated vasculitis.

Host affected

- The highly host-adapted *S. equi* is maintained only in equine populations in which clinical disease is occurring.
- Equids of all ages are affected but the disease is most common and most severe in young equids.
- Most animals develop a solid immunity during recovery (= >5yrs in 75% of animals)
- Pre-existing immunity may ameliorate expression of the disease

Source of infection

- **Sick animal**= a major source of contagion (nasal discharge and lymph node pus).
- **Animal on recovery stage**= Bacterial shedding also occurs during convalescence for up to 3–4 weeks after the cessation of clinical signs.
- **Carrier animals**= In a small proportion of recovered horses, a carrier state is established with intermittent shedding of bacteria in nasal secretions.
- **persisting contamination** of the environment is not an important source of the organism in outbreaks or in interepizootic maintenance.

Transmission

- *mainly transmitted via infected* droplets either by

✓ direct horse-to-horse contact or

✓ indirectly by **fomites** including personnel (hands, clothing, and footwear) and equipment (tack, feed utensils, and water buckets/troughs).

- **Aerosol transmission** over extended distances does not appear to be an important feature of *S. equi transmission.*
- Morbidity in this age group may be up to 100% with a mortality of 1–5%.
- Older animals show a lower morbidity and mortality

Clinical signs

- ***Severe "classical" strangles***
- The incubation period is variable (1–14 days)
- Pyrexia (up to 42°C) is the earliest clinical sign and persists for 2–3 weeks p.i.
- depression and listlessness
- Pharyngitis, laryngitis and rhinitis may occur and contribute to bilateral nasal discharge.
- The discharge is serous initially and rapidily becomes mucopurulent and then Purulent profuse and tenacious.

Clinical signs…

- LN enlargement (2–3 days pi) === > clinically apparent abscesses (usually 2–3 wks later).

- Abscesses usually develop in the submandibular and retropharyngeal lymph nodes.

- Retropharyngeal abscesses may burst and drain externally (over the lateral laryngeal region) or internally (dorsally into the guttural pouches).

Clinical signs…

- Large, unruptured retropharyngeal abscesses can cause
- ✓ moderate or marked **airway compression** with ventral deviation of the trachea
- ✓ **occlusion of the nasopharynx**, resulting in inspiratory dyspnea and possibly stertorous inspiratory noise.

- Other LN such as parotid & cranial cervical LN are also ferquently involved and may abscess.

Complications associated with strangles

1. **Metastatic abscessation**

- In a small proportion of horses the bacteria disseminate widely via the blood and lymphatics causing metastatic abscessation ("**bastard strangles**") in

- ✓ the abdomen (abdominal lymph nodes, viscera, and peritoneum),
- ✓ thorax (thoracic lymph nodes, lungs, pleura, and mediastinum),
- ✓ central nervous system,
- ✓ eye,
- ✓ skeletal and cardiac muscle,
- ✓ tendon and joint sheaths

Complications …

2. Immune Mediated complications

- purpura hemorrhagica; an immune complexmediated **vasculitis** === > petechial and ecchymotic hemorrhages and subcutaneous edema.
- Myositis
- Glomerulonephritis and Myocarditis

3. Agalactia

"Atypical" strangles

- **milder form of *S. equi infection***
- It resembles a URT viral infectious disease with pyrexia, depression, lymph node enlargement, and nasal discharge

- is frequently not recognized as *S. equi*.

- The more serious sequelae of "classical" strangles do not appear

Diagnosis

- Clinical signs of classical strangles are strongly suggestive

- Confirmation of *S. equi infection* requires culture of viable bacteria or detection of bacterial DNA

✓ Bacterial culture can be performed on nasal, nasopharyngeal, and abscess swabs; nasal washes; guttural pouch lavages; and abscess aspirates.

- Serology

Treatment

- Depends on the stage and severity of the disease
- **Animals with early clinical signs**
- Antibiotic therapy of new cases in the early acute phase with pyrexia and depression may be curatitve and prevent focal abscessation.
- “In-contact” horses can also be treated with antibiotics provided they can be moved to a clean area.
- Penicillin is the drug of choice Other antibiotics including cephalosporins and macrolides can be used.

Treatment…

- Potentiated sulfonamides should be avoided because, *this antibiotic* combination is ineffective in abscesses because of the high concentrations of folic acid present in pus

- **Horses with LN abscessation**
- Antibiotics should not be used in horses with developing abscesses:
- Therapy should be directed toward enhancing maturation and drainage of the abscesses.
- ✓ Topical treatments such as icthammol or a hot pack may be applied to promote maturation of the LN abscesses.

Treatment…

- **Surgical drainage** of lymphadenopathies is some times indicated if abscesses do not rupture spontaneously

- Daily flushing of the open abscess with a 3% to 5% povidone iodine solution should be continued until the discharge ceases.

- **NSAID** may improve the horses behaviour.

Treatment…

- Even in the face of detectable lymphadenopathy, if the animal is febrile, depressed, anorectic and especially manifesting dyspnea, antibiotic therapy is indicated to decrease abscess size and prevent compelet airway obstraction.

- Tracheotomy can be necessary if severe dyspnoea

- **Animals with complications**
- Should receive appropriate symptomatic and supportive therapy

Equine Viral diseases

- Equine influenza virus
- African Horse sickness
- Equine herpesviruses (EHV-1 and EHV-4)
- EVA …

Equine Influenza Virus

- IP 1 and 3days with a range of 18 h to 5 days

- Equine influenza virus, extensively damages the ciliated epithelial cells lining the conducting airways === > disruption of normal mucociliary clearance === >accumulation of mucus and bacteria in the airways and exposure of the lamina propria and irritant receptors=== > frequent coughing.

Equine Influenza Virus

- Fever
- dry, harsh and initially non-productive coughing
- will disappear within 1–3 weeks, in uncomplicated cases, given sufficient rest, .
- a mild rhinitis
- no obvious swelling of the submandibular LN.
- Serous nasal discharge to mucopurulent (bacterial infection).
- High Morbidity rate (up to 98%)
- Mortality rates are usually very low in uncomplicated cases, with the exception of young foals that have not acquired maternal-derived immunity.

Equine Influenza Virus

- In vaccinated horses clinical signs are not necessarily typical of influenza,
- ✓ one of the most common signs is nasal discharge, which rapidly spreads among horses.

- Tentative diagnosis: rapidly spreading disease manifested by a harsh, dry cough, high temperature, and nasal discharge

- Laboratory diagnosis: rapid antigen detection, virus isolation or serology

Equine Influenza Virus

- **Treatment of clinical cases**
- non-steroidal anti-inflammatory drugs
- antibiotics such as penicillin
- Rest

- **Prevention and control**
- Rapid detection of the disease
- Vaccination

African Horse Sickness

- AHS is typically a peracute or acute infectious, non-contagious, disease of Equids transmitted by *Culicoides midges.*
- AHS is endemic and series problem of horses and mules in Ethiopia and causes significant loss of equine
- AHS virus (AHSV) is in the genus Orbivirus in the family Reoviridae.
- There are nine serotypes of AHSV

Host range

- Mainly equidae

✓ Horses = most

✓ mules = less

✓ donkeys, zebras, camels are even more resistant

✓ Carnivores =mortality in domestic dogs following ingestion of AHSV-contaminated meat.

Transmission…

- *Culicoides spp. (biting midges)* main.

✓ *Culicoides imicola the* most significant vector.

- *the disease has often a* ***seasonal incidence****.*
- The frequency of outbreaks is higher from September to December in Ethiopia

- ***Windborne*** **dispersal** of infected vectors over **long** distances is possible.

Pathogenesis

- Inoculation by biting *Culicoides* ==== > *initial multiplication* occurs in the regional lymph nodes ==== > viremia ==== > infection of target organs==== > Replication particularly in capillary endothelium of these organs === > a secondary cell-associated viremia of variable duration and with clinical signs.

- Much of the pathology results from increased permeability of capillary endothelium.

Clinical signs

- The incubation period following AHSV infection in susceptible hosts is typically 5–7 days.
- The clinical signs are markedly affected by the host species and its immune status.
- There are four different forms of the disease:

1. the pulmonary form
2. the cardiac form
3. the mixed form, and
4. horsesickness fever.

the pulmonary form

- The disease is characterized by a massive pulmonary edema and hydrothorax, often with several liters of fluid accumulating.
- fever (up to 41°C) lasting up to 2 days
- severe dyspnea,
- paroxysmal coughing
- a copious nasal discharge of frothy, serofibrinous fluid
- Death can occur within a few hours of onset of signs and < 5% of animals with this form survive.

the cardiac form

- The cardiac or edematous form is chiefly characterized clinically by subcutaneous swelling of the neck and head, particularly the supra-orbital fossa.
- At post-mortem, yellow gelatinous edema of Sc and IM connective tissues is the most obvious feature.
- In more severe cases, in which mortality is more likely, all cranial soft-tissue structures are affected, along with the shoulders, neck, and chest.
- Petechial hemorrhages are a poor prognostic indicator.
- Mortality from the cardiac form is around 50%.

- **Mixed form**
- both pulmonary and cardiac signs are seen

- **Horse sickness fever**
- is the most common form in horses with pre-existent immunity to one or more serotypes & less susceptible spp.
- It is the mildest form of the disease and is probably very underdiagnosed.
- It is characterized by a pyrexia (39–40°C) lasting 1–6 days and some edema of the supra-orbital fossa.
- The pyrexia may also be accompanied by transient loss of appetite, slight dyspnea, and increased HR.

Diagnosis

- In most cases, in endemic or epidemic areas, clinical diagnosis of AHS will often be correct.

- Confirmation of diagnosis of AHS

✓ Serology

✓ virus detection through isolation

✓ the detection of specific viral RNA using PCR.

- Virus can be isolated from tissues or blood samples from acutely affected animals using a variety of different cell culture systems.

Treatment

- There is no specific treatment for AHS.
- Supportive care and treatment of complication of the disease should be provided.
- Rest

Control and prevention

- **Movement restriction**

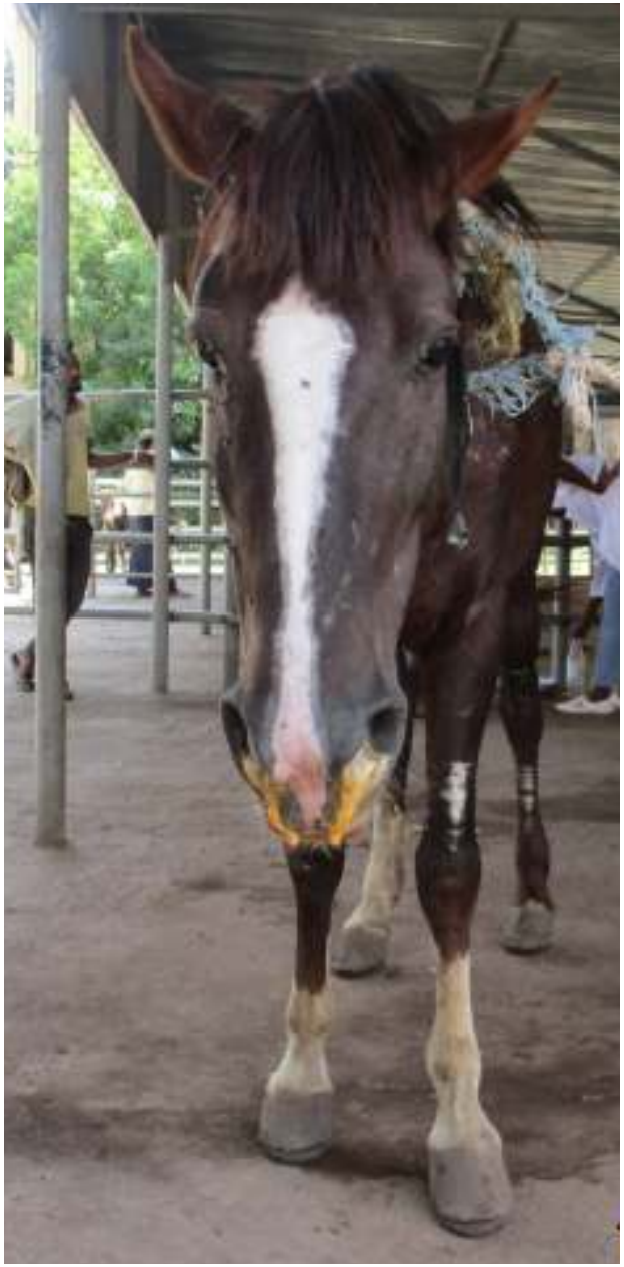
✓ prevent infected animals initiating new foci of infection,

- **Vaccination**

✓ *(monovalent and polyvalent)*

✓ AHSV 9, 6 & 4 in Ethiopia

- **Vector control**
- At a minimum, stabling from dusk to dawn, the period when *Culicoides* are most active, is recommended.



BACTERIAL PNEUMONIA

- **Causes**
- Under normal conditions the equine lung contains only small numbers of potential bacterial or fungal pathogens that are considered to be transient contaminants.

- These bacteria are removed by clearance mechanisms.

- When pulmonary defense mechanisms are overwhelmed, aspirated bacteria from the oropharynx may proliferate and cause pneumonia.

Bacterial pneumonia

- Predisposing factors include a variety of stresses such as

✓ long-distance transport,

✓ overcrowding in barns with poor air hygiene,

✓ hospitalization (especially being cross-tied for lengthy periods), and

✓ general anesthesia.

Bacterial pneumonia

- **Causes**
- The most common gram-positive bacteria involved:

✓ *Streptococcus zooepidemicus ,*
✓ *Staphylococcus aureus, and*
✓ *Streptococcus pneumoniae.*

- *The* most frequent gram-negative isolates are

✓ *Pasteurella*
✓ *Actinobacillus spp.,*
✓ *Escherichia coli,*
✓ *Klebsiella pneumoniae,* and
✓ *Bordetella bronchiseptica.*

Causes

- The **anaerobic bacteria** most commonly isolated are

✓ *Bacteroides fragilis,*

✓ *Clostridium sp., and*

✓ *Fusobacterium spp.*

Bacterial pneumonia

- *Polymicrobic infections are not* uncommon in cases of equine pneumonia and may represent a synergy between aerobic/facultatively anaerobic and anaerobic bacteria.

- *The* majority of cases are caused by ***S****. zooepidemicus alone or in* combination with other bacteria.

Bacterial pneumonia

- **Pathogenesis**
- Stress === > reduces mucociliary clearance and reduce the efficiency of phagocytosis by neutrophils and macrophages=== > resident microbes multiply and colonize=== > pulmonary inflamation === > increased caplilary permeability and pulmonary exudate
- It is mainly the cranioventral regions of the lung that are affected.
- Affected regions rapidly become consolidated, giving them a red appearance, and within these regions pulmonary infarction and necrosis can develop, resulting in a serosanguineous suppurative exudate.

Bacterial pneumonia…

- **Clinical signs**
- Bilateral mucopurulent nasal discharge
- Coughing
- Increased respiratory rate and effort
- Reduced exercise tolerance and lethargy
- Pyrexia and poor appetite
- Harsh lung sounds
- Dullness of lower lung areas on percussion
- Pale or cyanotic mucous membranes in severe cases

Treatment

- **Broad spectrum antimicrobial** therapy and excellent supportive care
- ✓ Trimethoprimsulphonamide
- ✓ Beta lactam antibiotics + aminoglycosides (procaine penicillin and gentamicin)
- metronidazole is effective against most anaerobes
- Rest in a clean, dust-free environment
- Offer a high quality palatable diet.
- Reduce pyrexia with the administration of NSAIDs
- Corticosteroids may be administered in severe cases including aspiration pneumonia.

Pleuropneumonia

- Pleuropneumonia is the condition in which infection involve the lungs, the pleura and pleural space.
- This may occur as an extension of severe pneumonia, if an animal is not rested to allow recovery, or from the rupture of an abscess into the pleural space.
- **Clinical signs**
- Respiratory distress – due to the pleural effusion
- Acutely painful (pleurodynia) – grunting on inhalation, abducted elbows, guarded behaviour when coughing, or flinching on percussion of the chest
- Nasal discharge – mucopurulent, bilateral. May be foul smelling if anaerobes are involved
- Inappetance

Pleuropneumonia…

- Percussion – dull resonance ventrally
- Auscultation -Attenuation of audable bronchovesicular sounds over the ventral lung fields

✓ Friction sounds are common with chronicity

- **Diagnosis**
- History and physical examination findings are often suggestive
- Definitive diagnosis is made on the basis of identification of septic fluid within the pleural space.
- Ultrasonography
- Radiography

Treatment

- Long-term antimicrobial therapy, as for pneumonia. Do not stop antibiotics before a complete recovery is made.
- Include metronidazole in the treatment protocol if anaerobic involvement is suspected.
- Rest
- supportive care including IV fluids therapy, particularly during when the animals are depressed, anorecic and dehydrated.
- NSAIDs
- Good nursing care and nutrition are important in the recovery phase.

Parasitic pneumonia

- Etiology

❖Dictyocaulus arnfieldi

❖ Parascaris equorum

Lungworm

- Lungworm causes inflammation of the bronchioles in adult equids.
- Donkeys rarely suffer clinical signs of lungworm, but they act as a reservoir of infection for horses.

- **Clinical signs**
- chronic coughing
- Slightly increased respiratory rate and lung sounds
- Non-progressive condition
- Poor body condition

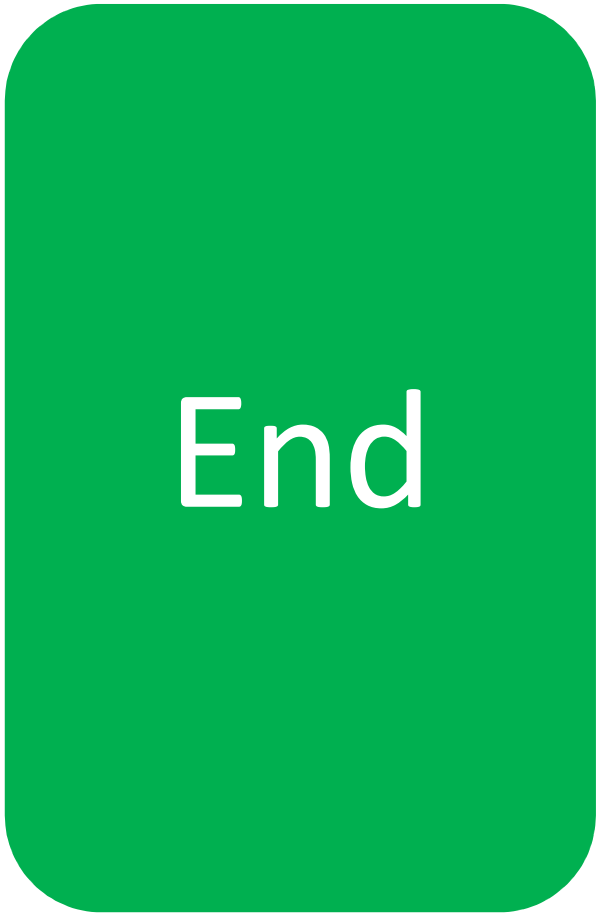
Lungworm...

- **Diagnosis**
- Patency (larvae in the faeces) is rare in horses. A bronchial lavage can be used to collect larvae for a definitive diagnosis.

- **Treatment**
- **Ivermectin** or moxidectin are effective.
- Lungworm can recur if pasture contamination is present.
- **Treat donkeys** that are resident with horses, even if they do not show signs, as they can be a source of continued infection.

Parascaris equorum

- Migrating larvae cause lung damage in foals.
- **Clinical signs**
- Frequent coughing
- Greyish-white nasal discharge
- Possible fever
- Other signs of worm infestation, e.g. weight loss, poor coat, colic
- **Treatment**
- Broad-spectrum anthelmintics are effective.
- The prognosis is good, but the disease can recur


End

Diseases and disorders of the urinary tract

For AAU, CVMA 4th year DVM students

Reta Tesfaye (DVM, MTADM)

Topic

- Acute Renal failure
- Chronic Renal Failure
- Urinary Tract Infections

Acute Renal failure

- is a clinical syndrome associated with abrupt reduction in glomerular filtration rate (GFR).

Acute renal failure

- Acute renal failure is usually a consequence of:

1. **Pre-renal factors**
2. **Renal (intrinsic) factors**,
3. **Post-renal urinary obstruction**.

Acute renal failure...

- **Pre-renal failure**

- is associated with conditions that result in
- ✓ decreased cardiac output or
- ✓ increased renal vascular resistance, or
- ✓ both,

- Pre-renal factors are probably the most common cause of ARF.

Acute renal failure...

- reductions in renal blood flow (RBF), GFR, and urine output usually result in **azotemia** and retention of water and electrolytes

- **Anesthesia** also may decrease cardiac output enough to result in a degree of prerenal azotemia.

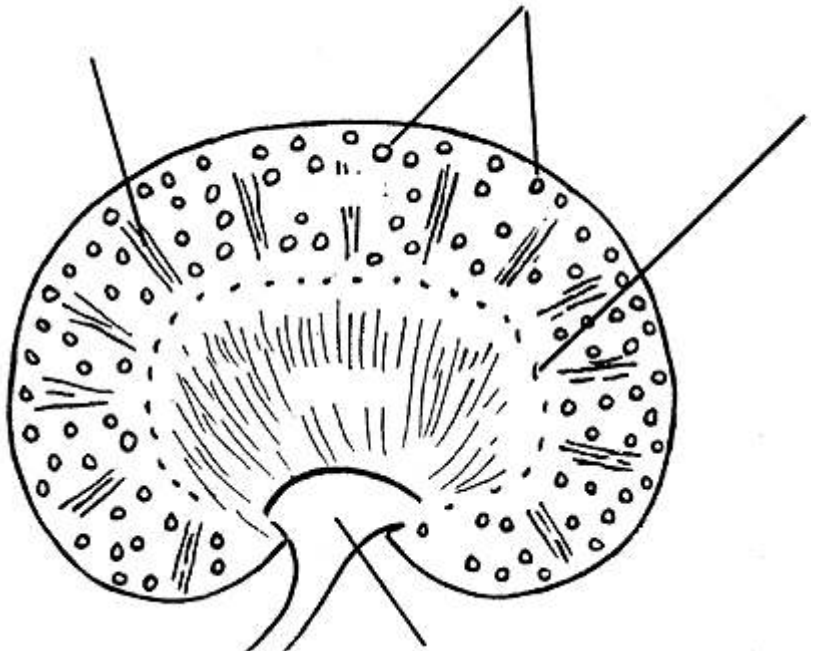
- **Nonsteroidal antiinflammatory drugs** (NSAIDs) also can precipitate prerenal azotemia in patients with decreased RBF.

Acute renal failure...

- **Pre-renal factors**
  - ✓ Dehydration
  - ✓ Endotoxemia
  - ✓ Hemorrhage
  - ✓ Cardiovascular insufficiency

Acute renal failure...

- **Renal (intrinsic) factors**
- **Prolonged pre-renal disturbance**
- **Nephrotoxins**
  - Drugs (aminoglycoside antibiotics & NSAIDs).
  - Toxic plants
  - Mycotoxins
  - Heavy metals
  - Hemoglobinuria
  - Myoglobinuria



Acute renal failure...

- **Intrinsic diseases**
- **Glomerulonephritis**
- **Pyelonephritis**
- **Renal lithiasis**
- **Hydronephrosis**
- **Neoplasia**

Acute renal failure...

- Postrenal obstructive failure

- can develop following disease of the renal pelves, ureters,bladder, or urethra.

Acute renal failure...

- **Post-renal factors**

✓ Urolithiasis (ureter/bladder/urethra)

✓ Bladder rupture

✓ Urethral trauma

✓ Obstructive neoplasia

Clinical Signs

- the clinical manifestations of ARF reflect

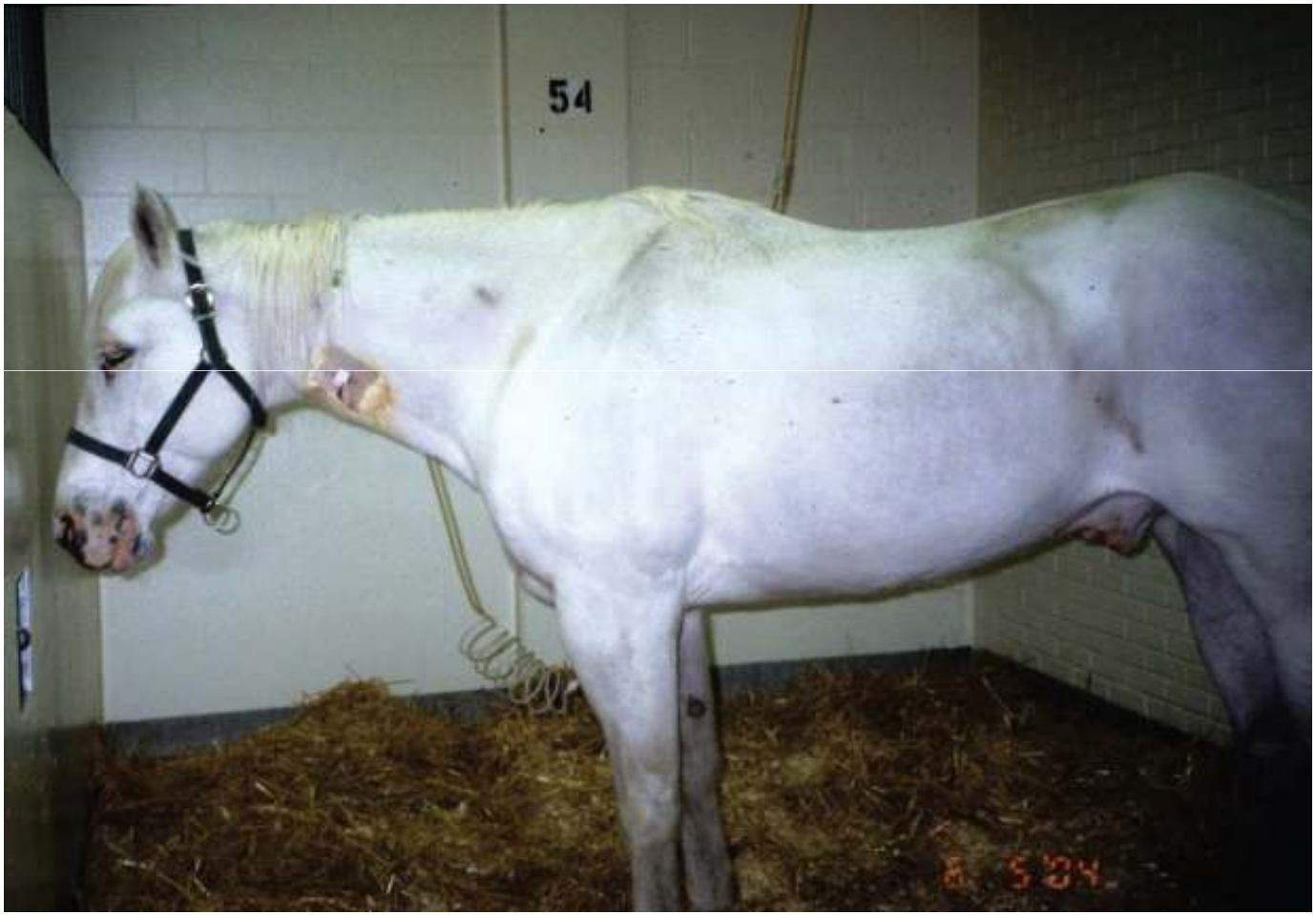
✓ the systemic effects of toxic substances usually excreted in the urine (i.e., uremia generally is reflected by **anorexia** and **depression**),

✓ urinary tract dysfunction, and

✓ Derangements of fluid, electrolyte, and acid-base balance.

- One may observe signs of encephalopathy in horses with severe azotemia.

A horse affected with acute renal failure



Diagnosis

- Increases in plasma **urea nitrogen** and **creatinine** concentrations (i.e., azotemia) are frequently the initial findings that suggest compromised renal function

Diagnosis…

- ARF should be suspected in horses that become
- ✓ anorectic and depressed within 7 days of an episode *a hemolytic crisis.*

- ✓ patients showing more marked **depression and anorexia** than would be expected with the primary disease process and in patients that **fail to produce urine** within 6–12 h of initiating fluid therapy.

Diagnosis…

- Nephrotoxic ARF should be considered when horses being treated with **aminoglycosides** become inexplicably depressed and inappetant **within 2–3** days of the start of treatment.

- Infectious acute renal failure is characterized by fever, partial anorexia, depression and **gross hematuria**.

Diagnosis…

- Pre-renal

➢ clinical signs are usually referable to the primary problem

➢ Oligurea with

✓ concentrated urine (Sg > 1.035) and

✓ rapid correction of azotemia with rehydration

Diagnosis…

- Renal

➢ Depression, colic signs, tachycardia, fever, laminitis

➢ may be accompanied by mild to moderate proteinuria, glucosuria, pigmenturia, and increased RBC and casts on sediment examination

Diagnosis

- Postrenal or obstructive uropathy usually is characterized by
- ➢ mild to severe abdominal pain and
- ➢ pollakiuria and
- ➢ Stranguria
- ➢ Recognized by rectal examination unless the bladder has ruptured.

Treatment

- Treatment of ARF in all ages of horse centers on **correction of the primary problem**.

✓ Hemodynamic disturbances such as acute diarrhea, hemorrhage, congestive heart failure and bacterial toxemia should be treated appropriately.

- Potential **iatrogenic causes of tubular necrosis** (nephrotoxic drugs) should be withdrawn.

Treatment

- If **septicemia** is suspected, IV therapy with combination of penicillin and gentamicin *for a prolonged period is recommended.*

Treatment

- Restoration and maintenance of **fluid,** electrolyte and acid-base imbalances

- In all cases, initial treatment is centered on **rational fluid therapy**.

- Sodium and chloride replacement can be accomplished by using **0.9% NaCl IV** and/or oral replacement therapy until hydration and electrolyte status are normalized.

Chronic Renal Failure

- Chronic renal failure (CRF) is recognized infrequently in horses.

- It appears to be a greater problem in older horses

- CRF may be divided by clinical and pathologic findings into two broad primary categories

1. Glomerular disease
2. Tubulointerstitial disease.

Clinical Signs

- Horses with CRF present relatively late in the disease course, when their owners note **lethargy, anorexia, and weight loss.**

- A **history** of months to years of

✓ polydipsia and polyuria in some cases supports renal disease of long duration.

✓ preexisting disease (colic, colitis, pleuropneumonia) or

✓ prolonged medication (aminoglycoside antibiotics or NSAIDs) may provide important information about the initiation and duration of renal failure

Clinical Signs…

- **Chronic weight loss** is the most common presenting complaint for horses with CRF.

- Partial anorexia,
- ventral edema,
- polydipsia and polyuria,
- rough hair coat,
- lethargy, and
- poor athletic performance are other owner concerns.

Clinical Signs…

- horses with advanced CRF may have a characteristic odor that likely reflects the combined effects of uremic halitosis and increased urea excretion in sweat.

- In the **oral cavity**, excess ammonia can lead to

✓ excessive dental tartar formation,

✓ gingivitis, and

✓ oral ulcers.

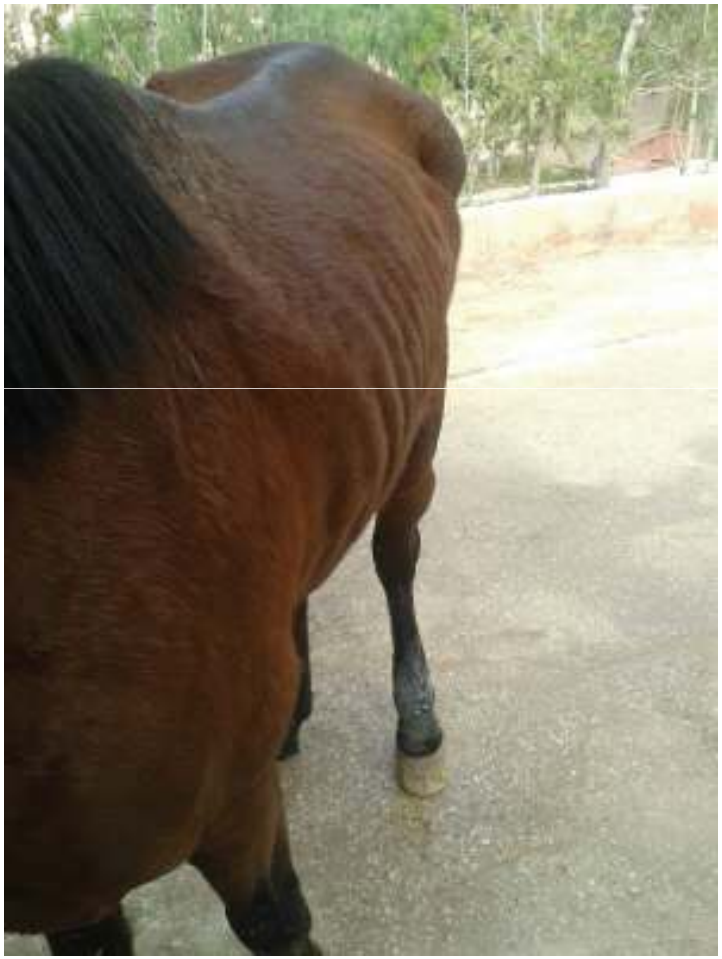
Clinical Signs…

- In the **gastrointestinal tract**, excess urea and ammonia can lead to

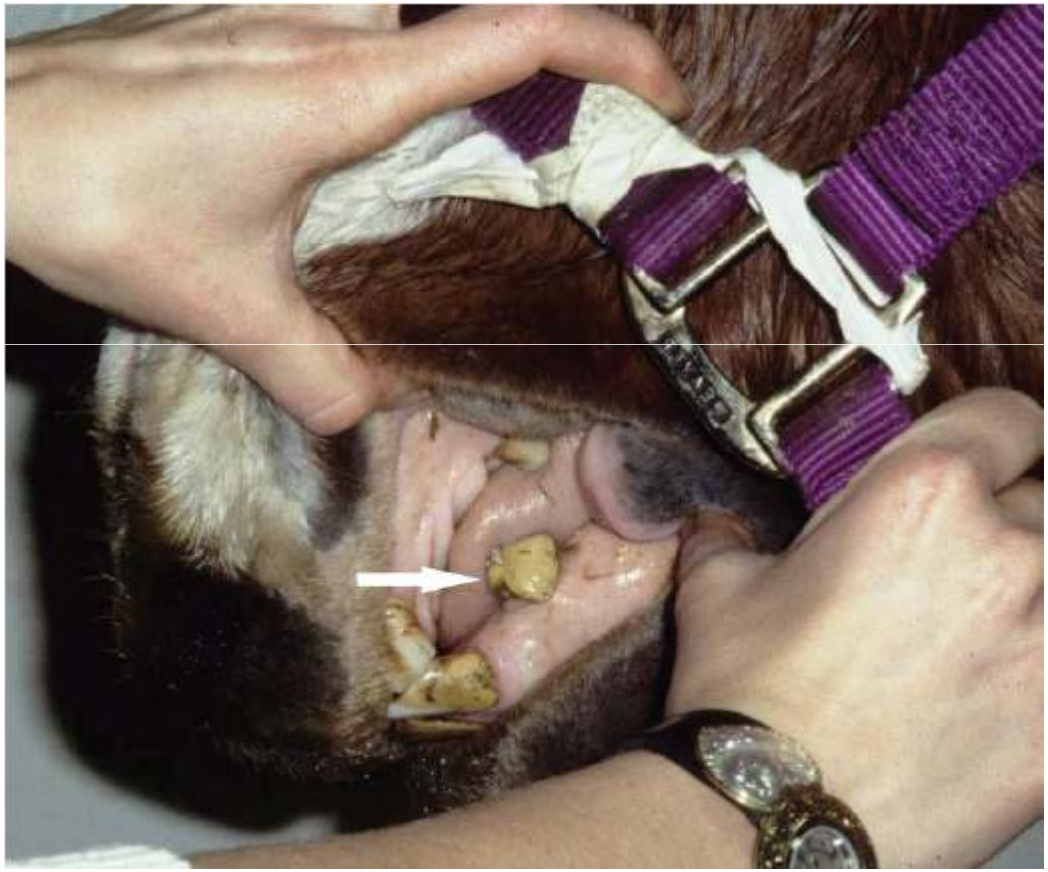
✓ ulceration and mild to moderate protein-losing enteropathy, and

✓ soft feces (severely uremic animals)

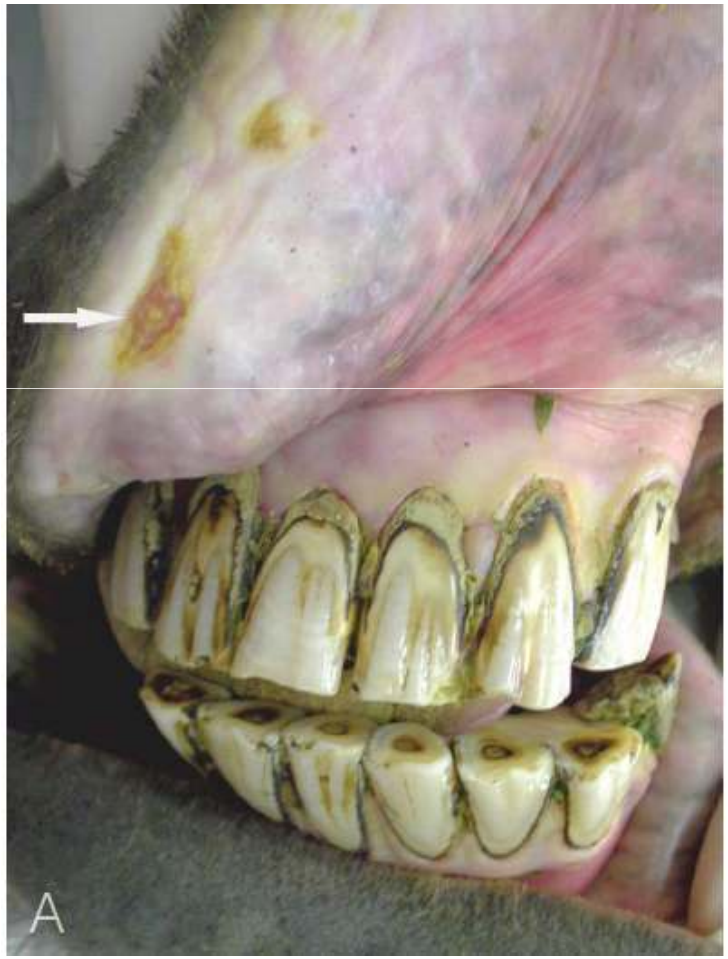
Chronic weight loss



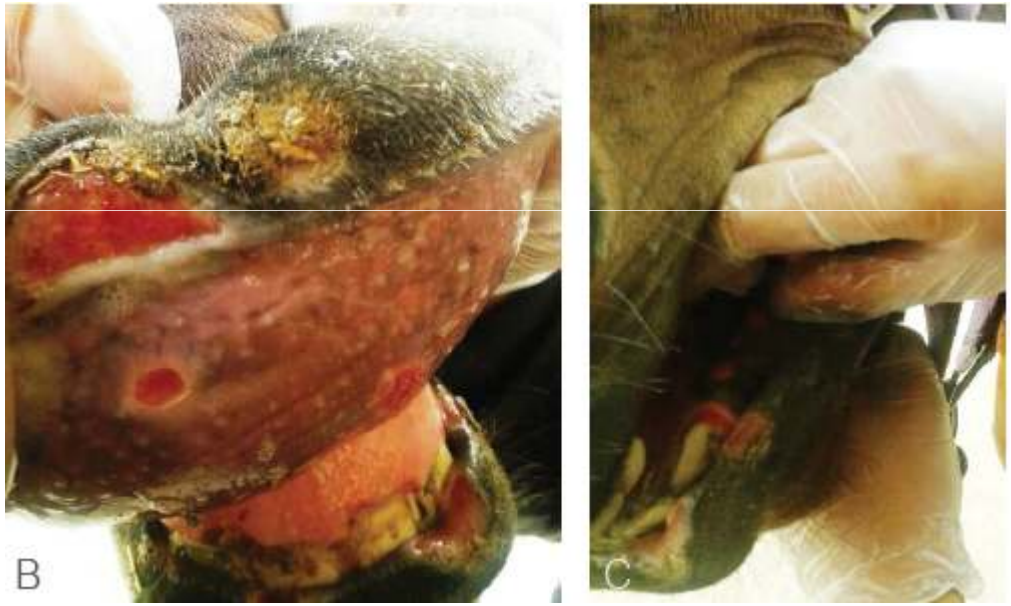
Dental tartar



Oral ulcers and tartar on the incisors



Multiple oral ulcers seen in a horse affected with CRF.



Diagnosis and Laboratory Evaluation

- One establishes a diagnosis of CRF when

✓ persistent isosthenuria (specific gravity of 1.008 to 1.014) accompanies azotemia and

✓ typical clinical signs.

Diagnosis and Laboratory Evaluation…

- further abnormal laboratory data accompanying CRF can include

✓ mild anemia,

✓ hypoalbuminemia,

✓ hyponatremia,

✓ hyperkalemia,

✓ hypochloremia,

✓ hypercalcemia,

✓ hypophosphatemia, and

✓ metabolic acidosis

Diagnosis and Laboratory Evaluation…

- **Disease Progression**
- One of the hallmarks of CRF is **the progressive nature of the disease**,

Treatment

- At the time of presentation most horses with CRF exhibit obvious weight loss and other clinical signs.

- Because of the progressive and irreversible nature of the renal disease, the long-term **prognosis is grave.**

- Specific corrective treatment for CRF (**renal transplantation**) is not available for equines, and maintenance by peritoneal **dialysis** or hemodialysis would only be practical for valuable breeding animals. Not in Ethiopia

Treatment…

- **Pyelonephritis** =antibiotic treatment
- providing plenty of **water,**
- to discontinue administration of **nephrotoxic agents,**
- povide a **palatable diet** to encourage appetite and minimize further weight loss.

Treatment…

- Administration of **B vitamins** or anabolic steroids for their touted appetite-stimulating effects may benefit some animals.

Treatment…

- **IV fluid therapy** to promote diuresis, usually with 0.9% NaCl solution, is of much greater benefit in cases of acute, reversible renal failure but also may benefit patients that suffer a sudden exacerbation of CRF.

- One must administer Iv fluid therapy cautiously because horses with CRF can develop significant peripheral or pulmonary edema.

Treatment…

- Supportive care also can include supplementation of **sodium bicarbonate** (50 to 150 g per day) when serum bicarbonate concentration is consistently less than 20 mEq/L.

- Edema is not usually a significant problem, and the horse should **tolerate mild edema** (rather than being treated with diuretics that could be ineffective or lead to further electrolyte wastage) unless the edema interferes with ambulation.

Urinary Tract Infections

- ascending UTI Vs septic nephritis
- Male vs female

- ascending UTIs are more common

- septic nephritis may be an occasional consequence of septicemia, especially in neonatal foals.
- Mares are at higher risk for UTIs than geldings or stallions because of their shorter urethra.

Urinary Tract Infections…

- Development of a UTI requires

✓ initial urethral colonization with pathogenic bacteria,

✓ entry of pathogens into the bladder, and

✓ subsequent multiplication in the bladder.

Urinary Tract Infections…

- pathogenic *E. coli are rich in specific surface adhesins*
- Normal vulvar and preputial flora protect against urethral colonization by pathogenic bacteria, but
- any anatomic defect leading to **turbulent urine flow** compromises maintenance of normal flora and may increase the likelihood of colonization by pathogens

Urinary Tract Infections…

- Once a pathogen has colonized the distal urethra, rapid proliferation between micturitions allows invasion of the proximal urethra and bladder, which do not have a protective flora.

Urinary Tract Infections…

- Although antibiotic therapy is highly effective in eliminating most UTIs, **recurrent UTIs** can be a challenge to manage

Urinary Tract Infections…

- **Etiology**

- the sterility of the urinary tract relies heavily upon a **flushing mechanism (rather than any secretory** function),

✓ alterations in the normal flow patterns as result of neurologic or physical obstructions commonly result in infection.

Urinary Tract Infections…

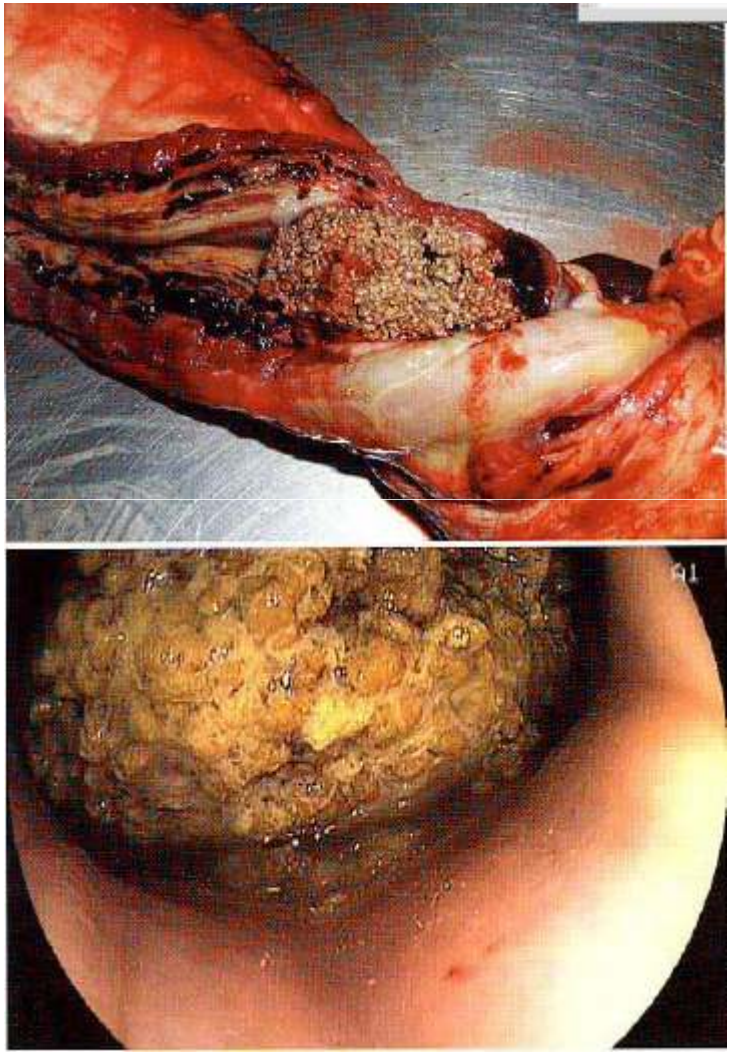
- risk factors for development of UTI in horses are:

1. Bladder paralysis.

- Horses with bladder paralysis or decreased urethral sphincter tone (from trauma or neurologic disease) are clearly at greater risk of UTI

Urinary Tract Infections…

2. urolithiasis.



3. Urethral damage,

✓ e.g. foaling trauma in mares, neoplasia or habronemiasis in males

Urinary Tract Infections…

- **Iatrogenic infection following bladder catheterization** is an accepted risk because it is virtually impossible to perform this in a sterile fashion.

- when urethral or bladder mucosa has been **damaged** or when urine stasis (bladder paralysis) is present, catheterization has a far greater risk of producing more persistent infection.

Urinary Tract Infections…

- *Escherichia coli,*
- *Staphylococcus spp.,*
- *Corynebacterium spp.,*
- *Pseudomonas aeruginosa,*
- *Proteus mirabilis,*
- *Klebsiella spp. and Enterobacter spp. are the most common* pathogens.

Clinical signs

- Upper tract inflammation produces **less specific, more subtle clinical signs** and so may be easily overlooked.

- Horses with upper tract infection often have concurrent signs of systemic infection such as **fever and weight loss.**

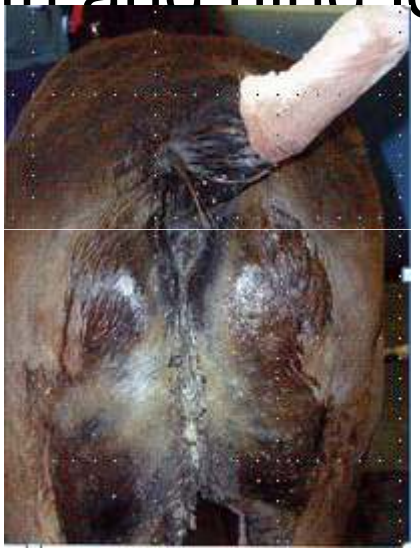
Clinical signs

- Lower tract infection typically shows **disturbances in urine** flow but seldom any evidence of systemic infection.

- Dysuria,
- stranguria,
- pollakiuria and
- incontinence may be present.

Clinical signs…

- Chronic infection may cause perineal urine scalding in mares and sheath and hind leg dermatitis in males.



- Gross **pyuria** may also be observed as passage of mucopurulent debris in otherwise clear urine.

Diagnosis

- The presence of urinary tract infection is based on **clinical signs and laboratory analysis** of blood and urine samples.

- **Bacteriologic culture** of urine obtained from the bladder may identify specific infections and their antibiotic sensitivity

Treatment

- Treatment is focused on the primary cause and the secondary effects of the infection, especially if they are long standing.

- Antibiotic selection is critical to success

Treatment…

- **Potentiated sulfonamides** are a mainstay of therapy; trimethoprim–sulfa combinations at 35 mg/kg PO (q 12 h) or IV (q 12 h) and maintained for up to 21 days or more without significant harmful effects in most cases.

- This combination is useful because both components are excreted into urine in high concentrations in a non-metabolized state.

- They are also **very safe** even in very ill or dehydrated horses.

Treatment…

- Penicillin administered parenterally (e.g. procaine benzylpenicillin 20 000 IU/kg q 12 h for 5–7 days) is effective for treating upper or lower UTIs caused by susceptible *Corynebacterium spp., Streptococcus spp., and some Staphylococcus* spp. and *Leptospira spp.*

Treatment…

- Gentamicin (6.0 mg/kg q 24 h IV) *and other aminoglycosides should be* **reserved** for resistant lower tract or acute life-threatening infections caused by Gram-negative organisms.

- The drugs are however nephrotoxic and should certainly not be used when there is renal failure.

Treatment…

- Cephalosporins, tetracyclines and chloramphenicol are concentrated in urine and can be useful in treatment.

Treatment…

- Prolonged courses of antibiotics are required in all urinary tract infections; courses of 1–6 wk may be indicated.

- Ideally, a midstream voided urine sample should be submitted for bacterial culture 2–4 days after initiation of therapy and again 1–2 wk after treatment has been discontinued.

Treatment…

- Recurrent infections with the same pathogen should alert the clinician to the possibility of a primary focus in the upper tract, e.g. nephroliths *or* other parenchymal disease.

Thank you

Disorders of the endocrine system for AAU CVMA 4th year DVM students

By Reta Tesfaye (DVM, MTADM)

Introduction

- An *endocrine gland is a ductless gland that releases its* secretory product—a hormone—directly into the bloodstream for general circulation.

- A *hormone* acts as a ‘chemical messenger’ that can influence the activity of other organs or tissues

The endocrine system...

Primarly consists of

- ✓ The pitutary gland
- ✓ Adrenal gland
- ✓ Thyroid gland
- ✓ Parathyroid gland
- ✓ Pancrease

Examination of the Endocrine system

- **History**
- In most cases, endocrine disorders present non-specific signs such as **weight loss, lethargy, skin or hair abnormalities and change in appetite**

Examination of the Endocrine system ..

- **Physical examination**
- Specific abnormalities involving skin, musculoskeletal and neurologic systems may alert the clinician to an endocrine disorders.
- Ex:

✓ Hirsutism

✓ Chronic recurrent laminitis or sole abscesses

✓ Neurologic dysfunction (blindness, or diminished responsiveness to pain)

Development of Endocrine system disease

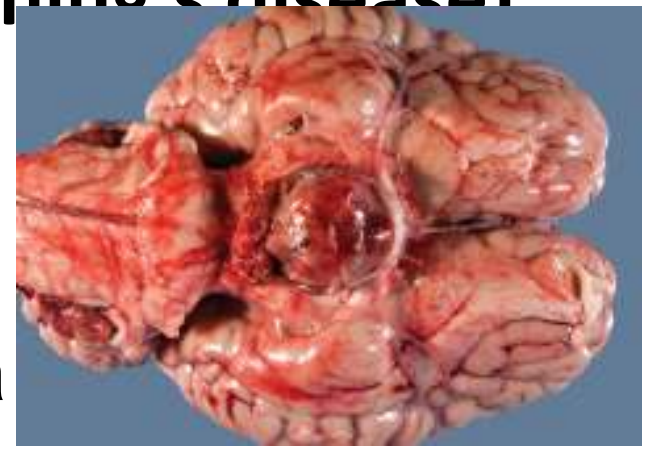
- Endocrine system diseases can develop

✓ when too much or

✓ not enough hormone is produced or

✓ when normal pathways for hormones to be used and removed are disrupted.

Pituitary Pars Intermedia Dysfunction;
Pituitary tumour (equine Cushing's disease)



- are common in aged ponies

- The tumour causes disease via

✓ **altered hormone production** (the pituitary gland and the adrenal glands), as well as effects

✓ due to **compression** of the base of the brain and optic nerves

equine Cushing's disease

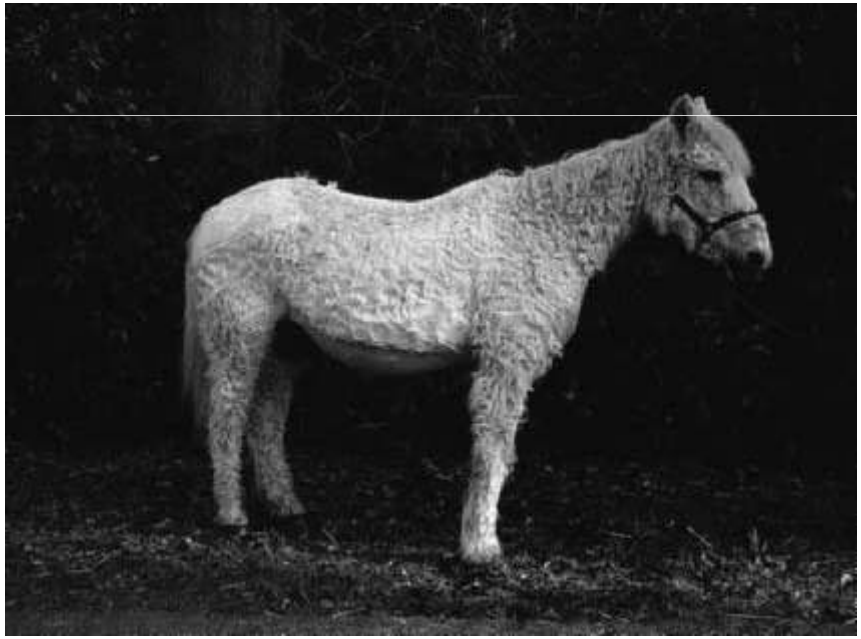
- **Clinical signs**
- The tumour causes a wide range of clinical signs, although the four most common signs are:

1. **Hirsuitism**
2. **Weight loss**
3. **Lethargy**
4. **Laminitis**

- A pony or horse that has this profile of clinical signs is very likely to have a pituitary tumour

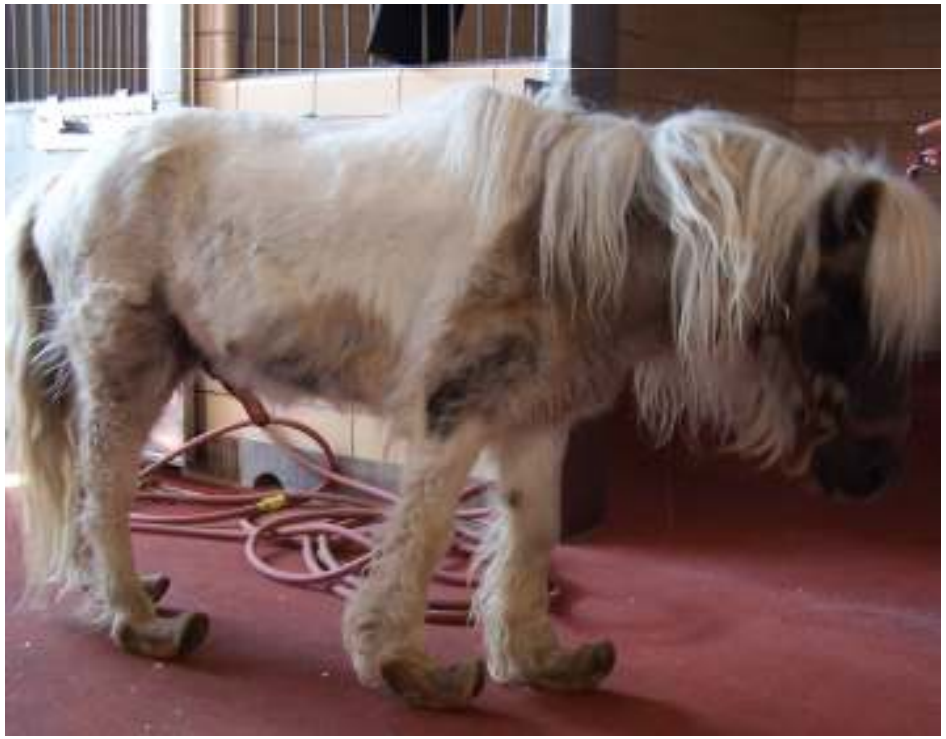
✓an abnormal and very thick, hairy coat, which will not be shed

mask the excessive sweating and weight loss



equine Cushing's disease

- **Laminitis**
- laminitis, which is extremely painful and often incurable (a welfare problem)



equine Cushing's disease...

- Other clinical signs include:

✓ Polyuria/polydipsia

✓ Excess sweating

✓ Tachycardia/tachypnoea

✓ Skin and other infections

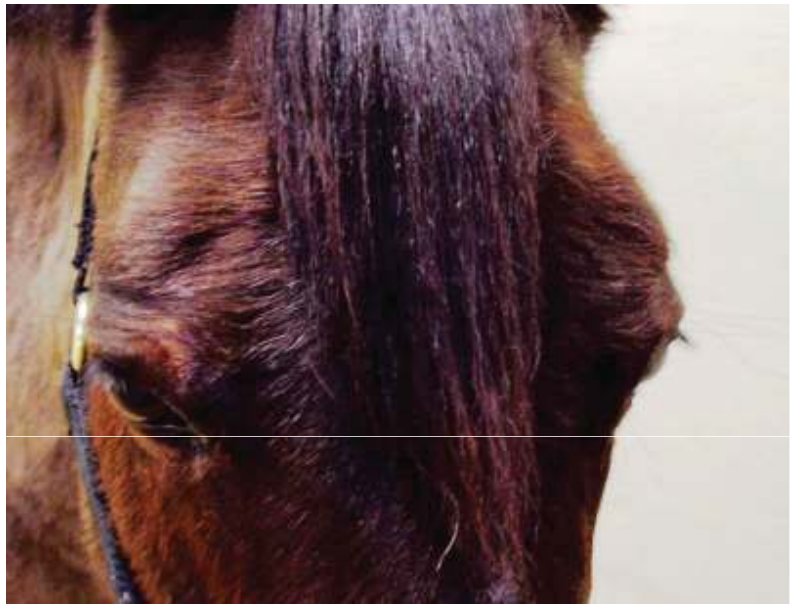
✓ Oral and colonic ulceration

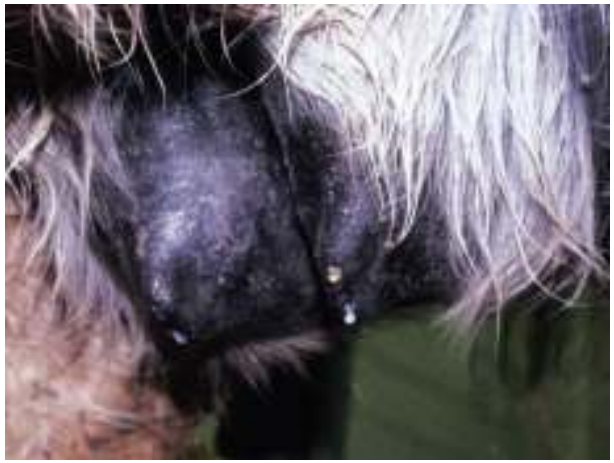
✓ Bulging supra-orbital fat pads.

✓ Neurological signs

✓ Irregular oestrus

✓ Lactation (without pregnancy).





equine Cushing's disease

- In donkeys clinical signs are not often seen hence PPID can go unnoticed.

- Donkeys typically have longer coats than horses but do look out for a coat that seems excessively long .

- try and monitor a donkey's water consumption, if you think that they may be drinking more than usual.

equine Cushing's disease

- a severe attack of laminitis or recurrent bouts of laminitis is often the first indication that a donkey may have PPID.

- an increased susceptibility to **recurrent** infections and parasite burdens.

- a 'pot-bellied' appearance and a generalised lethargy.

equine Cushing's disease

- **Diagnosis**
- Haematology and biochemistry may be helpful:

✓ Leucocytosis with neutrophilia, lymphopenia and eosinopenia

✓ Hyperglycaemia and glucosuria

✓ A persistently raised serum insulin is a good indicator of the disease

equine Cushing's disease

- ***Management***
- Pituitary tumour cases can be managed successfully although this is expensive and requires commitment.

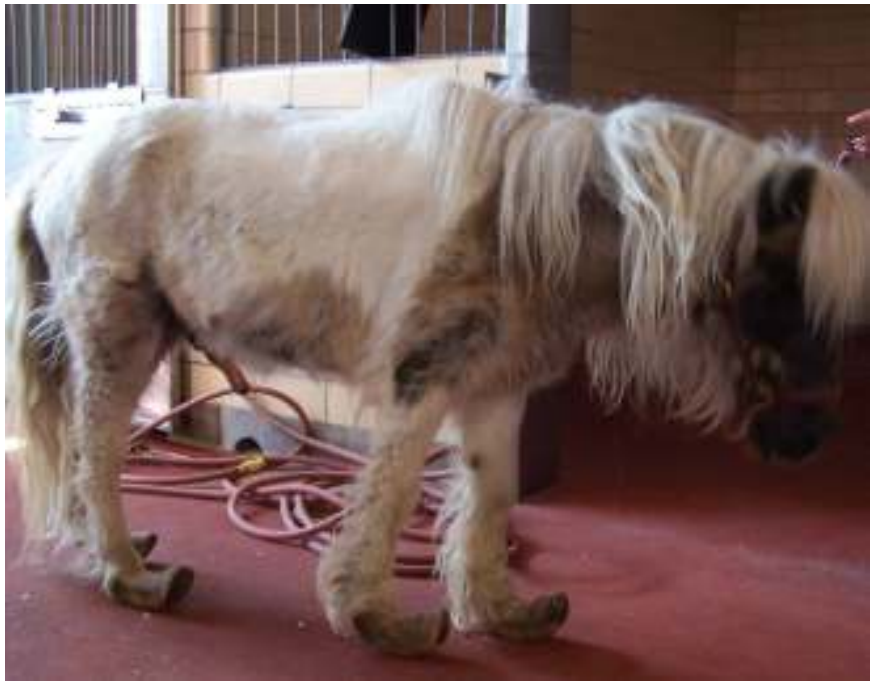
- Treatment is not curative but will prolong the animal's life.

- Treatment with drugs that inhibit the metabolic activity of the tumour are helpful:**pergolide, cyproheptadine**

equine Cushing's disease

- The coat should be kept **clipped and groomed**

- The feet require **regular farriery**; if **laminitis** is present this will need specific treatment



Anhidrosis

- The **inability to sweat** effectively in response to appropriate stimuli

- is characterized by a partial or total loss of the ability to sweat.

- **hyperthermia** may develop, because of the resulting impaired heat loss, .

Anhidrosis…

- **Cause**
- Altered sweat gland receptor function

- Hypothyroidism has been implicated as a cause of anhidrosis.

- **Historical findings**
- Tachypnea (especially extended after exercise) combined with a noticeable lack or reduction of sweating= predominant complain

Anhidrosis…

- *Clinical signs*
- **In acute cases**
- sweating is much reduced or absent;
- ✓ this is accompanied by respiratory distress, labored breathing, fever, collapse and occasionally death.

- **Chronic anhidrosis**
- is characterized by dry lusterless coat, flaky skin with alopecia, particularly affecting the face and neck.

Anhidrosis…

- ***Diagnosis***
- Diagnosis is based on a clinical history of non-sweating.

- Anhidrotic horses show a very slow sweat response to an intradermal skin test with various dilutions of adrenaline (epinephrin).

Anhidrosis…

- ***Treatment***
- Application **of cold water** to reduce body temperature is recommended.
- Affected horses should be **removed to a cooler**, less humid environment.
- If exercise is necessary **do during the cooler period** of the day.
- After exercise, make sure the horse is ``cooled off`` adequately by hosing it down with water

Hyperhidrosis

- ***Etiology***
- Factors resulting in hyperhidrosis include

✓ high ambient temperature,
✓ infection,
✓ administration of drugs,
✓ excessive muscular exertion,
✓ pain,
✓ hyperadrenocorticalism and
✓ temperament.

Hyperhidrosis…

- ***Clinical signs***
- Excessive sweating may be generalized or localized over the neck, base of ears, around the nostrils and thighs.

- The coat appears dark and wet with sweat.

- Specific therapy depends upon the cause.

Hyperlipaemia

- Hyperlipemia describes the clinical syndrome characterized by **depression, anorexia, weakness, and hepatic failure**

- is associated with dramatic elevations in **blood lipid** and **fatty infiltration** of the liver and other parenchymatous organs.

- A condition of donkeys, mules and small ponies,

- the incidence rises with the age of the animal.

Hyperlipaemia…

- ***Pathogenesis***
- Hyperlipaemia occurs when the body's fat reserves are rapidly mobilized in response to:

✓ **Energy demands** in late pregnancy and early-peak lactation

✓ **Stress** (transportation or illness, especially laminitis or severe alimentary disease)

✓ **Malnutrition** (poor teeth) or following underfeeding, e.g. to prevent laminitis

Hyperlipaemia…

- Most hyperlipemic episodes are associated with
- ✓ reduced dietary intake and,
- ✓ in large horses, an azotemic state, whether pre-renal (most common), renal or post-renal, which exacerbates the condition by inhibiting the peripheral removal of **lipoprotein**s.

- During fasting, the triglycerides of adipose tissue are broken down into free fatty acids and glycerol, which are then released into the blood.

Hyperlipaemia…

- fatty acids taken up by the liver are
- ✓ oxidized completely to provide energy or are
- ✓ re-esterified to form triglycerides and phospholipids.

- These either remain in the liver or are released into the plasma as very low density lipoproteins (VLDLs).

Hyperlipaemia…

- **Risk factors**

➢ Body condition

➢ Stress

➢ Age younger < older

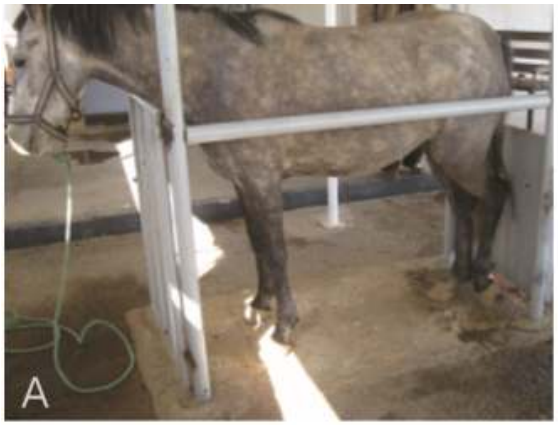
➢ Cushing‘s syndrome

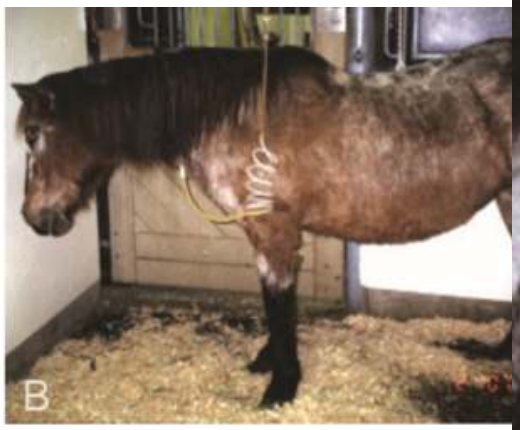
➢ concurrent disease

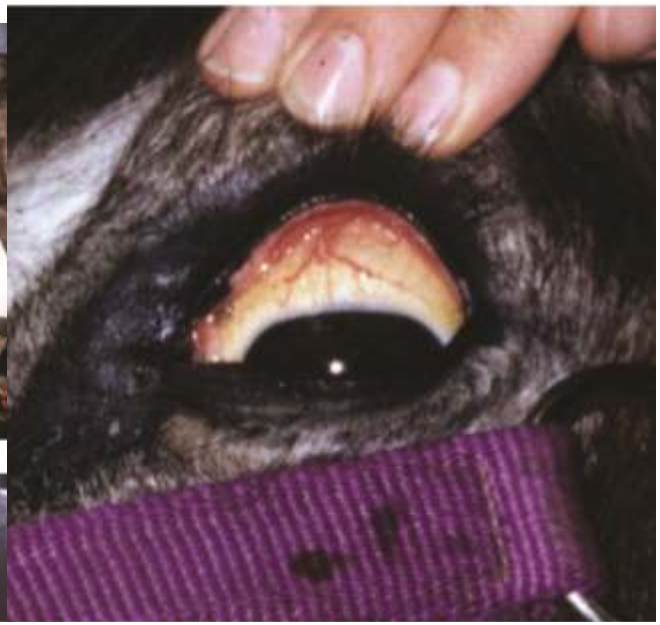
➢ Surgery

Hyperlipaemia…

- ***Clinical signs***
- Dull/depression, Inappetance/anorexia ‘ rapid loss of bodily condition, Abdominal pain
- The mortality rate can be high if treatment is not initiated immediately.

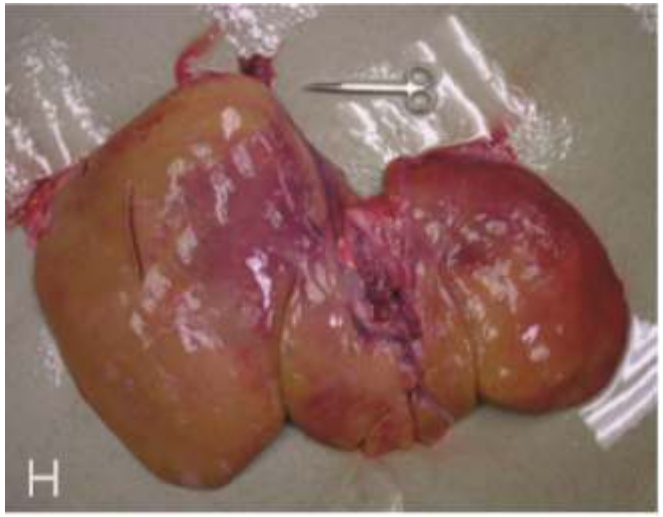






Hyperlipaemia…

Fatty and swollen liver is usually seen on postmortem examination



Hyperlipaemia…

- ***Diagnosis***
- History and clinical signs
- Plasma may appear cloudy or fatty
- Increase in plasma lipid levels (triglyceride levels >5mmol/L)
- Raised liver enzymes

Jugular blood sample
in plain tube

allow to stand for 3- 10
minutes

milky serum

Hyperlipaemia…

- ***Management***

1. Remove the precipitating cause:

(a) Wean lactating mares.

(b) Abort pregnant mares.

(c) Treatment of all cases with anthelminthics is sensible.

(d) Treat any underlying disease, especially laminitis.

Hyperlipaemia…

2. Prevent further lipid mobilisation:

(a) 5% Dextrose saline (5L/100kg/24h).

(b) Oral glucose (1 g/kg by stomach tube four times daily) may be sufficient in early cases.

(c) Feed slurry of complete pony cubes by stomach tube if it will not eat.

(d) Insulin therapy may help.

(e) Corticosteroids should not be used.

Hyperlipaemia…

(3) Promote movement of lipid into fat stores by

- using heparin, but use with caution because of the risk of haemorrhage.

(4) Treat dehydration and acidosis:

(a) Advanced cases become dehydrated with associated acidosis and azotaemia.

(b) lactated Ringer's solution to correct before moving on to glucose saline for maintenance.

Hyperlipaemia…

6. Symptomatic therapy

a. NSAIDs, analgesics, anti-ulcer medication
b. Multivitamines, anabolics
c. Antibiotics


END

Diseases and neurological disorder

By Reta Tesfaye (DVM, MTADM)

For AAU, CVMA 4th year DVM students

The nervous system

- The nervous system is classified as
- The central system: is composed of the brain and spinal cord
- The peripheral system: is formed by neuronal cell processes that extend from the central axis to the periphery.
-
- The autonomic nervous system: is formed by the cells and their processes that innervate the viscera of the body.

Disorders of the nervous system

- **Malformations**
- Malformations are the disorders that result from abnormal development of the nervous system.

- **Inflammations**
- the tissues' reaction to a microorganism or an immune system abnormality.

✓a bacterium, protozoa, or fungus, a viral agent or an immune system abnormality.

Disorders of the nervous system…

- **Injuries**
- Injuries occur when nervous tissue undergoes traumatic disturbance deriving from external or internal sources.

- **Neoplasias**
- Primary CNS neoplasias include the uncontrolled growth of nervous tissue cells.

- Metastatic neoplasia of the nervous system is the spread of primary neoplasms in other body tissues to the nervous system

Disorders of the nervous system…

- **Degenerations :** include the deterioration of cells due to

✓lack of blood supply (ischemia),

✓abnormal cellular metabolism caused by an inherited cellular defect,

✓exposure to exogenous toxins, and abnormalities in other body systems (renal disorders with uremia, diffuse liver disorders with hyperammonemia, cardiorespiratory disorders with hypoxia).

Neurologic Examination

- The **goals** of the neurologic examination are to

✓ determine whether disease of the nervous system exists

✓localize the lesion to a particular area of the nervous system,

✓describe and record the responses as a baseline for future evaluations.

❖repeated examinations over time may provide information that is useful in the diagnosis of various disorders.

Neurologic Examination...

- Signalment and history are important and should never be overlooked

Neurologic Examination...

- The evaluation is begun by a general observation of :

✓attitude and alertness (alert, depression, stupor, coma)

✓head and body position (Ex: head tilt)

✓position of the limbs (evenly and squarely placed under the horse)

✓symmetry of muscle development.

Neurologic Examination...

The components of the neurologic examination include

the cranial nerve examination

gait analysis

limb placing responses (one front hoof is lifted and placed across the othe
front leg. Horses should quickly move the hoof back into normal position)

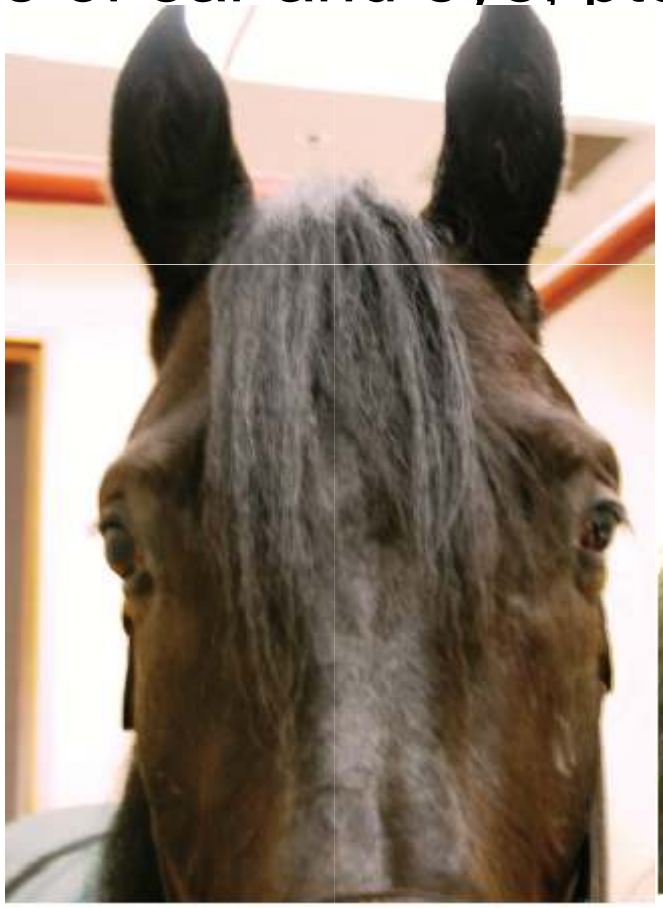
tail, anal, and panniculus reflexes. (stimulating Perianal skin and the skin
over the trunk, then observing for a “skinflick”response.)

the cranial nerve examination

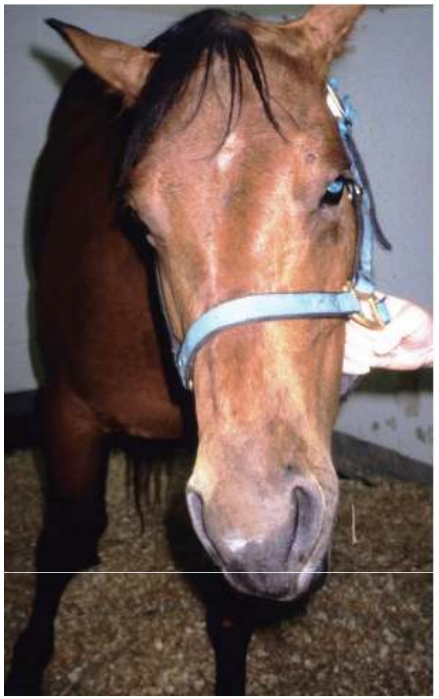
ssment	Nerve involved	Dysfunction
ace test	Optic, Facial	No eye blink; Blindness
lary light onse	Optic, Oculomotor	No response to bright light
er's syndrome	Cervical sympathetic	Sweating around base of ear and eye, ptosis
l sensation	Facial (sensory)	Failure to respond to stimulation of facial skin
l symmetry	Facial (motor)	Asymmetry of muzzle +/- ear droop, food impacted in cheek
ebral reflex	Trigeminal, Facial (motor)	Failure to blink
agmus	Oculomotor, vestibular	Nystagmus
low	Glossopharyngeal,Vagus	Inability to swallow
ue tone	Hypoglossal	Failure to withdraw tongue, or

Horner’s syndrome

- Sweating around base of ear and eye, ptosis

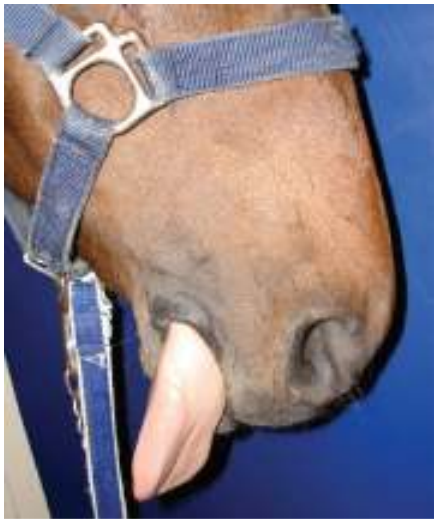


The cranial nerve examination



Facial symmetry (Asymmetry of muzzle, +/- ear droop, food impacted)

Tongue tone (Failure to withdraw tongue, or tongue weak when pulled



Evaluation of gait

- conduct the examination with the animal at rest, while walking and trotting

- Gait abnormalities that are observed commonly in horses with neurologic disease include

➢ataxia,

➢spasticity, and

➢weakness or paresis

Central nervous system Trauma

- is the most common cause of neurologic disease in horses

❖Cranial Trauma

❖Spinal cord trauma

Cranial Trauma

- Cranial injuries vary in severity and range from subtle alterations in mentation to unresponsive coma.

- Severe brain injury may occur with or without skull fractures

Causes

- collision with an immovable object (e.g., fence posts or another horse),
- falling down,
- falls over backwards,
- being kicked by another horse,
- Car accident
- Penetrating wounds

Causes…

- Although traumatic injury to the head is reported to be common in horses, subsequent CNS injury does not occur in all case.

Pathophysiology

- primarily mechanical damage (neuronal fiber and cell membrane disruption).

- the secondary injury characterized by

✓free radical formation,

✓excitatory neurotoxin release,

✓endogenous opiate release,

✓free fatty acid release,

✓loss of high-energy phosphates,

✓apoptosis,

✓CNS acidosis,

✓CNS ion imbalance.

Pathophysiology…

- Other important secondary events in traumatic brain injury (TBI) include

➢the development of an elevated intracranial pressure,

➢brain edema,

➢hypoxia, and

➢seizures.

Types of cranial trauma

- concussion, contusion, laceration, and hemorrhage.

1. Concussion is a short-term loss of consciousness and

✓is caused primarily by a direct blow to the head.

✓is reversible and occurs without an anatomic lesion.

Types of cranial trauma

2. Contusion is associated with the immediate primary mechanica injury and results from vascular and nervous tissue damage.

- occurs without major structural tissue disruption,
- may or may not be reparable.
- may be on the same or opposite side of the injury
- may result in intraparenchymal hemorrhage with later cavitation.

Types of cranial trauma

Cerebral hemorrhage and laceration

esult from penetrating wounds (gunshots) and fractures

nay be epidural, subdural (rare), intracerebral, or subarachnoid common).

Hematoma formation is of special concern because of the potential for devastating expansion within the rigid calvaria, as can occur with edema.

These processes displace brain tissue, and herniation, pressure necrosis, and brainstem compression are possible sequelae.

Types of cranial trauma

4. Hematoma formation is potentially devastating (hernation, pressure necrosis, and brainstem compression).

Physical examination

- The complete neurologic examination should include an assessment of
- mentation,
- cranial nerve function,
- posture, and
- ability to coordinate movements as well as its ability to regulate rate and range of motion.

Clinical signs

- Injury to the head may be visible.
- Neurological signs due to brain injury
  - Altered consciousness, e.g. coma, dullness, depression
  - Convulsions, seizures
  - Ataxia
  - Head tilt
  - nystagmus
  - Circling
  - Head pressing
  - Blindness
- hemorrhage from ears and nose and asymmetric vestibular signs

Treatment

- **Stablize the medical condition**

✓Maintain a patent airway

✓Control blood loss

✓Control seizures (detomidine & butorphanol (lowest doses), diazepam

Treatment

- **reducing intracranial pressure**

➢Osmotic diuretics

✓20% mannitol administered at 0.25 to 2.0 mg/kg intravenously over 20 minutes

✓glycerol administered at 0.5 to 2.0 mg/kg intravenously every 6 to 12 hours for 24 hours are effective in combating cerebral edema and increased intracranial pressure.

❖Patient receiving osmotic diuretics should be hydrated adequately.

❖The use of osmotic substances is warranted in any horse with worsening mental status, abnormal pupillary size, or development of paresis

Treatment

- furosemide has been found effective in decreasing intracranial pressure. A 1-mg/kg IV, q12h for 1 to 2 days

- Normal hydration status is required before furosemide administration.

- Horses with intracranial trauma often need fluid therapy to maintain hydration and blood pressure. Hypertonic saline (7.5%; 4 mL/kg) .
- Antibiotic treatment usually is given in cases of head trauma, especially when fractures are involved.

Treatment

- **Reducing free radical injury**
- administration of DMSO, 0.1 to 1g/kg IV as a 10% to 20% solution in saline or other polyionic fluid q12-24h for upto 5 days
- vitamin E, 20,000 units po q24h for adults).

- **anti-inflammatory and analgesic medications.**
- Flunixin meglumine, 1mg/kg q12h for 1 to 3 days
- magnesium sulfate (15 to 30mg/kg/h IV)
- **Fracture repair**
- **Euthanasia in sever cases**

Spinal cord trauma

- A thorough history
- Evaluate attitude, posture, and gait
- Palpation assessing evidence of localised pain,
- muscle wastage and asymmetry
- feeling for areas of patchy sweating or areas of reduced skin sensitivity .
- Hypalgesia and analgesia of cutaneous areas of the skin are good indications of a primary neurological lesion
- Local reflexes: useful in localising a lesion to a certain area of the central or peripheral nervous systems.
-

Spinal cord trauma

Causes

Falls (over a jamp, rearing over backward)

Collision with a fixed injury

Pathologic fracture resulting from osteomylitis

Spinal cord trauma

Clinical signs

Ataxia – lack of coordinated muscle movements

Postural deficits – wide-based stance, narrow-based stance, unstab

Paralysis and muscle atrophy

Weakness

Increased or decreased muscle tone

Hyperreflexia or hyporeflexia

Loss of pain – superficial and deep pain. Loss of deep pain is a very
poor prognostic indicator (this indicates extensive spinal cord injury)

Loss of autonomic process – urination and defecation

Treatment

- Recumbent equid

✓If the animal is recumbent, euthanasia is a necessity on welfare grounds.

✓Recumbency will rapidly result in muscle damage from lying in one position.

✓In cases of acute recumbency wait a minimum of 2 hours (with appropriate analgesia) before attempting to get the equid to stand.

✓If severe neurological damage has occurred then the clinical signs will not improve in this time

Treatment

- If treatment is attempted,

✓the equid should be able to stand, walk around without undue discomfort, be able to pass urine and faeces.

- Provide stall rest

- Dexametasone 0.1 to 0.3 mg/kg q12h IV for the first 1 to 2 days

Treatment

nitiate a course of NSAIDs once the effects of the steroid have vorn off to provide analgesia and long-term anti-inflammatory ffects.

lf there is no improvement after 1–2 weeks consider euthanasia or etirement as the chances of a full recovery are much reduced.

Recovery following nerve damage is often disappointing and slow

Broad spectrum antibiotics if patient is cases of recombency, a racture or wound

Treatment

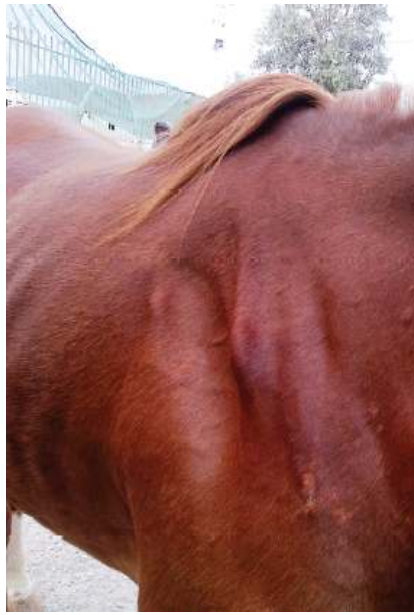
- Maintain hydration
- Cateterize and drain bladder if necessary
- Provide good nursing care
- Vit E, 2000 to 10,000 U/d PO per adult

Peripheral nerve trauma

Sweeny

This is seen in equids that pull carts or farm machinery with a yoke (a ring around the base of the neck).

The yoke can cause pressure and injury to the suprascapular nerve as it crosses in front of the scapula.



Clinical signs

atrophy of the supraspinatus and infraspinatus muscles

Radial nerve paresis or paralysis

- radial nerve paralysis cannot flex the shoulder joint or extend the elbow, knee, fetlock, or interphalangeal joints.

- The dorsum of the toe rests on the ground, and the elbow is dropped.

Radial nerve paresis or paralysis

- Evaluation of skin sensation may not be helpful.
- Atrophy of the triceps and other limb extensor muscles occurs after 2 weeks,
- Severely affected horses have difficulty rising and often collapse on the limb if it bears weight.

- More mildly affected horses may advance the leg by flinging or jerking it forward from the shoulder.
-

sciatic nerve paralysis

- sciatic nerve paralysis is most frequently associated with misplaced hypodermic injections and/or local abscessation at the site of the injection.

- Characterized by an inability to flex and advance the limb, such that it remains hanging behind the animal with the stifle dropped and extended.

- The dorsum of the foot rests on the ground, with the hock in flexion. The horse can bear some weight on the limbs since the femoral nerve innervates extensors of the stifle.

Femoral nerve paralysis

reported in mares after parturition, in horses after general anesthesia and rarely, after a penetrating wound to the caudal flank.

s characterized by inability to support weight on the limb or extend th stifle.

The horse rests the limb with all joints in flexion.

With bilateral involvement, the horse assumes a crouched position and has difficulty rising from recumbency.

Quadriceps atrophy will occur within several weeks.

Polyneuritis equi (cauda equina neuritis)

s characterized by chronic granulomatous inflammation of various
nerve roots, particularly the extradural spinal nerve roots of the caud
equina and cranial nerves.

The onset of clinical signs is usually insidious and slowly progressive

n the initial stages of the disease, signs of hyperesthesia around the
ail head and gluteal region and may rub and chew at this area.

ypalgesia or analgesia of the tail, perineum and penis or vulva an
gluteal area

Polyneuritis equi (cauda equina neuritis)

- Tail tone is absent and the urinary bladder, urethral sphincter, rectum, anal sphincter and penis or vulva are paralyzed. This leads to fecal retention and urinary incontinence.

- The bladder is atonic, distended and easily expressed.
-
- scalding of the perineum and inner thighs.

- In males, the penis may be relaxed and protruding, with decreased sensation of the perineal skin.

Treatment

- If damage to peripheral nerves results in in bruising or swelling, rather than complete transection, a full recovery may be possible with anti-inflammatory therapy and rest.

- If the nerve has been fully transected full recovery is unlikely.

- Treat all cases of peripheral nerve damage with anti-inflammatory medication, steroids initially, followed by a course of NSAIDs.

- Nursing care

Infectious conditions

Rabies.

- Is zoonotic disease
- is a fatal viral infection
- The virus can be present in saliva for up to 5 days before clinical signs appear
- Word wide in distribution
- Endemic in Ethiopia

Host affected

- all warm-blooded animals
- No sex, breed or age predliction
- Domestic dogs are the reservoir hosts in Ethiopia

Etiology

- Is caused by a ***Lyssavirus***
- Rabies virus (genotype 1) is the causative agent of the classical rabies
- Heat – labile
- Susceptible to most disinfectants

Pathogenesis

- Introduction of the virus === > replication of the virus at the site of introduction === > spread to the CNS via peripheral nerves === > multiplication === > travel peripherally along nerve axons.

- Destruction of spinal neurons results in paralysis

- when the virus invades the brain, irritation of higher centers produces manias, excitement, and convulsions.

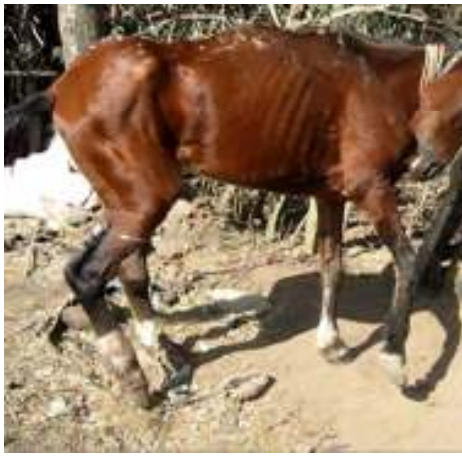
Clinical findings

- IP 2 weeks to several months
- Death within 3-5 days after clinical signs develop
- Clinical signs are Variable
- Do not intentionally eat or drink
- Rapid progression of clinical signs

Clinical findings

Common signs

- Aggression: Affected animals may attack inanimate objects or other animals, they may show sexual excitement.
- tenseness and hypersensitivity to sounds and movement
- Ataxia and paresis
- colic
- decreased sensation over the hindquarters
- paralysis of the tail, anus, and penis
- dribbling urine
- self mutilation
- recumbency; and eventually death



Clinical findings

Less common signs

- Abnormal vocalization
- Blindness
- Circling
- Drooling
- Head tilt
- Paddling while recumbent
- Pharyngeal paralysis
- Roaring
- Sweating
- Teeth grinding
- Tenesmus

Diagnosis

- Rabies should be considered when dealing with rapidly progressive neurologic disease.

- No pathognomonic signs for rabies

- History
- Clinical findings
- Laboratory findings

Laboratory Diagnosis

- **Fluorescent antibody test**
- Cell culture or mouse inoculation tests
- Direct immuno-histochemical test
- RT-PCR,
- ELISAs
- Electron Microscopy
- Rapid immunodiagnostic test

Managing a horse with rabies

- No treatment for rabies.
- Euthanasia If the disease is strongly suspected
- Minimize human exposure, especially individuals with open woun
- Wear protective clothing and gloves
- Wash hands thoroughly
- use sufficient restraint to ensure that you are not bitten

Tetanus

- Is highly fatal disease caused by exotoxins produced by *Clostridium tetani*
- Characterized by muscular rigidity
- Worldwide in distribution
- Common problem in Ethiopia

Host affected

- All species

- Horses is considered the most sensitive animal

Etiology

- Exotoxins produced by *C. tetani*
- Gram positive, anaerobic rod shaped organism
- Spores are heat and chemical resistant
- Survive in the environment months to years
- Ubiquitous in the environment (soil or feces)

Pathogenesis

- Infection by inoculation of the bacterium into unaerobic environment === > the spores germinate into vegetative form=== > proliferate and produce exotoxins.

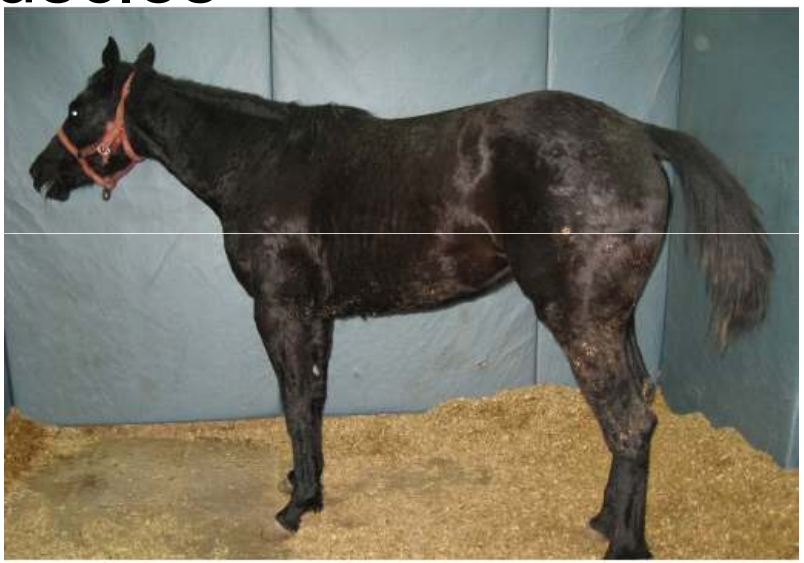
- Three toxins
- **Tetanolysin** promotes local tissue necrosis.

- **Tetanospasmin** diffuses to the systemic circulation and spreads to CNS and results in sign of tetany

- **Nonspasmogenic** exotoxin causes excessive stimulation of sympathatic nervious system

Clinical findings

- variable IP (Typically 2 to 21 days (median 7-10 days)
- Rigidity of head, neck, limb and tail muscles
- Head and neck extension
- Prolapse of third eyelid
- Flared nostril
- Erect ear , raised tailhead
- Inability to open mouth, dysphagia
- clinical signs exacerbated by stimulation
- Recumbency , respiratory failure and death



Diagnosis

- Clinical signs

- Anaerobic culture of *C. tetani* from the primary wound may be attempted

Treatment

1. Provision of safe and a quite environment

✓ Minimize stimulation (Dark stall and stuff cotton in the ears)

✓ Provide bedding

2. Elimination of *C. tetani. and unbound toxin*

✓ If wound is identified debride, clean/flush, open

✓ TAT (10,000 – 20,000IU) IV, IM, Sc, s.i.d

✓ Antimicrobials: penicillin (20,000 to 50,000 IU)for at least 7 days & Metronidazole (20-30mg/kg, q24hr, Po for 3 to 5 days)

Treatment

3. Sedation and muscle relaxation

- Acepromazine (0.04–0.10 mg/kg IV, IM q 4–6 h) until sever signs subside

- diazepam (0.01–0.4 mg/kg IV, as needed) in combination with xylazine (0.5–1.0 mg/kg IV or IM)

Treatment

- Nursing support
- Feed and water from a height to avoid need to lower head
- Ensure adequate water intake

Equine herpesvirus 1 myeloencephalopathy

- EHV 1 Causes respiratory disease, abortion and neurologic disease.
- is acute, and the signs are rapidly progressive (over 36–48 h).
- characterized by spinal cord signs alone, or less commonly, in combination with cranial nerve deficits.
- Often occurs in association with either of the other two
- Word wide in distribution

Hosts

- There is no breed or gender predisposition,
- foals are less likely to be affected with neurologic signs.
- Often multiple cases on the same farm within short period of time

History

- Abrupt onset of clinical signs that may progress rapidly
- a recent history of Upper respiratory tract infection *or*
- *abortion problems on the farm.*

Pathogenesis

- The pathogenesis is suspected to be an immune-mediated mechanism in the endothelium of blood vessels of the CNS.
- EHV-1 *is neurotropic*
- Causes vasculitis, thrombosis and subsequent ischemia of neural tissue.
- Impairment of blood flow results in hypoxia and dysfunction or death of adjacent neural tissue.

Clinical findings

- Fever (earlier or at the same time to as the neurologic signs

- Ataxia.

✓There is usually an acute onset of weakness and ataxia (in one or all limbs) or even recumbency.

✓Usually the hind limbs are symmetrically affected.

- Urinary bladder paralysis with urine dribbling and bladder distension

Clinical findings

- Decreased tail tone and perineal hypalgesia may be present, alor with subsequent constipation.
- Head signs (cranial nerve deficits, vestibular signs) are less frequent.
- are generally alert and have a good appetite.
- The signs may stabilize quickly or progress over several days.
- Complications associated with recumbency (bronchopneumonia, decubiti) may occur.

Diagnosis

- History and clinical findings
- CSF analysis
- Serology. Paired serum titers to EHV-1 showing a fourfold rise is good evidence of EHV-1 involvement.

Treatment

There are no specific treatments for EHV-1.

Supportive care is essential.

Stall rest.

Slightly affected animals that remain standing usually recover uneventfully with 2-3 weeks stall rest.

Recumbent animals are less likely to recover.

Extensive nursing care is needed to prevent secondary complications

pressure sores,

cystitis, and

hypostatic lung congestion.

Treatment

- Heavy bedding, sterile urinary catheterizations, manual rectal evacuationand frequent rolling of the animal are necessary for success.

- antimicrobial therapy for secondary infections (bronchopneumonia, cystitis) may be necessary

- Corticosteroids. controversial when indicated dexamethasone, 0.1–0.25 mg/kg b.i.d. IM or IV for 1–3 days


Thank
you

Diseases and disorders of integumentary system

Hanna Zewdu (DVM)

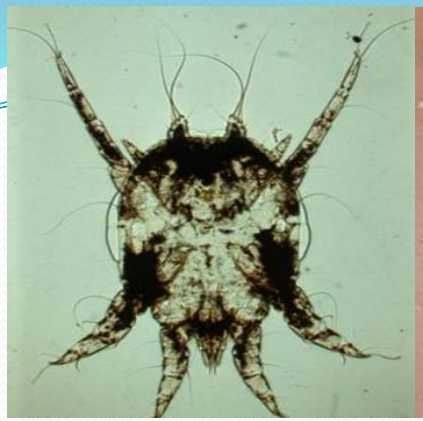
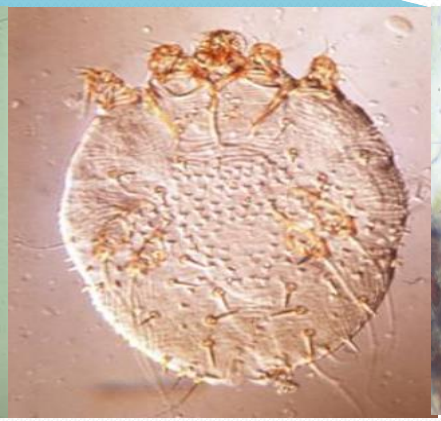
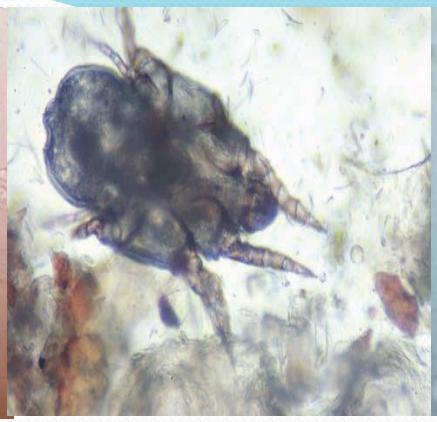
The Skin

- **largest** and most **visible** organ of the body
- Anatomic and physiologic barrier between animal and environment
- It provides protection from physical, chemical, and microbiologic injury
- Its sensory organ for heat, cold, pain, pruritus, touch and pressure

Parasitic skin conditions

1.Mange mites:

- Many are not host specific
- Prominent genera: Demodex, Sarcoptes, Psoroptes and Chorioptes
- Except for demodicosis, the most serious mite infestations are recognized during the late winter and early spring
- Crowding, prolonged stabling, and suboptimal nutrition all contribute to the relatively high mite burdens seen during this period


Psoroptic Sarcoptic Chorioptic Demodex

- **Psoroptic mange** is a pruritic dermatitis and/or otitis externa of horses and Caused by P. equi, P. natalensis
- **Sarcoptic Mange**: caused by Sarcoptes scabiei var. equi
- **Chorioptic Mange** (Termed leg mange), caused by Chorioptes equi, this is the most common type of mange in the horse

- **Demodectic mange (follicular mange):** caused by Demodex caballi (eyelids and neck area) and D. equi (found on the trunk)

N.B Demodectic mites are normal residents of the skin

CLINICAL SIGNS

- Transmission of mange mites is by direct or indirect contact
- **Psoroptic mange**: Infested horses may be asymptomatic or may show signs of ear disease, mane and tail Pruritusis
- **Sarcoptic Mange**: The chief clinical sign is pruritus, which usually begins on the head, ears, and neck and spreads caudally
- **Chorioptic Mange**: Clinical signs are seen on the distal hand limbs with the fetlocks, pasterns and tail being particularly affected
- **Demodectic mange:** Clinical signs mainly seen in immunocompromised (debilitation, concurrent disease, poor nutrition, or stress)

DIAGNOSIS

- The differential diagnosis includes
  - lice infestation,
  - fly-bite dermatoses,
  - allergic skin diseases,
  - oxyuriasis
- Definitive diagnosis is based on history, physical examination and examination of skin samplings collected by scraping, brushing, or combing
- Skin should be squeezed firmly and scraped deeply until blood is drawn(Demodicosis)

CLINICAL MANAGEMENT

- Conventional acaricides (Diazinon, malathion) sprays or dips can be effective when applied every 7-14 days for 3-4 weeks
- Ivermectin (0.2 mg/kg S/c) is very effective in the treatment of mange mites (two dose; a second dose 14 days after the initial treatment. Since ivermectin and other parasiticides do not kill eggs of the mites second therapy is cruial)
- All animals that have been exposed to infected animals should be treated, since asymptomatic infection may occur
- Infested bedding is eliminated and that all grooming equipment is segregated

2.TICK INFESTATION

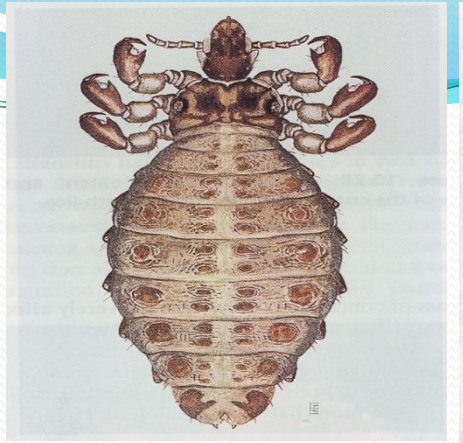
- Ticks may harm their hosts by:

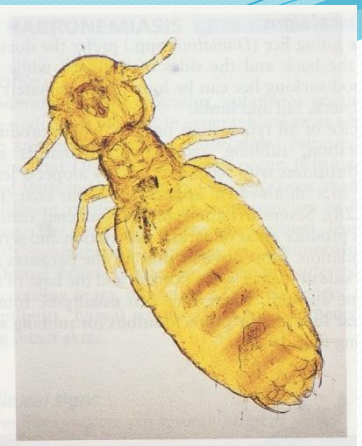
1. Bite injuries, which may predispose to secondary infections and myiasis
2. Sucking blood (a single adult female tick may remove 0.5-2 mL of blood)
3. Transmitting various viral, protozoal, rickettsial, and bacterial diseases
4. Causing tick paralysis

Tick Infestation cont…

- **Treatment and control**
  - Spraying , dipping or pour-on with Conventional acaricides e.g.Diazinon
  - Burning of pasture
  - Isolation of infested host
  - Avoid pasture with many ticks as long as possible
  - Maintain animal houses clean

3.LICE INFESTATION (PEDICULOSIS)





- Two species of lice may infest horses and other Equidae:
- Haematopinus asini: a sucking louse, which feeds on blood obtained by puncturing the skin; and
- Damalinia equi: which survives by feeding on scale and skin debris

Clinical Signs

- Varying degrees of scaling, patchy alopecia, erythema, excoriation and severe pruritus occur
- In most cases, the pruritus is mild to moderate. With close inspection of the involved areas, the nits or adults can be seen

Diagnosis

- Isolation of lice can be difficult when numbers are low, as adult lice are relatively small and being either pale pink or gray, merge easily into the coat
- A long and careful search of the coat under strong light is often essential to find the parasite
- Presence of eggs or nits firmly attached to hairs on the trunk and neck, or the base of the mane and tail

Treatment and control

- Pyrethrins or organophosphate in dust/solution form
- Systemic ivermectin is likely to be effective against sucking, although not necessarily the chewing lice
- Particular attention should be paid to stable hygiene as infestation can be transmitted on blankets, rugs and grooming equipment

Bacterial diseases

1.Ulcerative lymphangitis

- Caused by Corynebacterium pseudotuberculosis *which is a gram-positive, pleomorphic pathogen that survives and replicates in phagocytes*
- It is a soil-borne organism that gains access to tissue through wounds or insect bites
- Contact and inanimate transmission/flies is probable
- Infection occurs through abrasions on the lower limbs and is more likely when horses are crowded together in dirty, unhygienic quarters

Clinical findings

- Swelling and pain of the pastern(lamness)
- Development of subcutaneous tissue nodules especially around fetlock
- Creamy green pus from ruptured nodules
- Ragged edges and a necrotic base ulcers
- Lymphatics draining the area become enlarged and hard

Diagnosis

- Definitive diagnosis is based on direct smears and culture
- Histopathologic findings include diffuse or nodular, suppurative to pyogranulomatous dermatitis

Differential diagnosis

- Epizootic lymphangitis
- Glanders
- Sporotrichosis
- Rhodococcus equi

Treatment

- Local treatment of ulcers is the usual and most effective procedure but parenteral injections of penicillin or tetracycline may be necessary in severe cases

Control

- Good hygiene and management practices
- Careful disinfection of injuries to the lower limbs
- Affected animals should not be introduced to free area
- Early wound treatment and fly control

2.DERMATOPHILOSIS

- Dermatophilosis is a common infectious, superficial, pustular and crusting dermatitis caused by *D.congolensis*
- *D. congolensis* is a gram-positive, facultative anaerobic actinomycete
- Dermatophilosis is a contagious disease. The incubation period averages about 2 weeks but may be as short as 24 h or as long as 34 days
- Soil could act as a temporary reservoir of the organism
- Dermatophilosis is a rare zoonosis

Dermatophilosis cont.....

- Skin damage and moisture are impotant factor for the initiation and development of dermatophilosis
- Typical sources of moisture are rain, wetting and sweating under blankets and harnesses
- Important sources of skin damage include biting flies and arthropods, prickly vegetation and other inflammatory skin diseases
- Moisture causes the release of the infective, motile, flagellated zoospore form of the organism

Clinical Signs

- Distributed worldwide
- There are no age, breed, or sex predilections
- Follicular and nonfollicular tufted papules
- These lesions rapidly coalesce and become exudative, which often results in large ovoid groups of hairs becoming matted together (paint brush effect)
- Active lesions contain thick, creamy, whitish, yellowish or greenish pus, which adheres to the skin surface and to the under surface of the crusts

Clinical Signs cont....

- In horses exudative and crusted lesions are commonly found over the:

1. Rump and topline ("rain scald" typically associated with rainfall)
2. Saddle area (associated with trauma and increased moisture under tack)
3. Face and neck (associated with trauma and moisture under tack)
4. Pasterns, coronets, and heels, typically associated with poorly drained pastures and muddy paddocks

Diagnosis

- The differential diagnosis includes dermatophytosis, staphylococcal folliculitis, demodicosis, pemphigus foliaceus
- Definitive diagnosis is based on cytology, skin biopsy,and culture
- Direct smears of pus or saline-soaked and minced crusts may be stained with new methylene blue or Gram stain
- D. congolensis appears as railroad track appearance

Clinical Management

- Twice daily Wash with topical antibacterials e.g. chlorhexidine 2% and dry the skin
- Severe cases: Systemic antibiotics/Procaine Penicillin at 22,000 IU/kg intramuscularly twice daily for 5 to 7 days
- Eliminate exposure to excessive moisture and skin trauma
- Isolate affected animals

3.Epizootic lymphangitis /Equine histoplasmosis

- Epizootic lymphangitis is a contagious, chronic disease of equidae (horses are highly affected)
- Characterized clinically by spreading, suppurative, ulcerating pyogranulomatous dermatitis, lymphangitis and lymphadenitis
- It is caused by a dimorphic fungus, Histoplasma capsulatum var. farciminosum
- Lesions particularly seen in the neck, legs and chest
- Bedding, grooming, utensils, blankets, and harness can serve as fomites
- The fungus invades subcutaneous tissue, set up a local granuloma or ulcer and spreads along the lymphatic vessels

Clinical Signs

- Route of the entry of the organism determines the form of the disease
  - Dermal Form (through traumatized skin)
  - Ocular Form (biting flies feeding on ocular discharge and secretions)
  - Pneumonic Form (through the inhalation of the organism)
- Freely moveable intradermal nodules, approximately 2 cm in diameter
- Cording or thickening of the lymphatics (in chronic infections)
- Purulent nodules which usually ruptures externally realizing thick, yellow pus
- Purulent lacrimal discharge and tear scalding (ocular)

Diagnosis

- Laboratory tests
- Histopathology
- Stainings

- Differential dx:
  - Glanders
  - Ulcerative
  - Sporotrichosis

Treatment

- Early cases: excision/incision of affacted parts followed by frequent local application of tincture of iodine
- Early cases can be treated with sodium or potassium iodide
- Amphotericin B has also been used, but it is more expensive

Control

- Disinfection of infected equpment and premises by 1% sodium hypochlorite, formaldehyde and phenolic disinfectants and fly control
- Removal of all contaminated materials (grooming equipment or harnesses)
- Strict quarantine infected animals

4.Glanders(farcy)

- Glanders is an infectious, contagious, highly fatal disease characterized by the formation of nodules of the skin, lung and other internal organs, and ulcers of the upper respiratory mucosae of equines
- It is caused by the bacteria, *Burkholderia mallei*
- Glanders causes respiratory infection and skin lesions in equine and humans
- Flies may play an important role in the spread of glanders when feeding on the oozing lesions of an infected horse
- Horses tend to develop chronic form, mule the acute form

Clinical signs

- Through ingestion or contamination of skin abrasions the organism localizes in skin, lung, nasal mucosa and other organs
- **Horses**: Ulcer of nasal mucosa (Star shaped scar)
- Subcutaneous nodules (distal limbs or abdomen) which ulcerate and rupture discharging thick brown, honey like exudates
- Cording of lymphatics (especially in hind legs)
- Donkeys and mules: Acute system illness with pyrexia, cough, nasal discharge, dyspnea and often death
- Chronic/respiratory form: coughing, intermittent pyrexia, nodular lymph node enlargement and labored respiration

Diagnosis

- History
- Clinical signs
- Mallein test
- Bacteriological culture

Treatment and control

- Treatment is usually contraindicated b/c of the possibility of the carrier states
- Destroy or disinfect contaminated materials
- Requlat Mallein test and slaughter the reactors

5.Dermatophytosis "Ring worm"

- *Trichophyton equinum* is the most common cause of dermatophytosis in horses where as *Tr. mentagrophytes*,*Tr. verrucosum* in donkeys
- A higher incidence is seen in hot, humid climates than in cold, dry climates
- Mainly young or immuno-suppressed
- Dermatophytes are transmitted by contact with infected hair and scale or fungal elements on animals, in the environment, or on fomites
- Dermatothytosis has zoonotic importance

CLINICAL FINDINGS

- One or many circular/round patches of alopecia with variable scalding and crusting
- Classic ring lesion with central healing" b/c the fungi die under crust and move to periphery" and fine follicular papules and crusts at the periphery
- Pruritus is usually minimal or absent

Diagnosis

- Preliminary diagnosis is based on history, clinical signs and positive microscopy (ectothrix spores coating a hair shaft)
- Fungal culture for confirmatory diagnosis

Treatment and control

- Most dermatophyte infections are self-limiting
- Topical applications: Whitfield's ointment, weak solution of iodine
- Clipping wide margins around lesions followed by bathing in chlorhexidine– miconazole shampoo
- Correction of stressful and unsanitary conditions
- Separation of the affacted animals from non- affacted ones

Sarcoids

- The equine sarcoid is by far the most common nodule affecting the horse's skin; it also affects the skin of ponies, donkeys and mules
- Bovine papilloma virus (BPV) types 1 or 2 is a causative agent of sarcoid in equines
- It is the most common skin neoplasm of horses, donkeys and mules
- Transmission by close contact and infection of wounds
- Sarcoids frequently occur in areas subjected to trauma (wounds, tack, insects) and spreads to other area through biting, rubbing, fomites and insects
- The majority of affected animals are 7-years-old and younger

CLINICAL FINDINGS

- Sarcoid appears as single or multiple/ clusters lesions on the skin which is differ with forms of sarcoid
- The majority lesions occur on the lower limbs but also on the lips, eyelids, head , penile sheath, neck and around the base of the ears
- Forms of sarcoids:
    - Verrucous (warty) sarcoid
    - Fibroblastic (“Proud Fleshlike”) sarcoid
    - Occult (Flat) sarcoid
    - Nodular Sarcoid
    - Malevolent (“Malignant”) Sarcoid
    - Mixed Sarcoids

CLINICAL MANAGEMENT

- Surgical excision: but recurrence is common
- Cryosurgery
- Radiation therapy
- Laser therapy or chemotherapy, immunotherapy or combinations of these
- Application of ligation for raised and necked sarcoids at its base using non absorbable suture material

Allergic and immune mediated skin diseases

Pemphigus foliaceus

- This is a relatively common immune-mediated skin disease in the horse
- Cases in animals less than one year of age tend to be less severe, respond well to therapy and may spontaneously regress
- Antibodies directed against epidermal cell surface antigens result in loss of intercellular cohesion and the formation of intraepidermal vesicles or vesicopustules.
- Whilst the primary lesion is a vesicle or vesicopustule, the usual presentation is of a crusting, scaling dermatosis that often begins on the face and limbs, but commonly becomes generalised
- In some cases lesions are limited to the coronary band, chestnut and ergot regions
- Oedema of the extremities and ventrum may be present, often out of proportion to the surface skin changes

Environmental skin diseases

Photosensitization

- Ingestion of exogenous photodynamic agent/ from lush green stage and rapidly growing plants
- Exposure to a sufficient amount of certain wavelengths of Ultra Violet Light then, Dermatitis will occur
- Faulty excretion through the liver: metabolic products accumulate the tissues

Pathogenesis:

- Penetration of light rays to sensitized tissues causes local cell death and tissue edema
- Irritation is intense because of the edema of the lower skin level, and loss of skin by necrosis or gangrene and sloughing is common in the terminal stages

CLINICAL FINDINGS

- Intense irritation and the animal rubs the affected parts, often lacerating the face by rubbing to objects
- Local edema, Swelling of the eyelids
- Increased lacrimation/initial sign
- Skin lesions are initially erythema, followed by edema and crust
- Lesions are restricted to the unpigmented areas of the skin and to those parts which are exposed to solar ray

Treatment

- Primary treatment includes:
  - Immediate removal from direct sunlight
  - Prevention of ingestion of further toxic material
  -administration of laxatives to eliminate toxic materials already eaten
- Local treatment: Inflammatory drugs (NSAIDs) or corticosteroids parenterally

Neoplastic skin conditions

Squamous Cell Carcinoma

- It is a common malignant neoplasm of the horse arising from keratinocytes
- Mostly occurs at mucocutaneous junctions/eyelid and external genitalia

CLINICAL FINDINGS

- Prevalence ↑ with age
- Lesions are usually solitary, poorly circumscribed, beginning as nonhealing, enlarging, granulating ulcers often cauliflowerlike masses
- Necrosis and a foul odor are common

CLINICAL MANAGEMENT

Surgical excision, cryosurgery, laser surgery, radiotherapy, chemotherapy,immunotherapy or combinations of these

Wound

- Wound is an open mechanical injury of the skin/epidermis, underlying tissues and organs
- It is characterized by pain, gaping, bleeding and functional disturbance

- Causes?
- Types?
- Treatment?

Group one

- Describe the different types of mange in equines and mention some disorders they cause on the animal and treatment plans in each case.

Group Two

- Describe the different types of lice in equines and mention some disorders they cause on the animal and treatment plans in each case

Group Three

- How do ticks transmit diseases? Mention some identification characteristics of the genera and mention some diseases transmitted by them (ticks).

Group Four

- What is ulcerative lymphangitis? How will you differentially diagnose it from Epizootic lymphangitis, Glanders and Sporotrichosis?

Group Five

- What is Dermatophilosis? How will you differentially diagnose it from Dermatophytosis, Staphylococcal folliculitis and demodicosis?

Group Six

- Identify different types of Sarcoids in equines and possible ways of treatment of them.

Group Seven

- Identify Different Neoplastic skin conditions of equines and describe the possible treatment plans of each.

Group Eight

- Mention some Allergic and immune mediated skin diseases and describe the possible treatment plans for them.

Group Nine

- What is wound? What causes it? What are the different types? Mention the treatment protocol of them.

End!

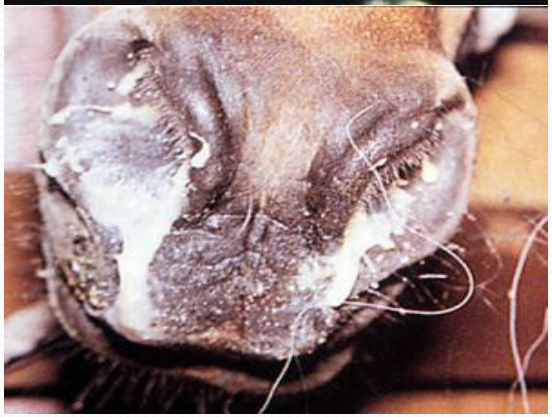
Disease and disorders of the respiratory tract of equines

Hanna Zewdu (DVM, MSc)

2020

- Most equids are active, **athletic animals** and need to efficiently and effectively breathe large quantities of air to perform to their full potential. This requires that the respiratory system be as healthy as possible.
- It has been estimated that respiratory problems are second only to lameness as a cause of poor performance.
- Problems with respiration are particularly significant, because many respiratory problems go unnoticed, particularly in the early stages.

Clinical signs



- Incresed/decreased respiratory rate
- Effort and patern of respiration
- Inspiratory/ expiratory noise
- Dyspenia
- incresed flaring of the nostrils
- Incresed abdominal muscle contraction
- nasal discharge (serous ; mucopurulent/ unilateral; bilateral)
- coughing (frequency/ character)

Aetiologies

- infectious agents
  - Primary or secondary bacterial
  - viral
  - Parasitic
  - Fungal
- allergic
- Physical (trauma, aspiration pneumonia, foreign bodies,…)
- Poisons (eg. Carbon mooxide)
- Physiological (metabolic, environmental,…)

Diagnosis:

1. *History taking* is important although not 100% realible
    - Number of animals affected
    - duration –(acute or chronic)
    - Signs seen by the owner and changes/ progression/ weight loss
    - Presence of disease locally
    - Movements of the animal recently
    - Environment
    - Vaccination status

2. *clinical examination*

- Rate and character of breathing (adult 8-20/ min; foals 30/min)
- Rectal temprature (37.2 – 38.5)
- Type and volume of nasal discharge
- Type and incidence of cough
- Lymph nodes (swelling, heat, pain, …)
- Auscultation
- Percussion (to check pleural effusion, start from the sinuses)*check for dull sound
- Effect on exercise

3. *aids to diagnosis*

- Haematology
- Endoscopy
- Microbiology, Virology, Serology
- Response to treatment
- Response to managment
- Tracheal wash
- Radiography

DEVELOPMENTAL DISORDERS

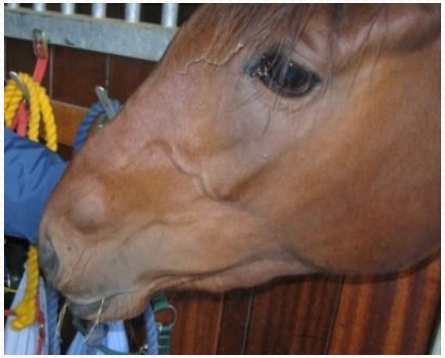
Guttural pouch tympany

- Guttural pouch tympany occurs when the guttural pouch becomes **abnormally filled** with air, causing **non painful swelling** just behind the jaw.
- The condition occurs in **young horses** (from birth to 1 year of age) .
- It may be caused by inflammation or by a congenital (present at birth) defect that allows air to enter the pouch but **prevents it from returning** to the pharynx.
- **carrying the head in an extended position.**
- The diagnosis is based on the signs and x-rays of the skull.
- Treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) and appropriate antibiotics is successful in most horses in which inflammation is the cause.
- If tympany is due to a congenital defect, **surgery** is required

Non Infectious disorders of upper respiratory tract

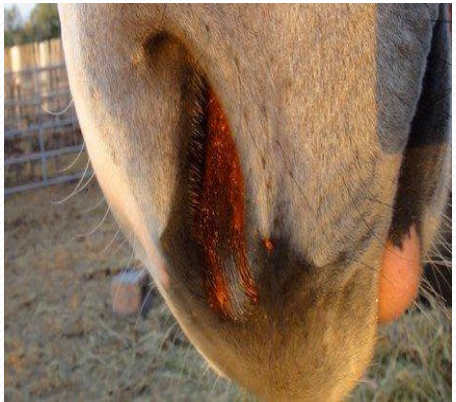
Atheroma

- Sebaceous cyst that creates a firm round swelling in the nasoincisive notch (false nostril.
- Treatment is usually requested from a cosmetic point of view



Epistaxis

- Bleeding from nose
- Trauma, forein bodies,….



Infectious diseases of the upper respiratory tract

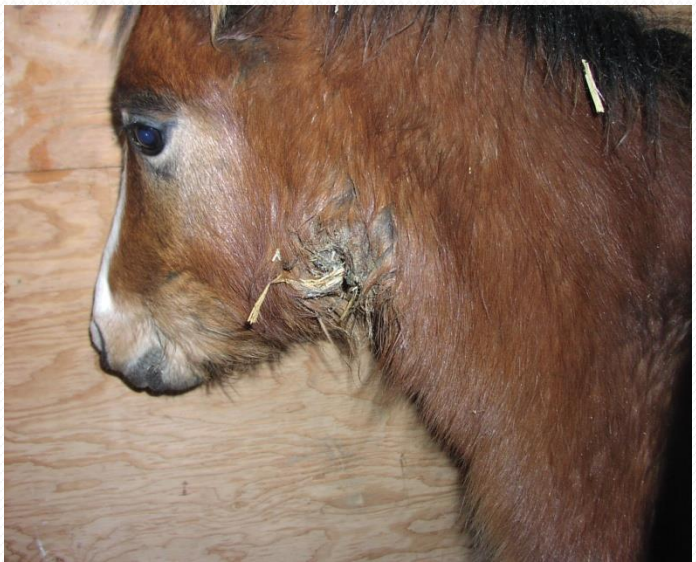
1. strangles
2. Guttural pouch empyema
3. Guttural pouch mycosis
4. Nasal aspergilosis

1. Strangles/Equine distemper

-It is acute, highly contagious bacterial infection of the upper respiratory tract of Equidae

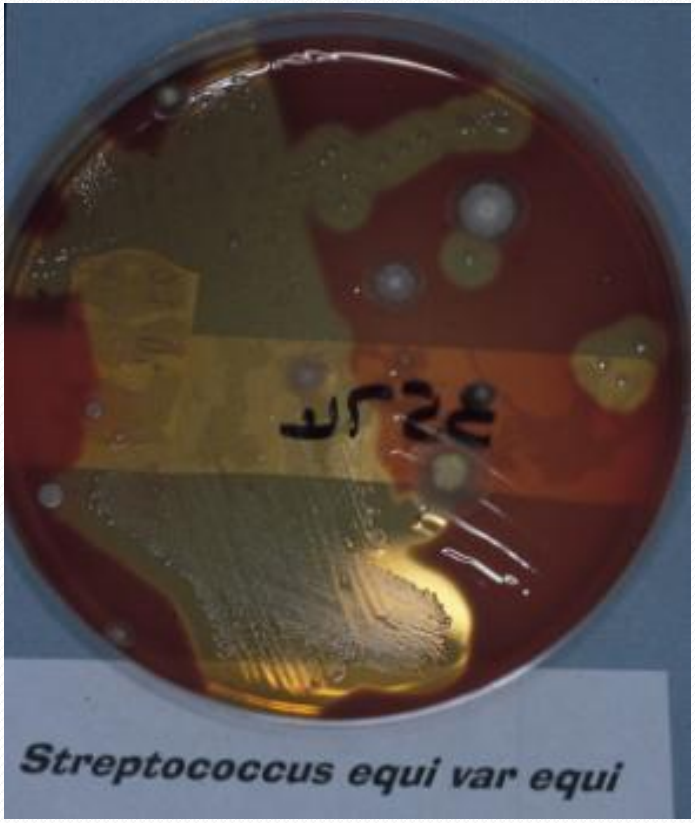
-Most common in equids 1 to 5 years of age

-It is characterized by *mucopurulent inflammation of the nasal passages, pharynx, and associated lymph nodes*



Etiology

- *Streptococcus equi subsp. equi is a Gram positive cocci*
- It is a primary pathogen of the equine respiratory tract.



Epidemiology

–Infection occurs via the oral cavity (*ingestion*) or upper respiratory tract (*inhalation*)

–Purulent discharges from equids with active and recovering strangles are an important source of new infections among susceptible equids

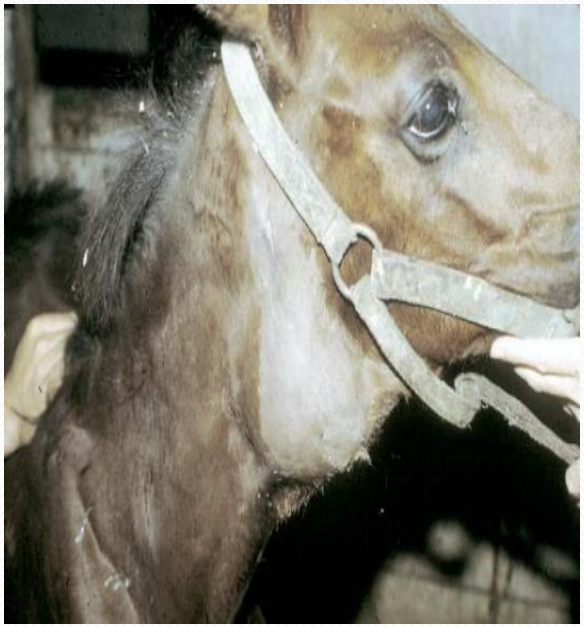
–Some horses may become long-term asymptomatic carriers and may have persistent guttural pouch infection with *empyema or chondroids*.

-Infection rates increase with:

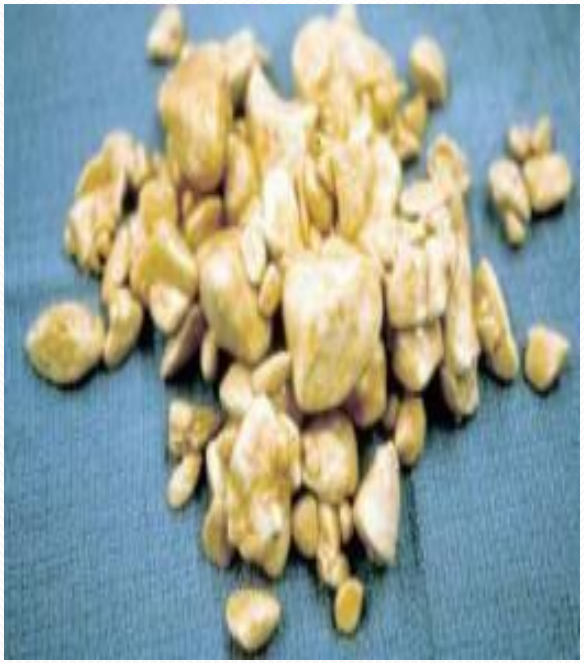
- increasing group size
- increased movement of horses
- increased mixing of horses
- communal feeders and drinkers
- younger horses

Clinical signs

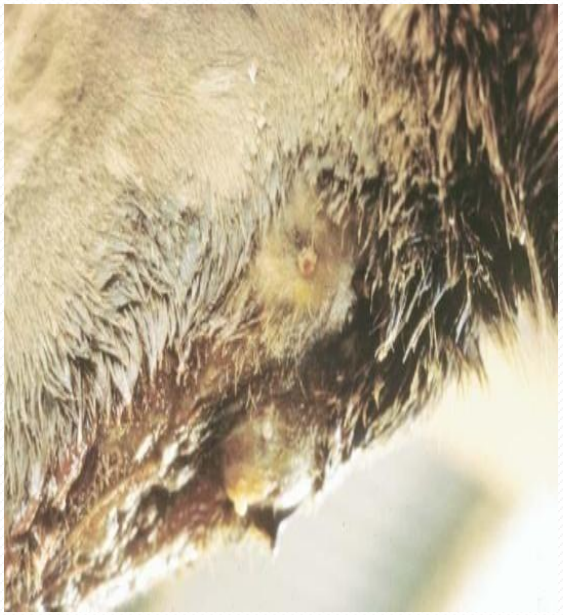
- ***Marked fever*** (up to 40^{o}_{C}) develops during the acute phase and may subside until the lymph nodes abscess, at which time a second wave of fever may develop.
- **Bilateral**, serous to mucoid nasal discharge which later becomes mucopurulent as the disease progresses
- **Moist cough** may develop in some cases
- The submandibular and retropharyngeal lymph nodes are involved most often and become enlarged, firm, and painful
- Abscessation of these nodes typically ruptures in to the pharynx or guttural pouch (empyema)



Guttural pouch empyema



Chondroids



Submandibular lymph node abscessation

- Metastatic or internal abscesses "**bastard strangles**" can occur if *S. equi subsp. equi gains access to the circulation and seeds internal* ***lymph nodes or other organs, most commonly the lungs, mesentery, liver, spleen, kidney, brain.***

Diagnosis

–Clinical signs determine presumptive diagnosis

–Definitive diagnosis requires isolation of *S. equi subsp. equi via bacterial culture of nasal swabs, nasal washes or pus aspirated from abscesses.*

–PCR and serology (ELISA) can be used in conjunction with culturing (the gold standard).

Differential diagnosis

–Viral respiratory tract diseases, bacterial pneumonia, guttural pouch empyema and abscessation

Treatment

- Treatment is dependent on the stage and severity of disease at the time of presentation
- In most cases symptomatic treatment and nursing care are sufficient
- The affected animals should be kept in a clean dry environment and offered soft and palatable feed
- Abscesses are encouraged to mature and rupture by use of poultices and hot packs.
- Surgical lancing of the submandibular abscess can be performed
- Lavage with 3-5% povidone iodine solution will facilitate resolution of discharge

- Antibiotic of choice is **procaine penicillin** 22,000 IU/kg, IM, q12h or **Crystalline penicillin** 20,000 IU/kg, IV, q6h for 10 to 14 days.
- Others include **trimethoprim-sulfadiazine** (TMS) 15 mg/kg, PO, q12h and ceftiofur 5 mg/kg, IM, q12h.
- Most fully recovered equids develop a solid immunity for a period of five years
- Vaccination is available. It reduces morbidity and severity of the disease but does not prevent infection.

2. Guttural pouch empyema

- Guttural pouches are *paired extensions of the eustachian* tubes that connect the pharynx to the middle ear.
- Empyema of the guttural pouch is the presence of **purulent material or chondroids** within one or both guttural pouches.
- **Chondroids** are solid concretions that consist of purulent material.
- Guttural pouch empyema can affect **horses of any age** but usually occurs in young animals.
- It usually occurs secondary to upper respiratory tract infections

Clinical signs

- Bilateral mucopurulent nasal discharge, often persisting after recovery from an URT infection and fever
- Distension of the affected pouch into the pharynx may produce obstructive dyspnoea, abnormal respiratory noise and dysphagia
- The discharge is greatest when the head is lowered and when external pressure is applied to the parotid region on the affected side

Diagnosis

- **Endoscopy** may reveal mucopurulent discharge from the guttural pouch opening
- **Aspiration of the pouch contents** confirms the diagnosis and allows culture and sensitivity testing
- **Radiography** reveals a distinct fluid line in the guttural pouch

Treatment

- Acute empyema may respond rapidly to **systemic antibiotics**
- If drainage persists or the condition is chronic when diagnosed, **lavage** via an indwelling catheter is indicated
- **Surgical draining** is indicated if the discharge persists after several days of lavage(**chondroids** cannot be removed by lavage)
- Surgical intervention is a last resort because of the risks of iatrogenic nerve damages

3. Guttural pouch mycosis

- Guttural pouch mycosis is a fungal infection of the pouch wall that often interferes with the associated neurovascular structures.
- It is usually associated with ***Aspergillus nidulans***, *although other fungi have been also implicated.*
- Infection is usually **unilateral** and typically involves the roof of medial compartment overlying the internal carotid arteries and sometimes the external carotid artery
- ***Epistaxis*** at rest can occur due to invasion of arterial wall, and *fatal haemorrhages* may result
- Neuropathies including pharyngeal paralysis and dysphagia may also occur

Diagnosis

- **Endoscopic examination** of the pouch interior is important in confirming the diagnosis
- The lesion appears as brown, yellow or black and white necrotic, diphtheritic membrane raised from the surface of the pouch wall

Treatment

- *Direct placement* of topical antimycotic medication (itraconazole) on the lesions via endoscopic guidance is the preferred method of administration.
- Ligation of the affected arteries are recommended

4. Nasal aspergillosis

- Mycotic plaques caused by *Aspergillus spp. (usually A. fumigatus) may occasionally occur on the mucosa of the nasal passages.*
- Lesions of the nostrils are often **ulcerative granulomas** and sometimes concurrently occur with pulmonary aspergillosis.
- Affected animals have a slight purulent nasal discharge with or without epistaxis
- Diagnosis of aspergillosis is based on identification of the organism in tissue biopsy and exudates.
- Daily topical natamycin with oral itraconazole have provided full recovery from the disease

Non Infectious disorders of lower respiratory tract

1. Exercise induced pulmonary hemorrage
2. Acute allergic airway diseases
3. Chronic obstructive pulmonary diseases

1. Exercise induced pulmonary Haemorrhage

- EIPH is **bleeding from the pulmonary vasculature** as a consequence of the cardiopulmonary changes during exercise.
- It occurs in activities that require strenuous exercise for short periods of time

Clinical signs

- **perform poorly.**
- Respiratory distress
- increase in the rate of swallowing after exercise (to clear blood ascending from the lower airway)
- Rarely blood discharges from the nostrils (epistaxis)

Diagnosis

–Finding of blood upon **tracheoscopy** (30-120 minutes after exercise) or by detecting increased intracytoplasmic hemosiderin content in alveolar macrophages (hemosiderohages) with macrophagic bronchiolitis and fibrosis

–Thoracic radiography demonstrates **alveolar or mixed alveolar-interstitial opacities**

Differential diagnosis

–Other causes of dyspnea, viral respiratory disease, pharyngeal lymphoid hyperplasia, RAO and other causes of epistaxis (e.g., guttural pouch diseases, nasal tumors, ethmoid hematoma)

Treatment

–Furesmide appears to decrease the severity of haemorrhage (administered 3-4 hours before exercise)

2. Inflammatory airway disease (IAD)

- IAD describes a **heterogeneous group** of inflammatory conditions of the lower respiratory tract that appear to be primarily noninfectious.
- IAD is a common cause of impaired performance.

Etiology

–allergic airway disease, recurrent pulmonary stress, deep inhalation of dust, noxious gasses (NH_3, H_2S) atmospheric pollutants (ozone), and/or persistent respiratory viral infection

Clinical signs

- chronic cough
- mucoid to mucopurulent nasal discharge
- Fever and auscultable pulmonary abnormalities are rarely observed
- Poor exercise tolerance at maximal speed

Diagnosis

- clinical signs observed
- Endoscopic examination reveals mucopurulent exudate in the pharynx, trachea, and bronchi
- Bronchoalveolar lavage(BAL) is performed to characterize the type of pulmonary inflammation (cytologic evaluation)

Treatment

–The type of inflammation in BAL(cytologic evaluation) dictates therapeutic plan

–A mixed inflammatory cytologic profile is treated with immunostimulant or immunomodulatory drugs (interferon-alpha)

–An eosinophilic or mast cell cytologic profile is treated with a combination of anti-inflammatory (systemic corticosteroids) and immuno-suppressive drugs (sodium cromoglycate or nedocromil sodium)

3. Recurrent Airway Obstruction (RAO)

(Heaves, Chronic obstructive pulmonary disease)

- RAO is also known as Chronic Obstructive Pulmonary Disease (COPD) or 'heaves' (it is similar to asthma in humans)
- RAO is a common, performance limiting, allergic respiratory disease of horses characterized by chronic cough, nasal discharge and respiratory difficulty.
- Incidence increases with age (8 years or older)

Etiology

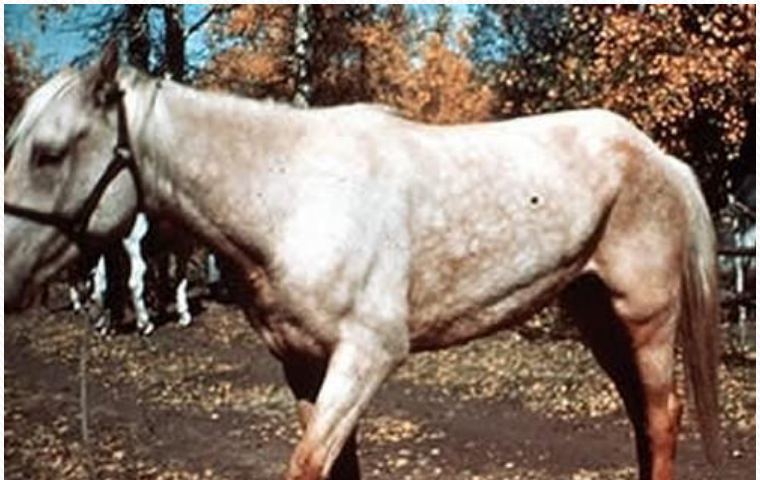
- Allergic bronchitis and bronchiolitis from exposure to various allergens including dust in straw and hay.
- Various molds associated beddings and feedstuffs

Clinical signs

–Flared nostrils, increased respiratory effort and dyspnea after strenuous exercise and a soft cough, particularly in association with feeding and exercise (exercise intolerance).

–In long standing cases, there will be a '**heave line**' along the ventral rib cage caused by the persistently increased respiratory effort

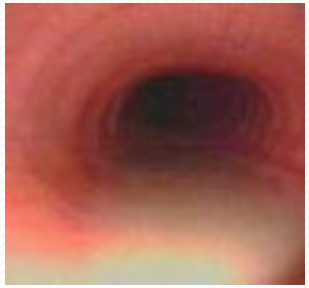
–Auscultation of the chest reveals **wheezing sounds** (most easily heard if rebreathing bag is used), prolonged expiratory phase of respiration and tracheal rattle and crackles



Diagnosis

–History and characteristic signs

– Endoscopy reveals increased amount of yellow viscous material with in the trachea and larger bronchi



–BAL fluid usually will reveal marked **neutrophilic inflammation**, up to 50-70% of the total cell count

Treatment

–The most important aim of therapy is to **prevent exposure to environmental allergens**

–The horse should be housed in a well ventilated barn with access to the outside

_Avoid airborne dust; pelleted feed or haylage should be substituted for hay and horses should be bedded on moist wood shavings or clay.

–**Corticosteroids** reduce the inflammatory response and resolve signs e.g., Dexamethasone 0.1mg/kg, IV, SID

–**Bronchodilators** provides relief of bronchoconstriction e.g., Clenbuterol 0.8-3.2mg/kg, BID

Infectious disorders of lower respiratory tract

1.Pneumonia

1.1 Bacterial pneumonia

1.2 interstitial pneumonia

1.3 Parasitic pneumonia

1.4 Aspiration pneumonia

2. Pleuro-pneumonia

3. Pulmonary congestion

4. African Horse Sickness

1. Pneumonia

Pneumonia is inflammation of the ***pulmonary parenchyma*** usually accompanied by inflammation of the ***bronchioles and often followed by pleuritis.***

1.1. Bacterial pneumonia

–The most common source of contamination of the lower airways is aspiration of microorganisms from the upper respiratory tact.

–Gram positive pathogens: *Streptococcus equi subsp. zooepidemicus, Staphylococcus aureus, Streptococcus pneumoniae*

–Gram negative pathogens: *Pasterella, Actinobacillus spp., Escherchia coli, Klebsiella pneumoniae, Bordetella bronchiseptica.*

–Anaerobic organisms: *Bacteroides fragilis, Fusobacterium spp.*

Pathogenesis

- Bronchopneumonia occurs after colonization of the lower respiratory tract with bacteria
- Usually follows damage from viral infections or some stressful events:
- long distance travel, strenuous exercise, aneasthesia, congregation of large numbers of equids, head tied up for long periods, poor air hygiene – dust inhalation, following smoke inhalation
- May lead to pleuropneumonia or pulmonary abscess

Clinical signs

- Depression, fever, anorexia
- Coughing during physical exertion or at rest with advanced disease
- Respiratory distress, weight loss and purulent nasal discharge with a fetid odour, thoracic pain and epistaxis

Diagnosis

- Clinical signs, physical examination
- Thoracic radiographs (extent, severity)
- Transtracheal aspirates (cytology, culture and sensitivity test)
- Rebreathing test (avoid if severe dyspnea occurs at rest)

Treatment

–Rest (resolution of infection may take 1-2 weeks and resolution of inflammation may take 2-4 weeks)

–Broad-spectrum antimicrobial therapy (until sensitivity test) Gentamicin (6.6 mg/kg IV, q24h and crystalline penicillin 20,000 IU/kg IV or IM, q6h followed by Procaine penicillin 15,000 -20,000 IU/kg IM, q12h or Timethoprim-sulphonamide (15mg/kg, PO, q12h and ceftiofur 2.2-4.4 mg/kg IV or IM, q12-24h

–**Bronchodilators** (aminophylline or clenbuterol HCl)

–**NSAID** (Flunixine meglumine) and supportive care

–If full response to treatment fails, pleuropneumonia develops (worsens the prognosis)

1.2. Interstitial pneumonia

–This is a relatively rare condition caused by infectious, toxic agents and immune mediated processes which includes Hendra virus infection, *Rhodococcus equi in foals, Aspergillus sp., Cryptococcus sp. and Hisptoplasma sp., Pnemoncystis carinii, Parascaris equorum, and Dictyocaulus arnfiedli, toxic plants and chemical agents (e.g., smoke, pesticides) and silicosis*

Clinical signs

–Chronic cough, fever, nasal discharge, tachypnea, tachycardia and severe respiratory distress, weight loss, exercise intolerance, cyanosis (end-stage)

Diagnosis

–Pulmonary auscultation: crackles and wheezes or absent sounds in severely affected cases

–Thoracic radiography (discrete or diffuse nodularity)

–Neutrophilic leukocytosis in cytologic evaluation

Treatment

–Generally, unresponsive to antimicrobial and NSAID

– Prognosis for survival is very poor in cases with cyanosis

1.3 Parasitic/Verminous pneumonia

- It is either a lungworm disease associated with the nematode parasite *Dictyocaulus arnfieldi or pneumonia due to Parascaaris equorum*

–All ages are susceptible

Clinical signs

–Exercise intolerance and poor body condition

–Coughing, respiratory distress

–Crackles and wheezes

–Mucoid to mucopurulent nasal discharge, fever, depression with secondary bacterial infection

Diagnosis

–Clinical signs, lack of evidence of bacterial infection

_ **Esinophilic pneumonitis** (Cytologic examination of a sterile tracheobronchial aspirate reveals abundant eosinophils (5%-50%, normal <2%) and **neutrophilic inflammation** may occur with secondary bacterial infection and rarely eggs/larvae)

–In some cases **fecal floatation** may reveal parasite ova (donkeys)

–Response to therapy also supports diagnosis

Treatment (anthelmintic +/- antibiotic therapy)

-ivermectin (0.5 mg/kg may be repeated after 15 days)

1.4 Aspiration pneumonia

–Aspiration or inhalation/ drenching pneumonia is a common and serious disease

_ Cases occur after careless drenching or passage of a stomach tube during treatment for other illness, for example administration of mineral oil to horses with colic

–Paralysis or obstruction of the larynx, pharynx, or esophagus may aspirate food or water when attempting to swallow

–There is no specific treatment. Treatment is supportive and includes anti-inflammatory drugs, antimicrobials, and oxygen. The prognosis for recovery is poor

2. Pleuropneumonia

- Pleuropneumonia (Infectious pleural effusion, pleuritis) is infection of the lungs and pleural space.

Etiology

–Viral respiratory infection, long-distance transportation, general anaesthesia, and strenuous exercise are common predisposing factors that impair pulmonary defense mechanisms allowing secondary bacterial invasion.

–In most instances, it develops secondary to bacterial pneumonia or penetrating thoracic wounds.

Pathogenesis

–Proliferation of bacteria in small airways, alveoli and lung parenchyma inflammation

–Spread of infection involves the visceral pleura and impair drainage of pleural fluid and increased permeability of pleural capillaries leading to accumulation of excessive pleural fluid, which then becomes infected.

–Fibrin deposition and necrosis of lung causes formation of **intra-thoracic abscesses**

–**Death is due to sepsis and respiratory failure**

Clinical signs

–Depression, in appetence, sweating and pleural pain

–**Pleural pain (pleurodynia)** evident as short strides, guarding, and flinching on percussion of the chest, stand with their elbows abducted, reluctance to move, cough, or lie down and have anxious facial expression

–Rapid and shallow respiration

Diagnosis

–Thoracic ultrasound (regions of poor or absent breath

- Thoracocentesis is performed for diagnostic and therapeutic purposes, and the ideal site (most ventral site) for drainage is determined via thoracic ultrasound

–Thoracic radiographs are obtained after drainage of the pleural cavity

sounds, thoracic pain and/or dull thoracic percussion)

Treatment

–Broad-spectrum antimicrobial therapy (combination of penicillin, gentamicin and metronizazole)

–Effective thoracic drainage

–Anti-inflammatory drugs and supportive care

–Treatment can be prolonged, expensive and complex

3. Pulmonary congestion

– is caused by an increase in the amount of blood in the lungs due to engorgement of the pulmonary vascular bed.

–It is sometimes followed by pulmonary edema when intravascular fluid escapes into the parenchyma and alveoli.

Etiology

- Primary pulmonary congestion

–Early stages of most cases of pneumonia, inhalation of smoke and fumes, anaphylactic reactions, hypostasis in recumbent animals, race horses with acute severe exercise-induced pulmonary hemorrhage

- Secondary pulmonary congestion

–Congestive heart failure (cardiogenic pulmonary edema), including ruptured chordae tendineae of the mitral valve, and left-sided heart failure

Clinical signs

–In acute pulmonary congestion there are harsh breath sounds but no crackles are present on auscultation.

–When pulmonary edema develops, loud breath sounds and crackles are audible over the ventral aspects of the lungs.

–In long-standing cases there may be emphysema with crackles and wheezes of the dorsal parts of the lungs, especially if the lesion is caused by anaphylaxis

–Coughing is usually present but the cough is soft, moist and is not painful

Diagnosis

–Diagnosis is always difficult unless there is **a history of a precipitating** cause such as an infectious disease, strenuous exercise, ingestion of toxicants, or inhalation of smoke or fumes.

Treatment (must first be directed to aetiologies)

–Furosemide, digoxin or arterial vasodilators

–NSAIDs or glucocorticoids

–correction of low plasma oncotic pressure (plasma, synthetic colloids)

–Epinephrine (for anaphylactic reactions)

4. **African horse sickness (AHS)**

- AHS is **non-contagious, infectious, insect-borne** of disease of equids caused by African horse sickness virus (AHSV).

•AHS is characterized by **pyrexia, edema of the lungs, pleura and subcutaneous tissues and haemorrhages in the serosal surfaces of internal organs**.

Etiology

–AHSV is the member of the genus Orbivirus in the family Reoviridae. It is non-enveloped, double stranded RNA virus. It has 9 antigenically distinct serotypes (AHSV-1, AHSV -2, AHSV -3, …. , AHSV-9)

Epidemiology

–Epidemiology of AHS depends on interaction between infected host, a competent vector and susceptible non-infected equidae.

–AHSV is biologically transmitted by midges; the principal vector being **Culicoides imicola**. Other Culicoides spp., and other hematophagous insects can transmit mechanically

–AHS has **seasonal occurrence**, its prevalence is influenced by climatic and other conditions that favor the breeding of the vectors.

–Epizootics of the disease occur in years in which there is longer drought period followed by heavy rains.

- AHS is endemic in eastern and central Africa and spreads regularly to southern Africa.

–A recent study in Ethiopia identified other serotypes: AHSV-2, AHSV-4, AHSV-6, AHSV-8 and AHSV-9 (Aklilu *et al., 2011).*

–AHS affects equine animals. Horses are most susceptible to the disease and mules are less susceptible. Most infections of donkeys and zebras are subclinical.

–Zebras are considered to be reservoir of the AHSV.

–Dogs may die from peracute infection after eating infected horse meat

Pathogenesis

–The primary sites of AHSV replication are thought to be lymph nodes, lungs and spleen and other lymphoid tissues and endothelial cells of small blood vessels.

–Viral replication in endothelial cells results in vascular damage and an increase in vascular permeability leading to hemorrhage and edema.

Clinical signs

–There are four forms of the disease:

–Pulmonary form or “Dunkop”, Cardiac/edematous form or “Dikkop”, mixed form and horsesickness fever.

Pulmonary form or "Dunkop"

–This form is the peracute form of AHS and occurs in fully susceptible horses (<5% recover) and dogs

–It is characterized by rapid rise in body temperature (40-410C), marked and rapidly progressive respiratory failure

–Forelegs spread apart, head extended, nostrils dilated, forced expiration (abdominal heave lines)

–Profuse sweating and paroxysmal coughing may be observed terminally, often with frothy, serofibrinous fluid exuding from the nostrils.

_ Dyspnea suddenly happens and death follows

Cardiac form

–This form is the **subacute form** of AHS and the incubation period is usually 7-14 days (mortality greater than 50%).

–Fever (39-410C) occurs and may persist for 3-4 days

–**Edema of the underlying tissue in the supraorbital fossae**

–Edema extends to the conjunctiva, lips, cheeks, tongue, the neck, shoulders and chest and sometimes colic

–Petechial hemorrhages on the conjunctivae and on the ventral surface of the tongue occurs

–Hydropericardium, endocarditis and pulmonary edema

Mixed form

–It is acute and most common form but rarely diagnosed clinically (often seen at necropsy) – mortality ~ 70%

–Its incubation period ranges from 5 to 7 days.

–Signs include combination of “dunkop” and “dikkop”

Horse sickness fever

–This is the mildest form of AHS

–Involving only mild to moderate fever (39–400C) of the remittent type

–There is no mortality from this form

Diagnosis

–Clinical signs combined with an appropriate history and epidemiological information may be sufficient for a presumptive diagnosis

–Laboratory diagnosis is essential for confirmation

–AHS virus may be isolated from blood, spleen, lung and lymph nodes (keep at +4oC during transportation and storage)

–ELISA, CFT, virus neutralization test and PCR are commonly used in diagnosis

Differential diagnosis

–equine encephalosis virus, equine viral arteritis (EVA), equine infectious anemia, Hendra virus infection, purpura hemorrhagica, trypanosomosis (surra) and the early stages of equine piroplasmosis (*Theileria equi and Babesia caballi) particularly when the parasites are difficult to demonstrate in blood smears*

Treatment

–There is no specific treatment for AHS (only supportive therapy, nursing, rest)

–Prevention in enzootic area involves annual vaccination

Control

The control measures in epizootic situations include:

–delineation of the area of infection

–Strict movement control within, into and out of the infected area

–Stabling of all equids at least from dusk to dawn (high vector activity period)

–Insect control measures

–Immediate vaccination of all susceptible equids with an attenuated polyvalent vaccine

–Notification to OIE

–The NVI of Ethiopia is producing a trivalent live attenuuated AHS vaccine against AHSV serotypes 2, 4 and 9

Thank you!!!

Diseases and disorders of Alimentary Tract

Hanna Zewdu (DVM, MSc)

Diseases of oral Cavity

1. **Cleft palate**

- Disorder that is commonly recognized in foals
- Congenital condition(etiology unknown)
- Result in ill thrift

Hx and PS

- Presented because of milk or water discharging from nares
- Failure to gain weight

1. Cleft palate…

CF

- Poor body condition
- May have secondary pneumonia due to inhalation of material
- When attempts to drink, milk often discharges from the nares

DX

- Lesions can be defined by oral examination or via endoscopic examination of the mouth and palate
- Usually Dx on the basis of clinical signs and visual inspection

DDX

Pneumonia from other causes

Dysphagia

choke

1. Cleft palate…

Treatment

Surgical repair for small cleft

2. Dentigerous cysts

- **Tumor** like structures of epithelial origin that frequently contain structures with tooth like appearance

HX and PS

- Usually in younger animals
- Presence of facial/mandibular distortion is commonly reported

CF and Dx

- Commonly found near the ear(temporal cysts)
- Other sites include maxillary sinus and elsewhere on the face
- **Discharge** from the cysts draining either into or near the ear may be seen
- In some cases, a draining tract may be present
- Radiographs usually reveal a **cystic cavity** with a high likelihood of dental like structures within the cavity

2. Dentigerous cysts

DDX

- Retained tooth caps with secondary alveolar periostitis
- Dental or paranasal sinus tumors
- Other causes of maxillary or facial deformity
- Local abscess
- Trauma
- Paranasal sinus cysts

Treatment

- **Resection** of the dentigerous cyst with particular care being taken to remove all the lining of the cystic cavity
- After removal, the **cavity is packed** and the dead space is allowed to heal by second intention

3. Gingivitis/stomatitis

- **primary stomatitis**: vesicular stomatitis, horse pox,candidiasis.pseudomonas and Rhodococus spp
- **secondary stomatitis**: periodontal disease, phenylbutazone toxicity,photosensetization, uremia and mercury toxicity

HX and PS

- In appetence or reluctance to eat
- Depression
- **Salivation**
- Hx of NSAIDS

CF and DX

- Generalized **swelling and redness** with ulceration noticed around the stomal and gingival tissues
- In severe cases, there can be **large ulceration** that result in **increased salivation and discomfort**

3. Gingivitis/stomatitis…..

- Progressive dehydration due to reluctance or inability to consume fluids

DDX

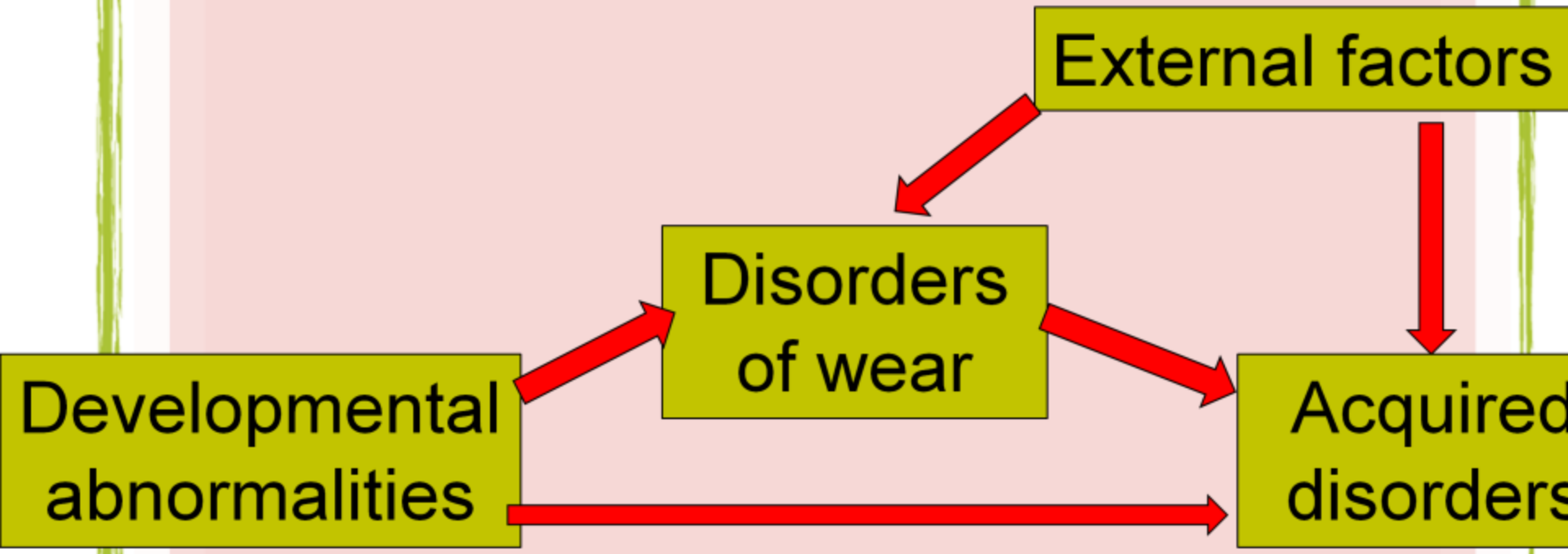
- Neoplasia
- Dental problems(retained tooth caps, malocclusion, enamel points, periodontal disease)

Treatment

- Reduce local inflammation
- Lavage: 0.05% chlorohexidine, povidone-iodine
- Discontinue incase of phynylebutasone toxicity
- Abs incase of infection(PPF)
- Fluid and feed via naso gastric intubation
- Treating the underlying cause in case of secondary once

4. Diseases of the teeth

Common Dental Pathology



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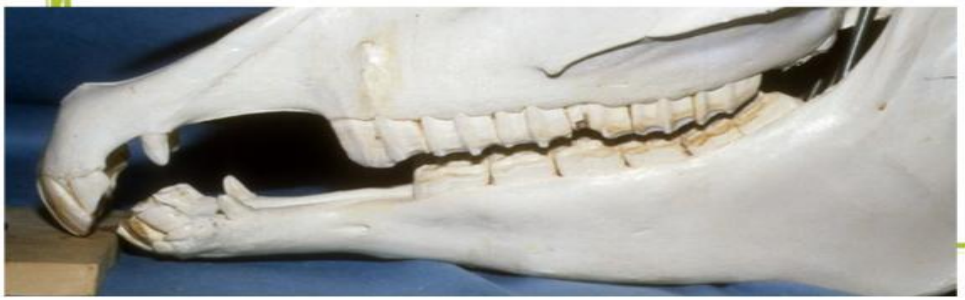
For the working animals of the world

Developmental abnormalities can be **birth defects** or caused by **disturbances during eruption**.

Brachygnathism (parrot mouth)

- Mandible/maxilla mismatching
- Often prehend well
- May need to reduce incisors
- Inherited trait – do not breed
- **Check cheek teeth**



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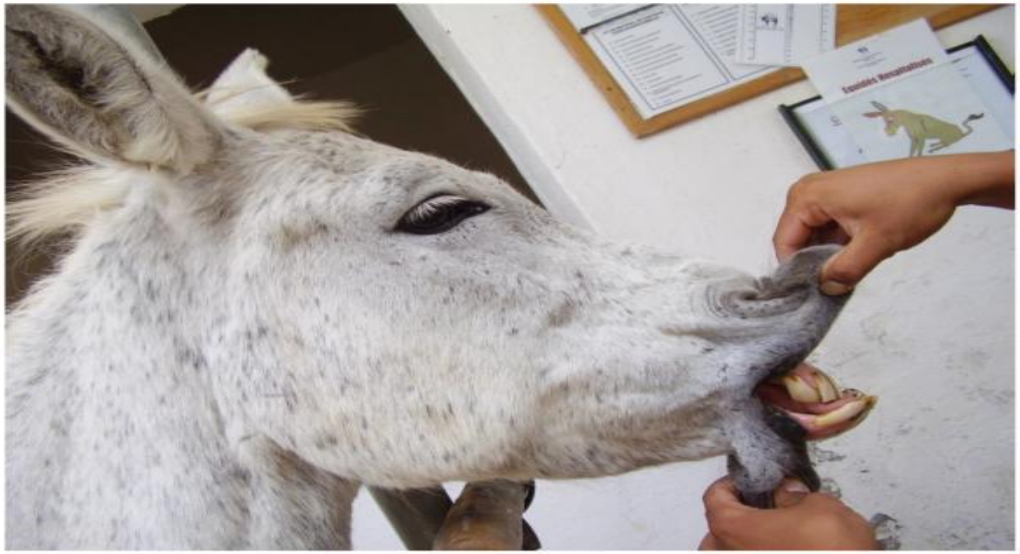
- Parrot mouth occurs when the mandible and maxilla are mismatched.
- Dramatic cases can be seen with no associated problems of prehension.
- Extreme cases may need incisor reductions if they are causing trauma to the palate.
- The important thing with these is to check the cheek teeth as they are likely to have far more serious problems.

Prognathism (sow mouth)



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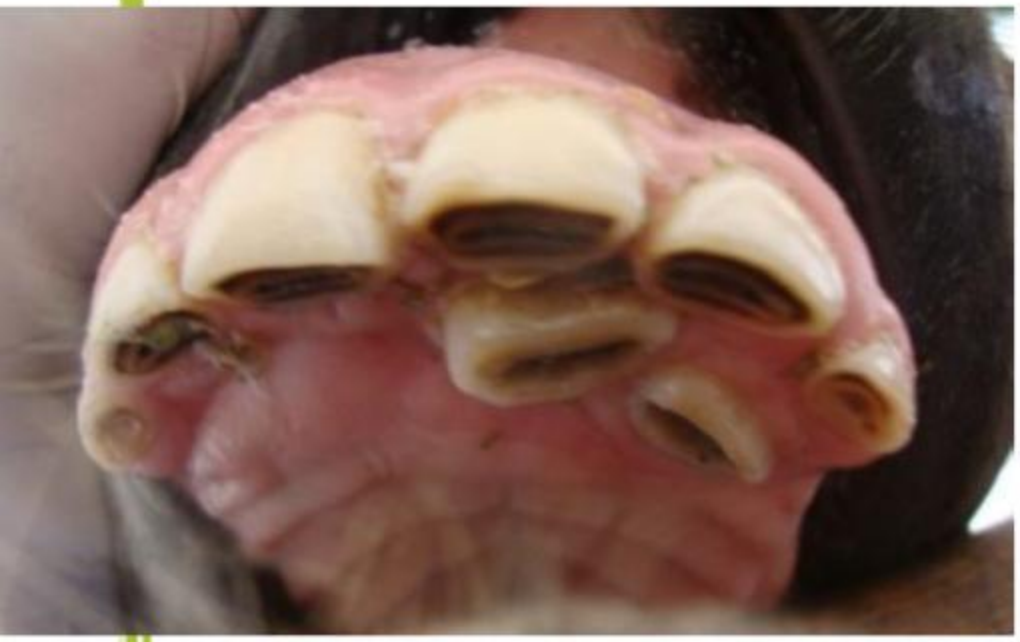
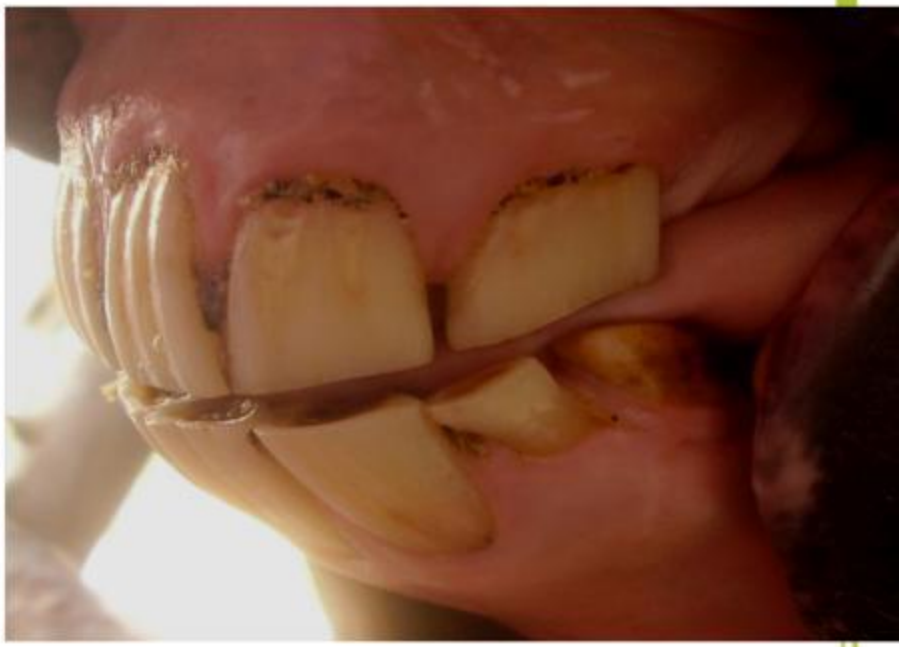
Sow mouth is **very rare** and again causes **few problems** other than the inevitable cheek teeth sequalae

Retained deciduous incisors





- Retained deciduous incisors may force permanent incisors caudally
- Do not confuse for supernumary teeth

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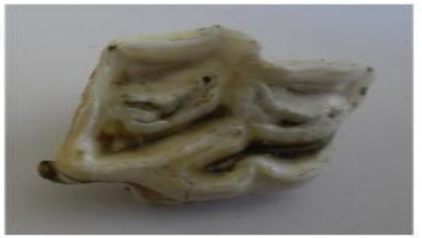
❖Retained deciduous incisors may force permanent incisors caudally, resulting in **wear disorders**, so should be removed.

❖However, it can be easy to mistake them for **supernumary** teeth, which are very difficult to remove but usually cause few problems and are best managed by reducing them.

❖If in doubt, leave it and re-examine later

Retained deciduous cheek teeth

- Premolar 'caps' normally lost at 2 ½, 3 and 4
- Loose caps can cause oral pain
- Do not remove if not loose
- Delayed eruption can
  → mandibular eruption cysts and
  → periapical infection



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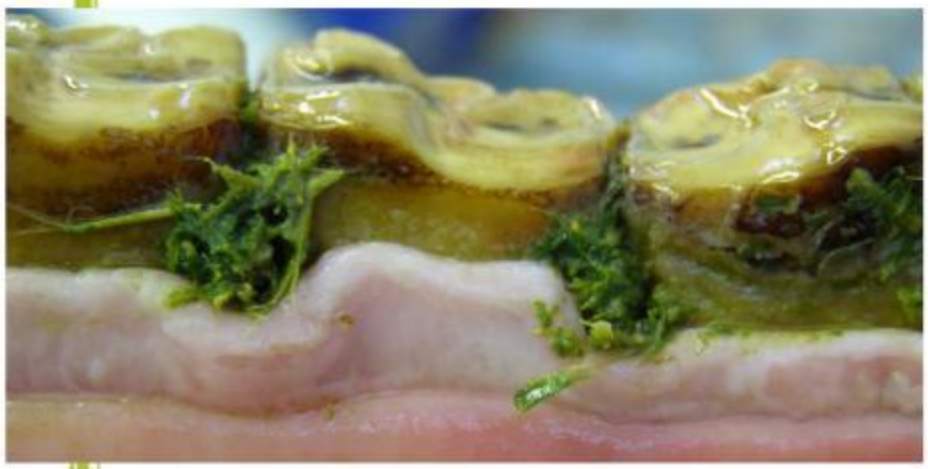
- The close contact between the premolars may slow eruption, causing **retention of caps** and **eruption cysts** – large bony swellings of the mandible caused by an exaggeration of normal thinning of the bone over the tooth apex.
- These should regress as the tooth erupts, but the thin bone predisposes to apical infections

Displaced teeth

- Tooth bud displacement
- Overcrowding
- → soft tissue trauma
- → periodental infections
- → oral pain
- → tooth loss
- →opposing overgrowth 'step'

- Tooth displacements can happen due to developmental tooth bud displacement, or overcrowding.
- Subsequent food entrapment will lead to periodental infections and the displaced tooth will cause soft tissue trauma.
- Oral pain and difficulty eating will often occur, as will tooth loss and subsequent opposing tooth overgrowth

Diastema



- Reduced angulation of cheek teeth
- Missing or displaced teeth
- Old age
- Incisors
- → periodental infection, pain, and tooth loss

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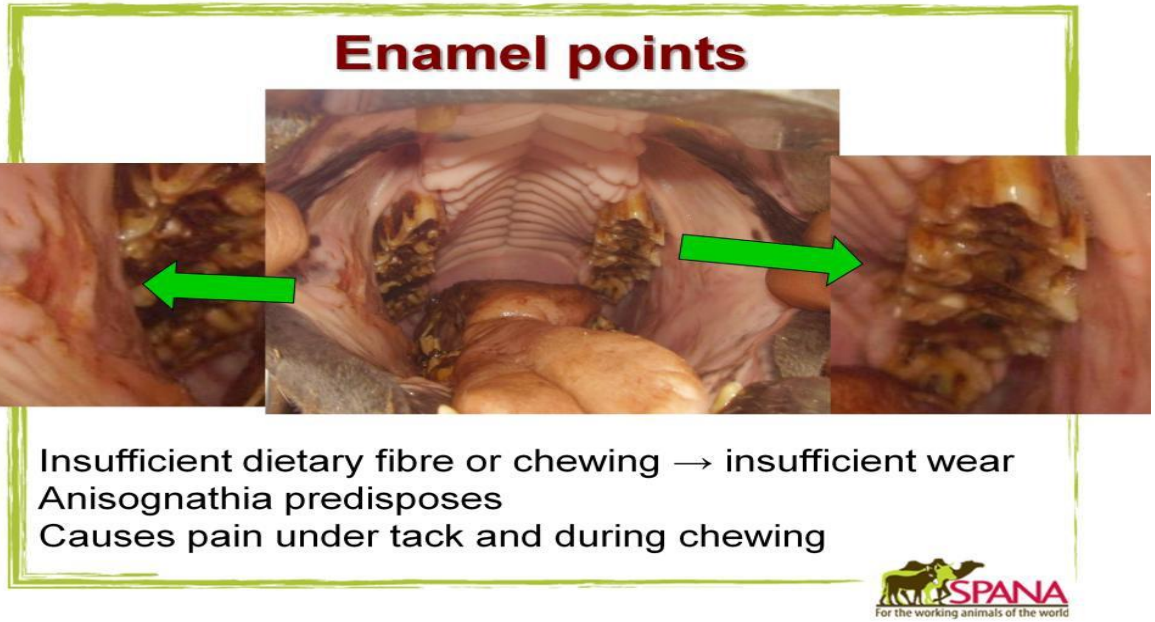
❖**Diastema** (gaps) can occur due to a reduced angulations of the front and back cheek teeth, or secondary to a missing or displaced tooth resulting in reduced compression of the arcade and drifting of teeth apart.

❖It is very common in old horses as the first and last cheek teeth get shorter and exert less pressure.
❖The triangular shape of incisors means diastema are inevitable if the tooth survives for long enough.
❖Food impaction will lead to secondary → periodental infection, pain, and tooth loss, and the lack of wear of the opposing tooth, causing focal overgrowths which then push more food into the space.
❖Techniques for management include removing entrapped food, treating infections, reducing opposing overgrowths and more radically widening the space so food does not get trapped.
❖A pump spray with a needle with the sharp end removed creates a narrow, high-pressure jet, useful for flushing out impacted food.

Disorders of wear

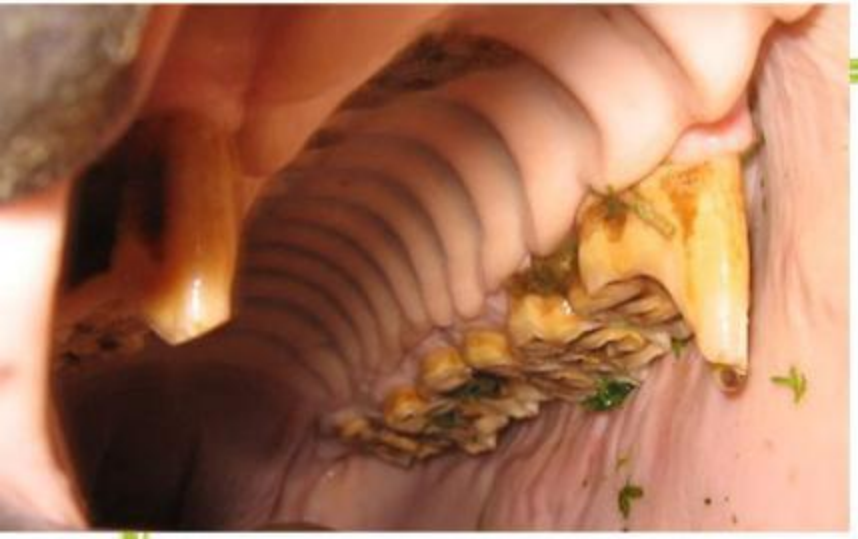


❖In the wild horses ,they may graze for 18 hours a day.

❖Domestication means this is much reduced, and this lack of grinding predisposes to **enamel overgrowths on the lingual aspect of the mandibular cheek teeth and the buccal aspect of the maxillary cheek teeth.**

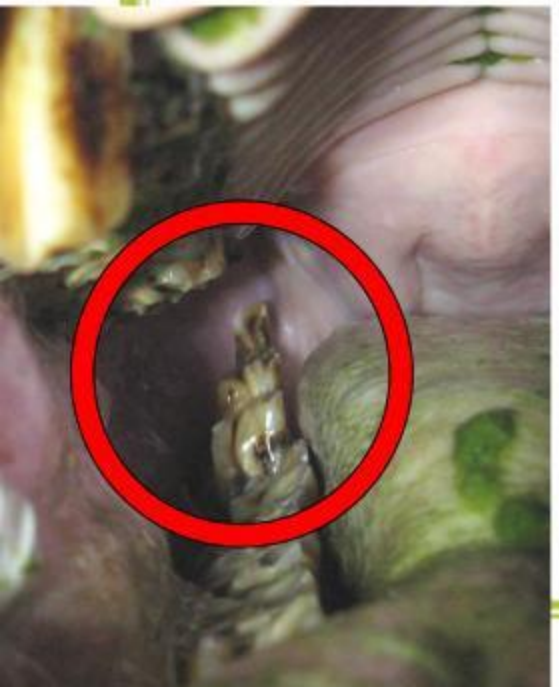
❖Sharp points cause soft tissue damage which causes pain under tack/path, with tight nosebands pulling cheeks on to sharp teeth and bits pushing the tongue against the inside of the lower cheek teeth.

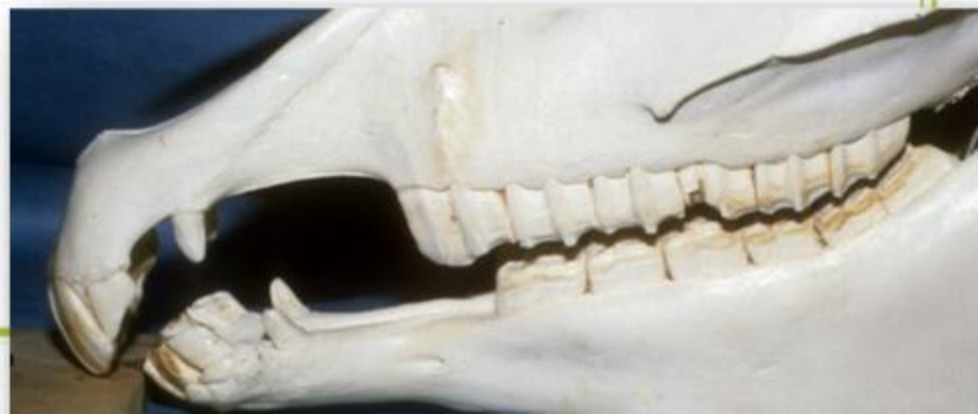
❖Masticatory pain may alter the chewing pattern, reducing the normal side to side pattern.



Hooks

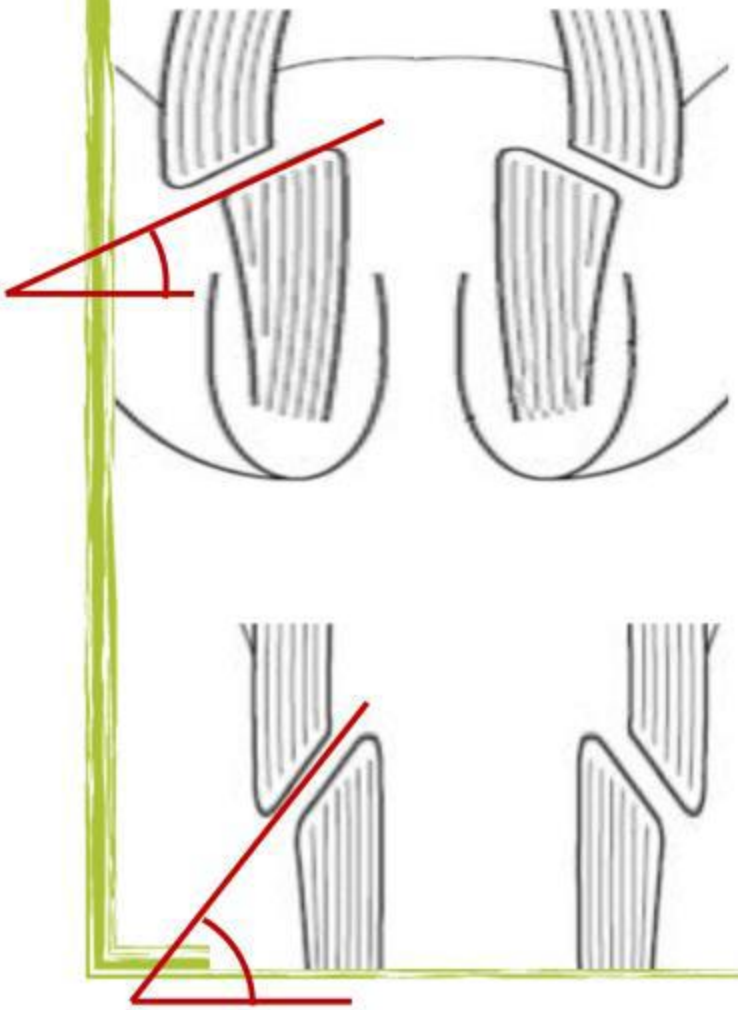
- rostral positioning maxillary CT
- opposite way rare
- unequal arcade lengths
- pain eating
- restriction of rostro-caudal movement
- reduce gradually

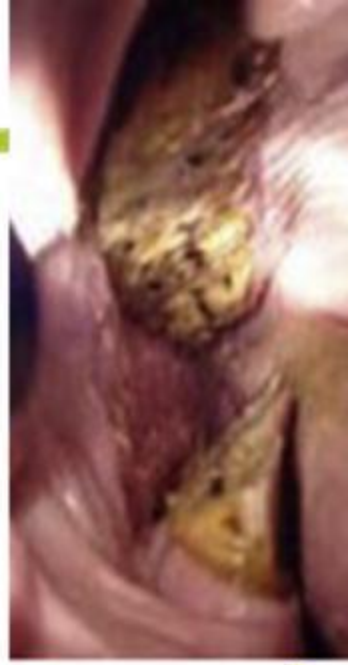




- Hooks form due to **rostral positioning** of the maxillary cheek teeth arcade with respect to the mandibular cheek teeth arcade, or from unequal arcade lengths.
- They may cause pain when eating and will restrict the rostro-caudal movement of the TM joint during chewing.
- Gradual reduction in stages every 2-3 months is necessary to avoid iatrogenic pulp penetration and resulting infection.
- Motorised equipment makes reducing large overgrowths much easier.

Shear mouth





- untreated enamel overgrowths
- restricted side to side movement

→ up and down chewing action

→ steepening of the occlusal angle > 30°

→ further reduction of side to side movement

- rasp back to normal angle gradually

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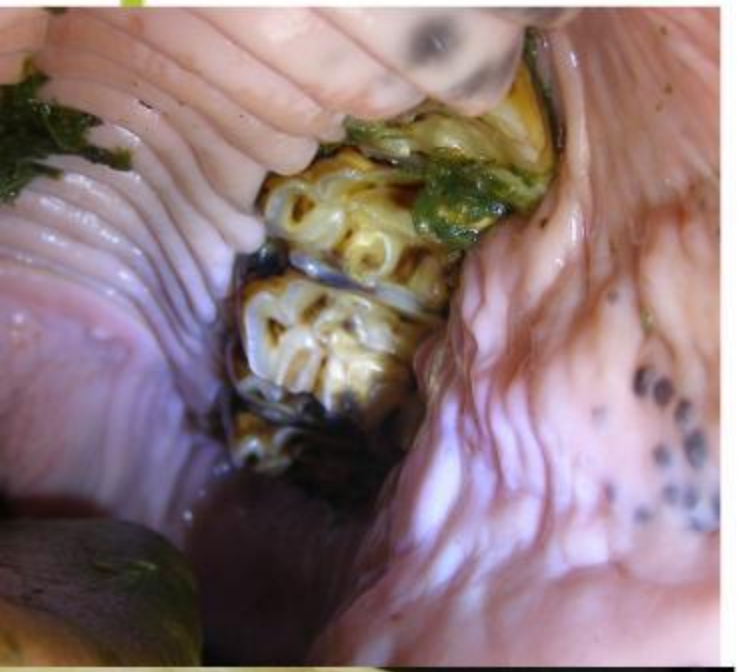
- Shear mouth is due to the upper arcade being markedly wider than the lower arcade
- Normally the occulusal surfaces of the cheek teeth sit a slight angle of 10-30 degrees.
- If the angle becomes greater than 30 degrees this is termed as sheer mouth
- Untreated enamel overgrowths → restricted side to side movement → up and down chewing action → steepening of the occlusal angle → further reduction of side to side movement.
- Manage by rasping back to normal angle in gradual stages

Wave mouth



- Uneven eruption & wear → wave mouth.
- Common in aged horses
- Impossible to correct
- Reduce worst overgrowths 2 x year

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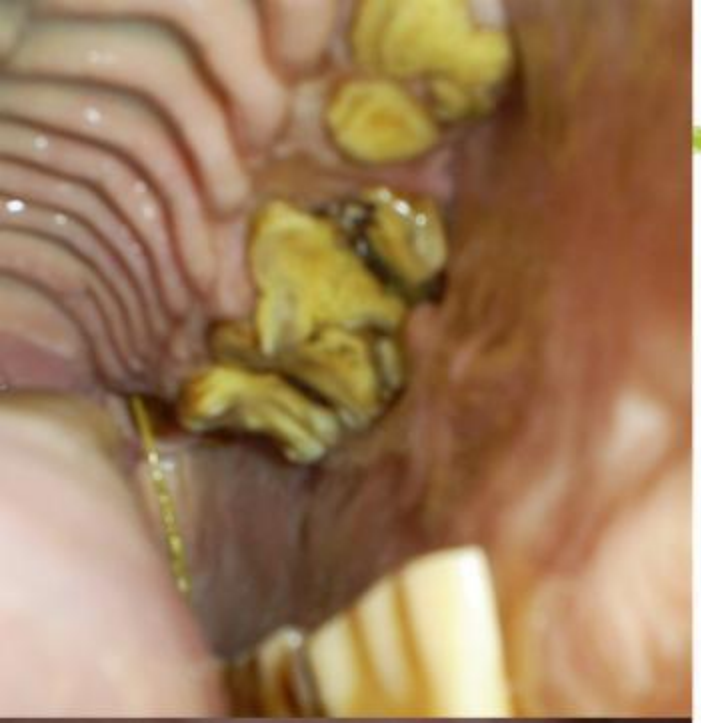
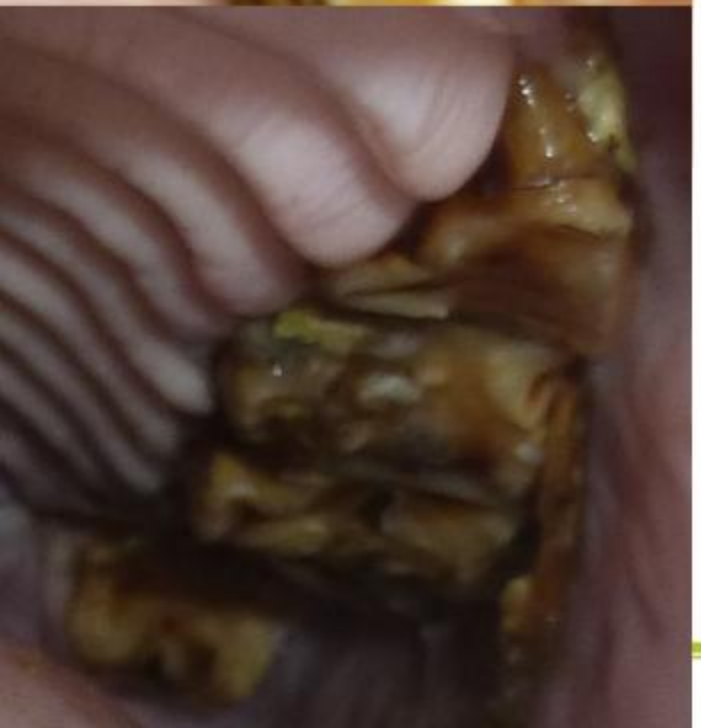
- This is when there is a wave like or undulating appearance to the arcade in a rostro-caudal direction
- Seen in older horses and donkeys
- Any tooth disorder causing uneven eruption & wear e.g. periodental disease and shear mouth, can cause wave mouth.
- It can also progress from steps and focal overgrowths.
- It is common in aged horses accompanying smooth mouth.
- It is impossible to correct but should be managed by reducing the worst overgrowths twice yearly.





Smooth mouth

- No remaining enamel
- Tooth smooth and glassy
- No grinding ability
- Old horses
- Excessive wear – abrasive diet

- Avoid overzealous rasping
- Maintain what is left functioning

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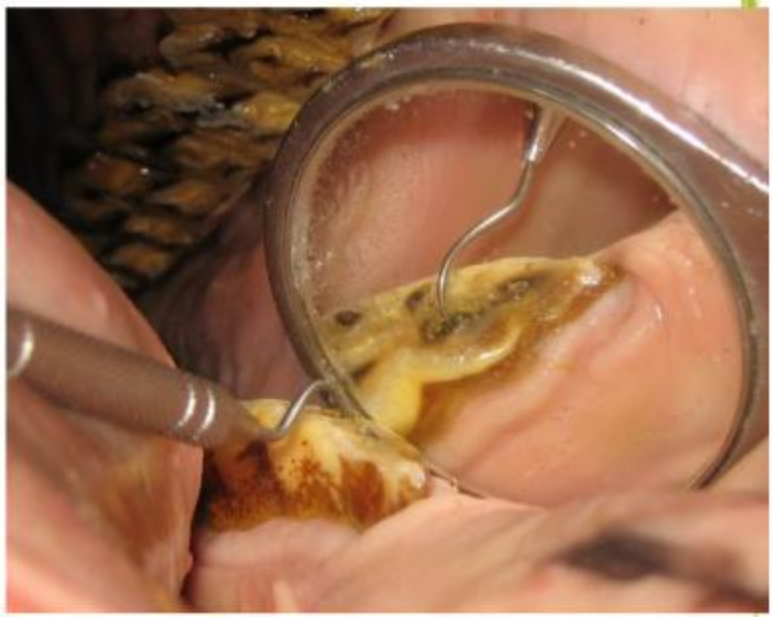
- Smooth mouth occurs when all the tooth enamel has been worn away leaving first only dentine and cement, and then the roots visible.
- The teeth feel glassy and have no useful grinding capacity left.
- To reach this stage before very old age, teeth have been subject to excessive wear from abrasive food or overzealous rasping of occlusal surfaces.
- In very old horses, we should be careful of rasping sharp edges (unless causing evident trauma), as these may be the only useful teeth the horse has left.

Acquired disorders

Periapical infection



Aetiology unclear
Possibly advanced caries or pulp exposure

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❖It is not known exactly why periapical infections occur.

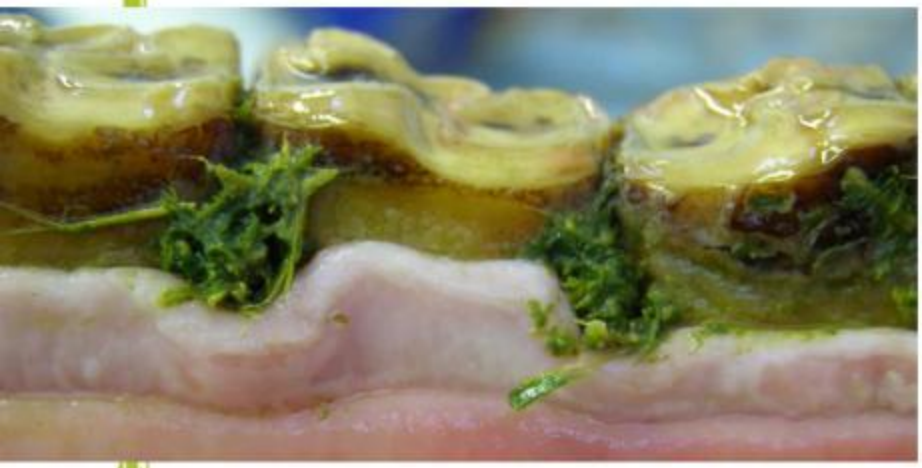
❖ It may extend from **infundibular caries or pulp exposure** on the tooth surface, which happens when the rate of attrition exceeds the rate in which secondary dentine is laid down to seal the pulp cavity.

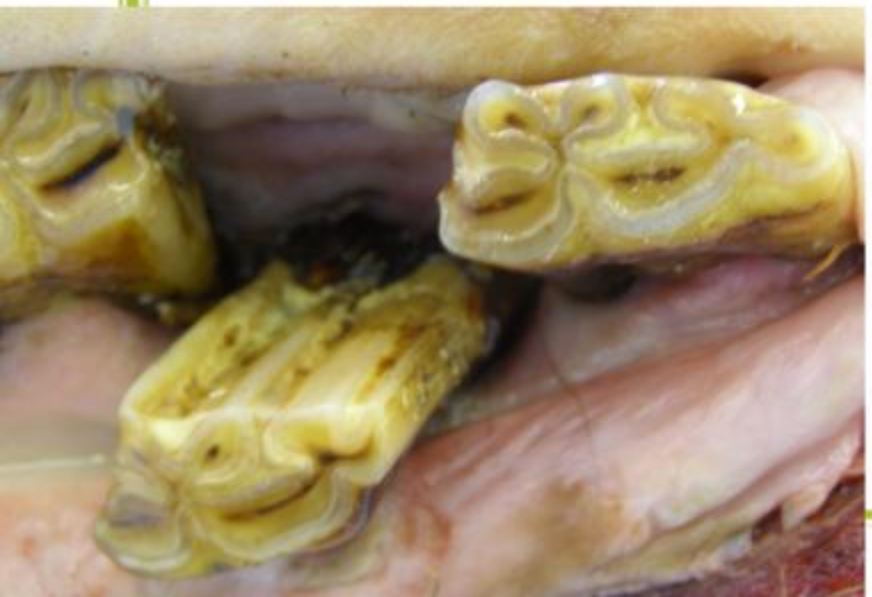
❖It may occur in **young horses** due to thinning of the mandible round dental eruption cycts.

❖Pulp infection can lead to pathological fracture, loss of the tooth and infection of the alveolar bone.

❖Early infections may be treatable with prolonged antibiotics e.g. TMPS for 1-2 weeks, but advanced cases may need tooth removal – a difficult procedure

Periodental disease





Disruption of normal arcade

Food impactions

Periodental ligament destruction → pain, halitosis, loosening of tooth and tooth loss

Remove loose teeth
Antibiotics

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- Is the inflammation and infection of the gums(alveoli, gingiva, periodental ligamnts and cement)
- Periodental disease is a common sequel to anything that disrupts the continuity of the cheek teeth arcade, e.g. diastema or displaced teeth, resulting in food impaction.
- Periodental ligament destruction causes loose teeth and pain chewing.
- Infection drains via the mouth and may cause halitosis.
- Painful, digitally loose teeth should be removed, which is usually quite easy, after sedation, local anaesthesia and elevation.
- If only slightly loose, remove impacted food and treat the infection with prolonged antibiotics; and reduce any opposing teeth out of occlusion that may forcing food into spaces.
- Taking the loose tooth out of occlusion and treating the infection may give it a change to tighten up.
- If the tooth can be preserved it is better than removing it, as the missing tooth will result in an opposing overgrowth, ‘step mouth’, which will require reducing every 6 months for the remainder of the horse’s life.

Tooth loss & step mouth

Tooth loss causes opposing tooth overgrowths
= ‘step mouth’
Treat as for hooks by gradual reduction

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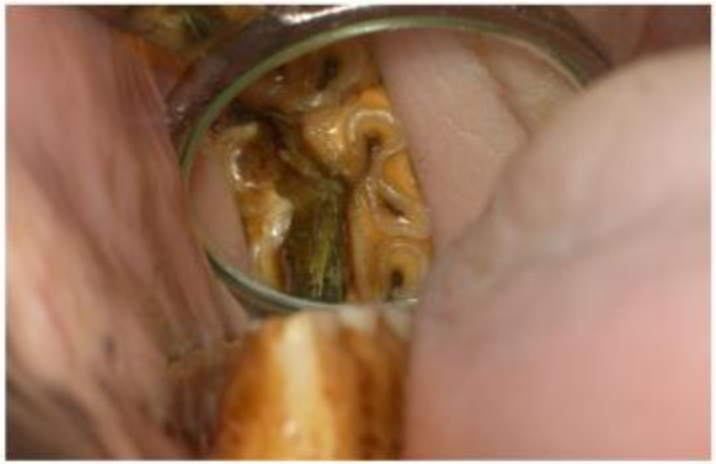
Fractured teeth



- Often found incidentally
- → 2° pulpitis
- Fragments may cause soft tissue trauma
- Remove loose fragments or infected material

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Treatment options

Rasping

Mechanical reduction

Tooth or fragment removal

Medical treatment

Disease of esophagus

Esophageal obstruction(Choke)

- Oesophageal obstruction

–Proximal third of oesophagus

–Thoracic inlet

- Commonly due to rapid ingestion of food
  - –poor dentition
  - –Inappropriate feedstuff, eg mango stone
- Less commonly due to
  - –Intra-luminal/intra-mural mass
  - –Megaoesophagus
  - –Oesophageal stricture/diverticulum
  - –Extra-oesophageal swelling (cervical abscess; lymphadenopathy
  - –Foreign body

Clinical signs

- Similar, irrespective of cause
- Inappetant, coughing, gulping, arching or stretching of the neck
- Salivation and nasal return of saliva, water and food material
- Also occurs with dysphagia
- Left sided cervical swelling may be palpable

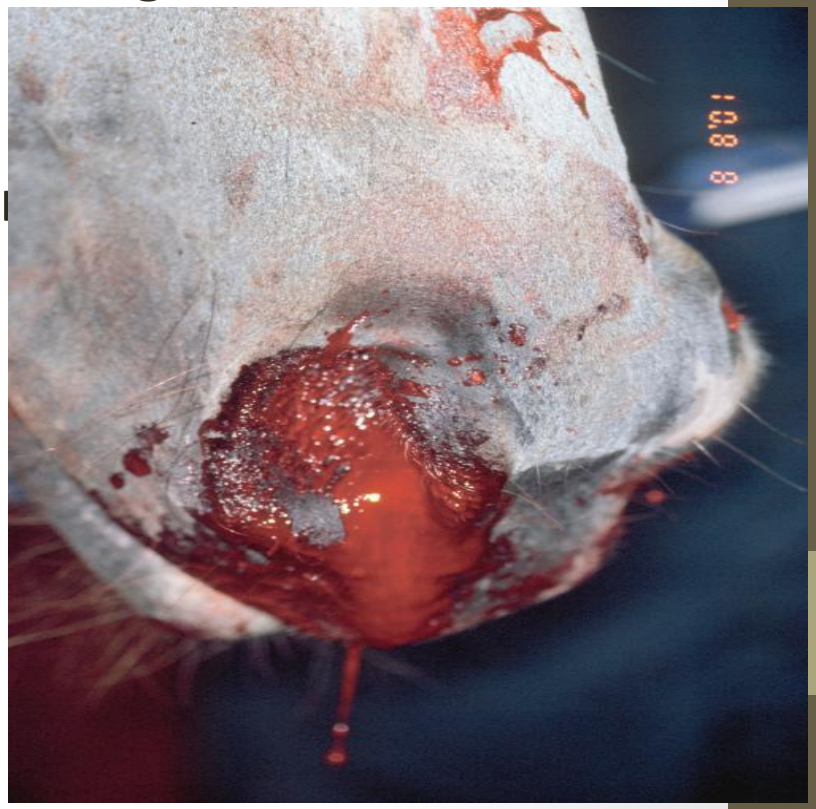
❖History and clinical signs
❖Inability to pass naso-gastric tube all the way to the stomach
❖ Many obstructions resolve spontaneously
❖ Passage of large bore oesophageal tube can provide rapid and effective resolution of choke but must be undertaken with care
❖ Repeated lavage using nasogastric tube
❖ Do not force the tube down the oesophagus
❖ Risk of rupture of oesophagus (fatal)
❖ Use of cuffed endotracheal tube

Medical management of choke

1. Xylazine: 0.5-1 mg/kg- for muscle relaxation, sedation to facilitate naso-esophageal intubation, lowers head to avoid risk of inhalation
2. others: Acepromizine(0.02-0.4 mg/kg,hyocine(0.2 mg/kg)

Complications of choke

1. Epistaxis following intubation
2. Pharyngeal inflammation
3. Risk of aspiration pneumonia if prolonged choke
–Antibiotics
4. Ulceration and stricture formation
5. Oseophageal rupture



Diseases of the stomach

Gastric impaction, Dilation and rupture

- Impaction of stomach is uncommon
- Most commonly results from poor dentition or consumption of low quality food stuffs(e.g straw)
- Dilation occurs as a primary event as a result of food(grain),water or air
- Most common cause of secondary dilation is bowel obstruction(SI and LI)
- Because horses are unable to vomit, rupture is a frequent sequel to dilation

HX and PS

- Evidence of dental abnormalities
- Rapid eating
- Consumption of poor quality feed or large volumes of feed or water ,especially grain
- Colic

CF and Dx

- Abdominal pain(dog sit-to decrease pressure on the stomach
- Increased cardinal signs, dehydration and shock in severe cases
- Significant gastric reflux(5-15L)
- Distended loops of small and large bowel in cases of secondary dilation
- Gastric rupture accompanied by reduction in pain
- Abdominocentesis reveal evidence of ingesta

Treatment

- Gastric decompression
- Gastric lavage may assist in removal of grain and ingesta
- Fluids –dehydration
- Pain control (xylazine,detomidine,romifidine, flunixine meglumine etc

Pyloric stenosis

- Rarely occurs secondary to gastroduodenal ulcer disease or because of fibrous masses in the pylorus

HX and PS

- Most common in horses less than one year
- Previous Hx of ulcer disease
- Signs of gastric dilatation
- Weight loss,colic and possibly diarrhea

CF and DX

- Signs of colic usually after eating
- Bruxism(grinding of teeth) and increased salivation
- Positive gastric reflux may be found
- Endoscopy,radiography

Treatment

Supportive(fluids and electrolytes, pain control and anti ulcer

Gastric parasitism

- Infection with Gastrophilus SPP.(Bots) is the most common cause of gastritis
- Infection with Habronema SPP., Draschia Megastoma and Trichostrongylus axei also occurs
- Although infection with any or all of these parasites is common ,clinical signs are rare

Hx and Ps

Clinical effects are rare, although ill trift,colic,and gastric dilataion/rupture have been ascribed to these parasites, particularly bots

Bots eggs seen on horses legs or bot maggots

- Generally, no signs are shown
- In some cases, non specific signs like poor body condition or signs of recurrent low grade colic may be noticed

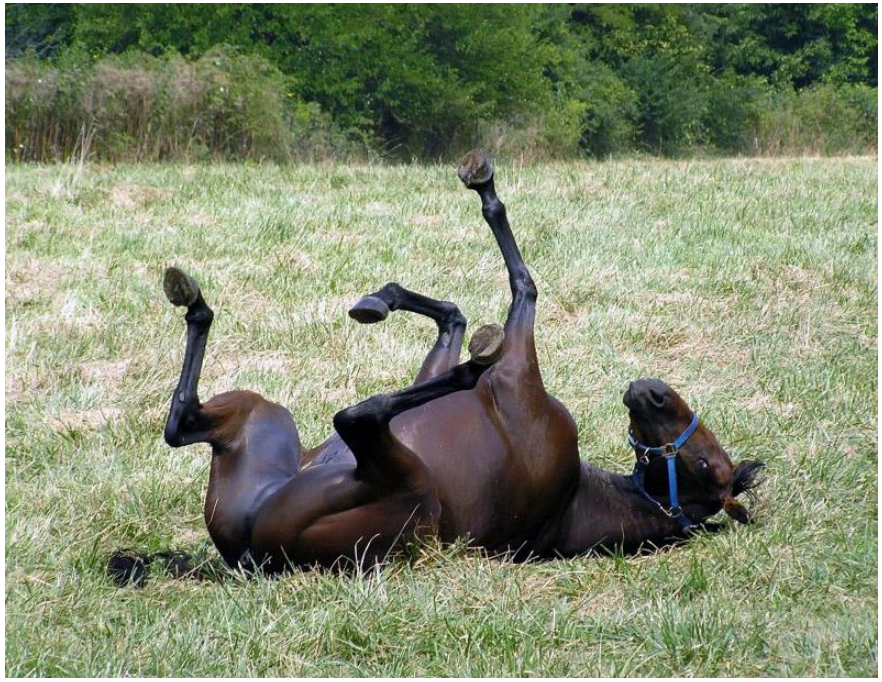
RX

- Ivermectin (0.2 mg/kg orally)

Diseases of small Intestine and large intestine

(Colic and diarrhea)

Colic



Colic

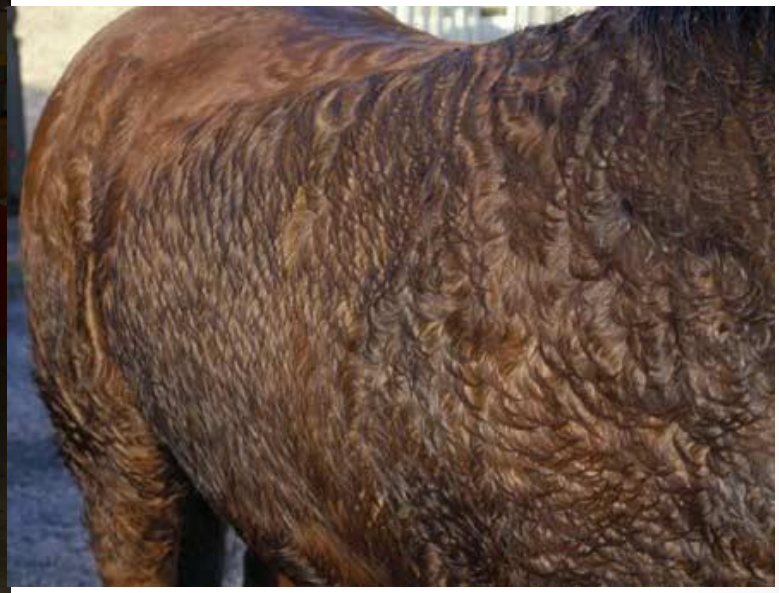
What is colic?

- Colic can be defined as abdominal pain.
- It is a clinical sign, not a disease.
- Severity varies from mild to fatal.
- Many different causes
- Most horses with colic recover spontaneously or respond to simple medical management.

Signs of Colic

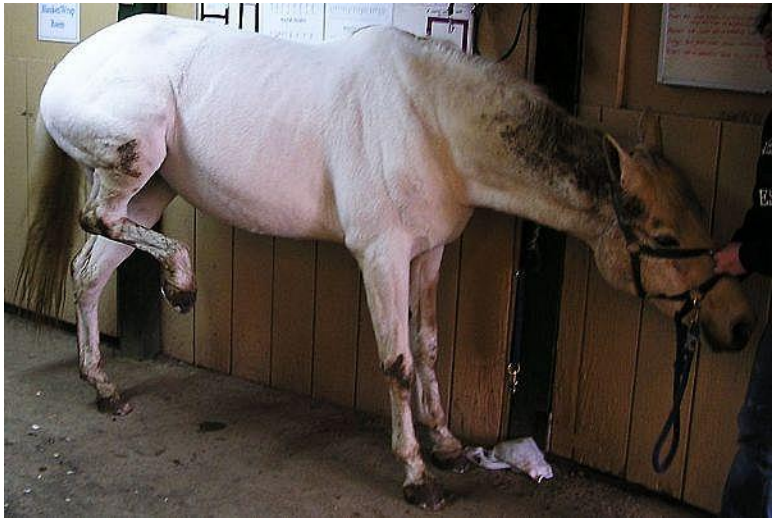
Pawing the ground

Sweating



Signs of Colic

Kicking abdomen



Stretching and posturing to urinate sometimes attempting to urinate

Signs of Colic

Flank watching

Rolling

Most Common Types/Causes

- Spasmodic colic
- Impaction
- Tympany
- Displacements
- Torsion, volvulus, intussucception
- Obstruction (strangulating/non strangulating)
- Colitis
- Foreign body ingestion
- Parasitism
- Dental problems

‘False’ Colic

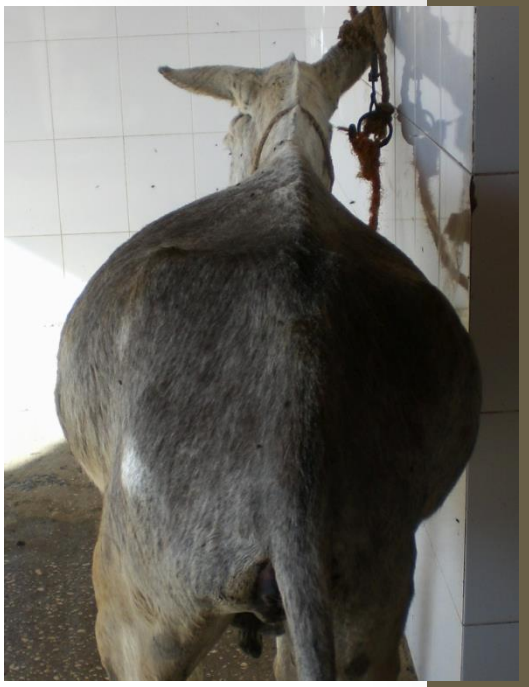
Non-gastrointestinal tract (GIT) associated

- Reproductive tract: Uterine torsion, pneumovagina, post foaling uterus involution or injury
- Urinary tract pathology e.g. cystitis or bladder calculi (RARE!!)
- Azoturia)
- Aorto-iliac thrombosis) post exercise
- Laminitis (lying flat out)

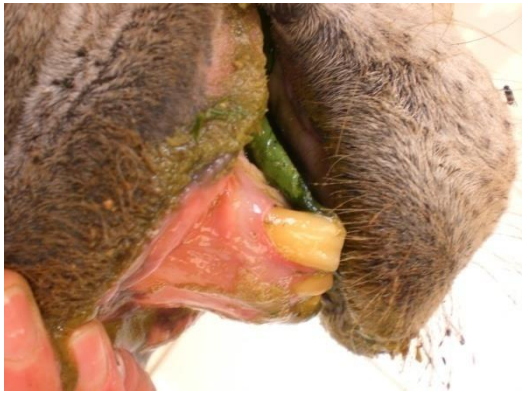
Case History: questions for the owner

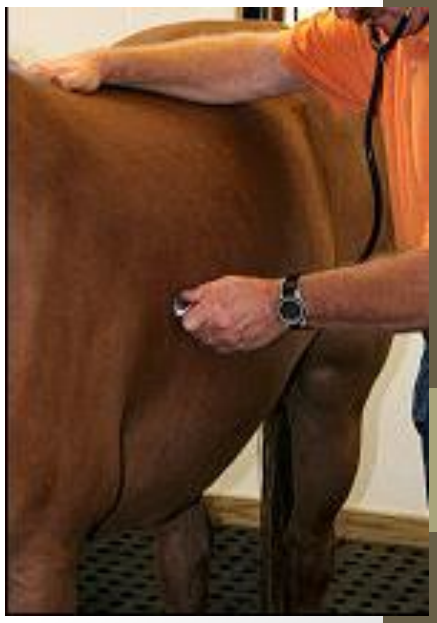
- Diet and changes, when last ate
- Habitat
- Routine
- Access to drinking water, when last drank
- Behaviour problems
- Previous medical history (and of other horses similarly managed)
- Parasite control
- (Pregnancy/breeding history)
- Onset and severity of pain, progression
- Faeces – when last passed, normal/dry/loose
- Urination – they often think this is the issue!
- Any treatment / medication given and response

Clinical examination



- Attitude
- Pain
- Abdominal distension
- Skin tent
- TPR
- Mucous membrane colour, moisture and capillary refill time (CRT)
- Auscultation and percussion
- Check scrotum in stallions (if safe)





Further clinical tests

- Rectal examination
- Naso-gastric intubation
- (Abdominocentesis)
- (Blood tests)

Rectal examination

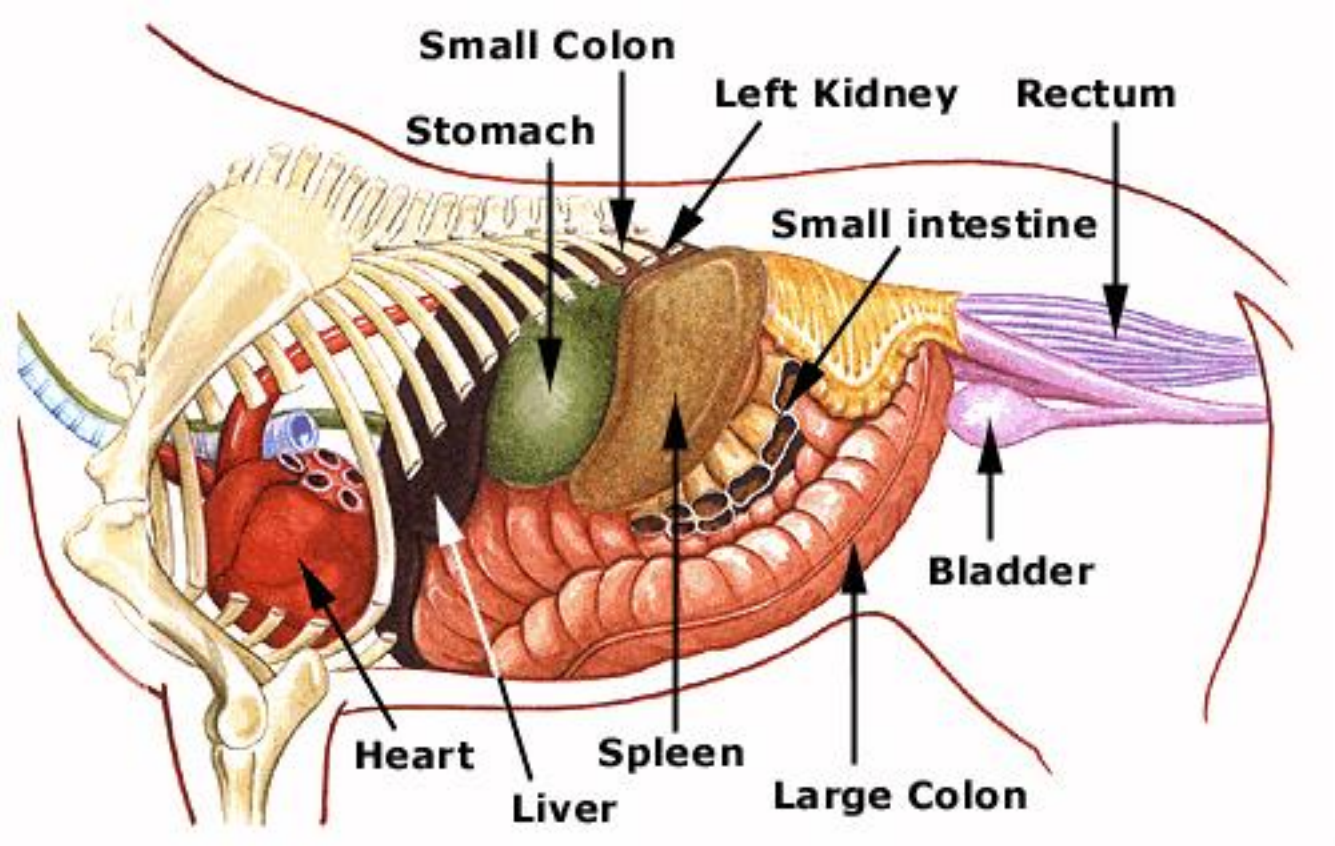
Ensure horse restrained

Wear full length gloves and use plenty of lubrication.

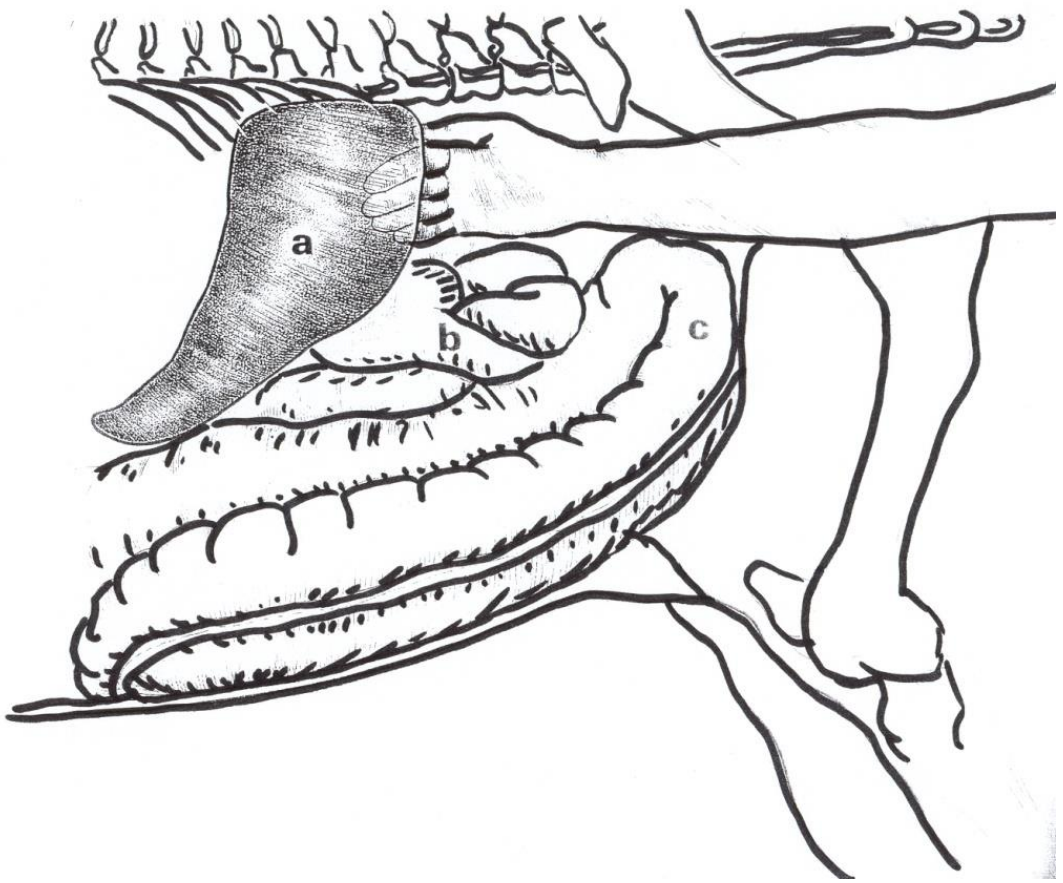
Risk of rectal tear.

- Faecal consistency, parasites, sand test
- Impacted, distended, or displaced large intestine and tight taenial bands. Thickened bowel

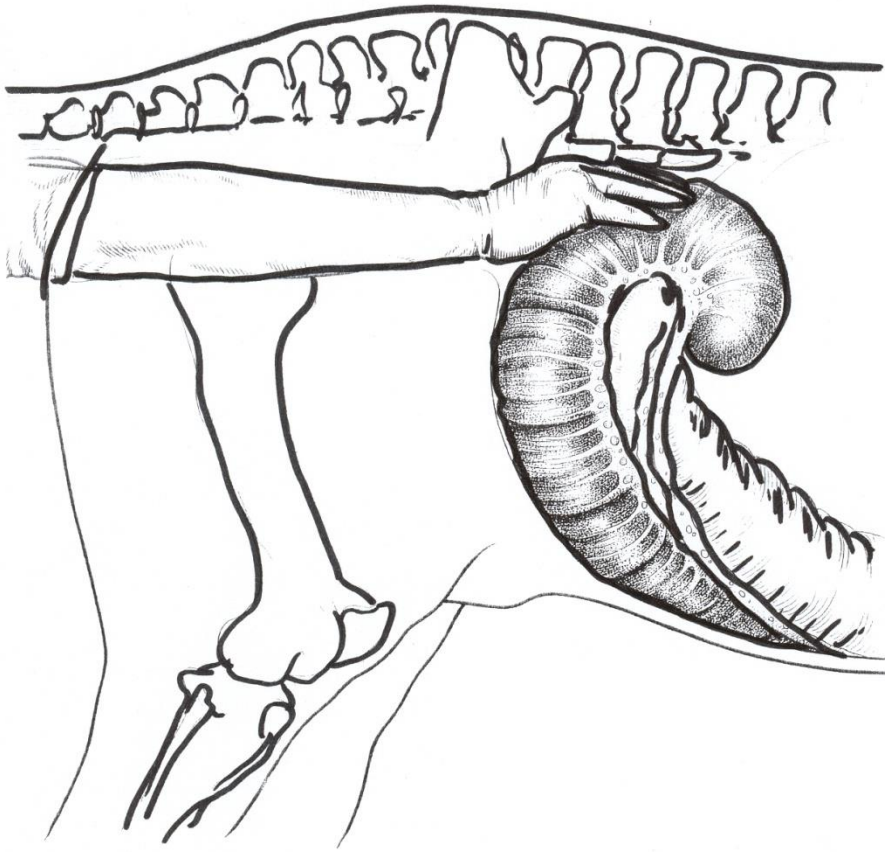
Internal Anatomy



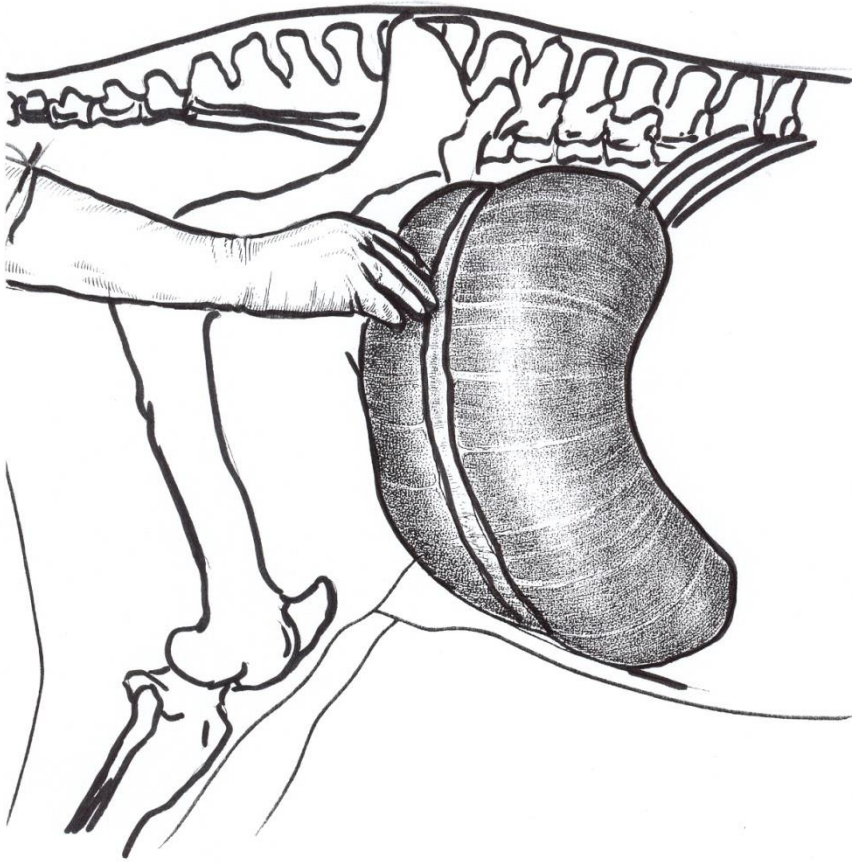
Palpate the inner edge of the spleen and nephrosplenic space



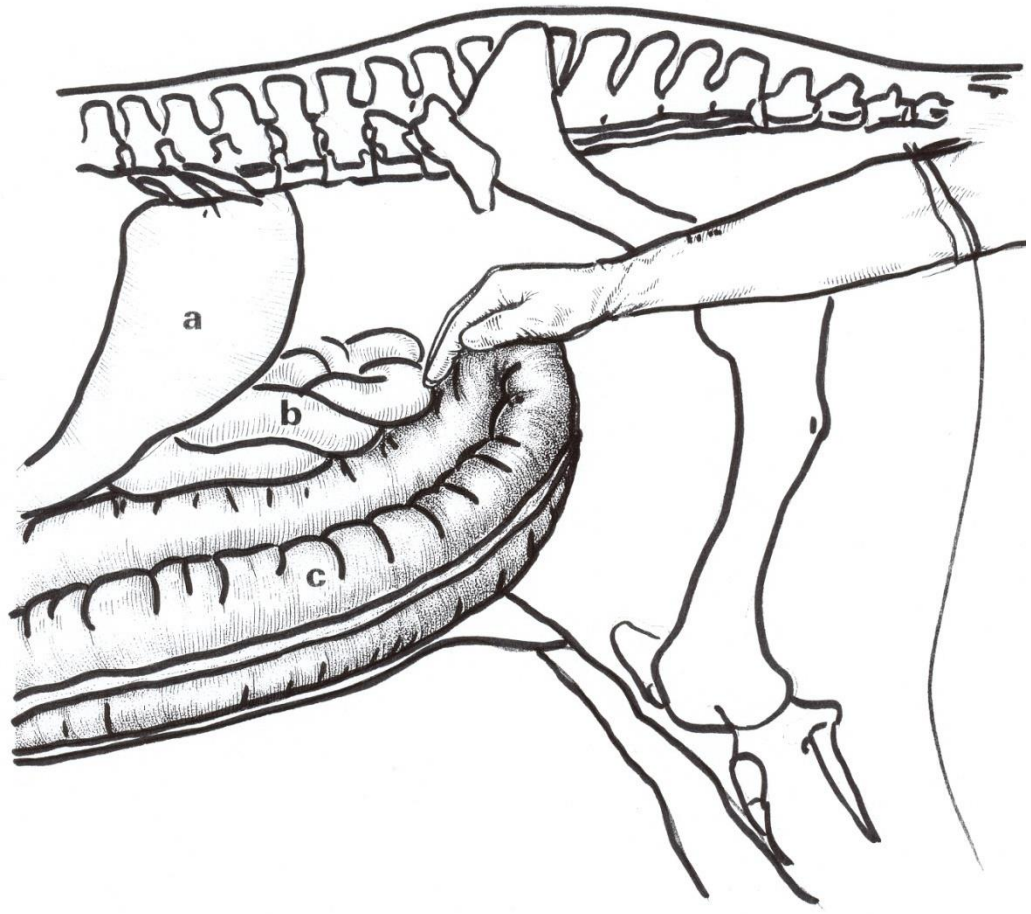
Palpating the caecum



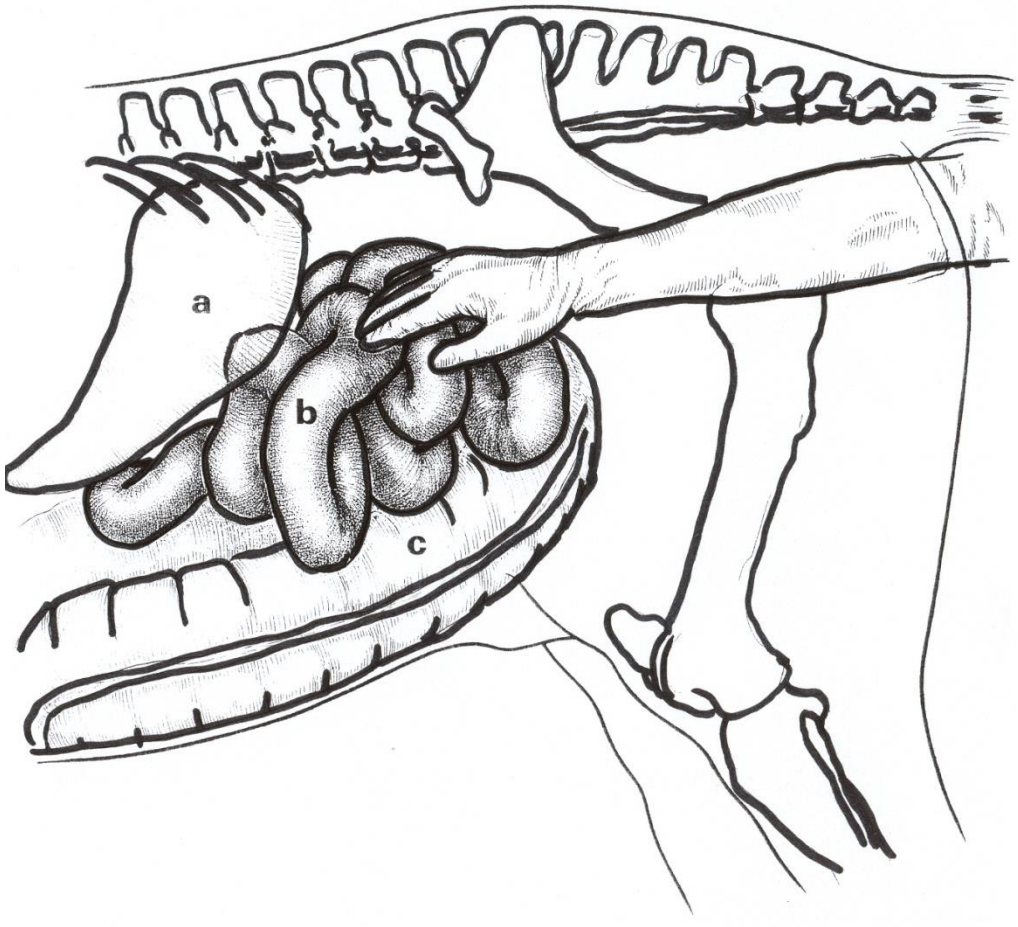
Caecal tympany



Palpating the pelvic flexure



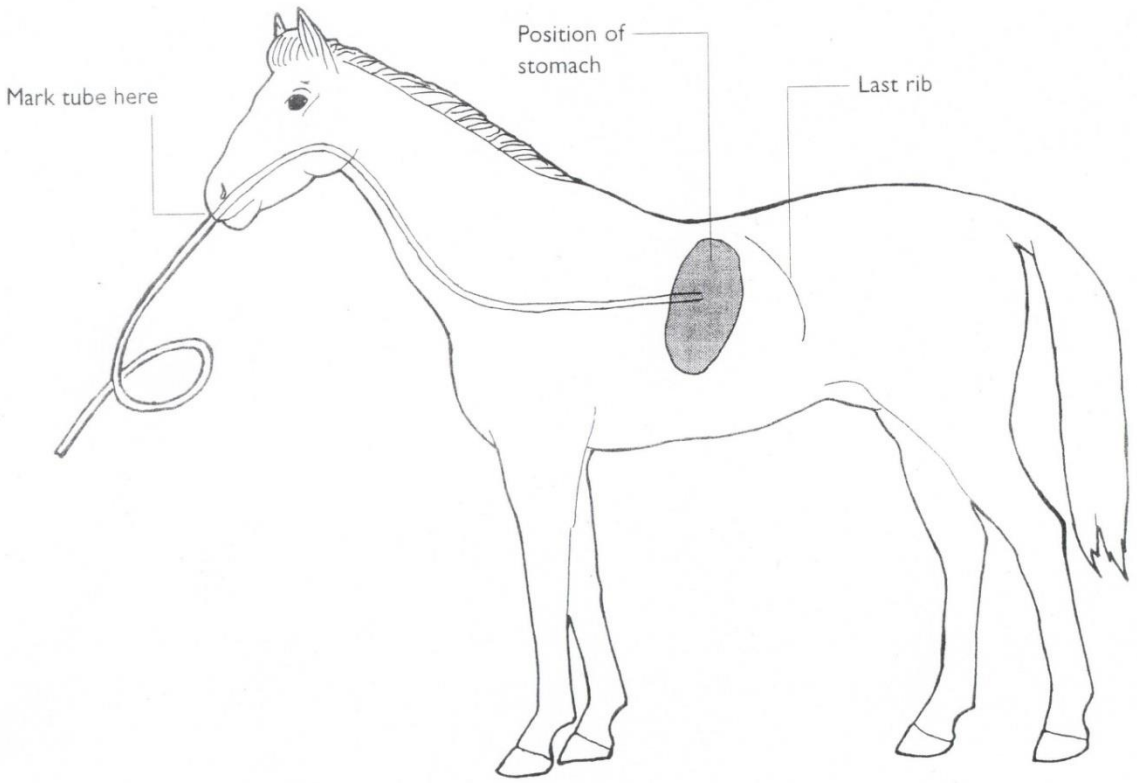
Distended small intestine



Rectal examination

- Inguinal rings
- Small colon
- Bladder

Naso-gastric Intubation



Naso-gastric Intubation - Tips

- Risk of epistaxis – warn owner!
- If in dorso-pharyngeal recess (feels like hard dead end), withdraw tube a few cms, rotate through 90 degrees and re-insert, should then be at larynx (soft dead end)
- If other nostril blocked by holding nasal bones, will make horse throw his head up, and also open his arytenoids further, making tracheal entry more likely.

Passing a nasogastric tube

Decompressing or ‘refluxing’ a horse

Medical Management

- Control of pain
  - NSAIDS
  - Sedatives
  - Antispasmodics
- Fluid therapy – IV and oral. Frequent oral water more effective way of dissolving impactions than over-hydrating with IV fluids.
- Exercise

- Frusemide ‘Lasix’ NOT recommended.

Control of Pain

NSAIDS

- Flunixin meglumine 'finadyne' –1.1mg/kg iv q12 – 24h for analgesia, 0.25 mg/kg iv, im q8h for endotoxaemia. Lasts 8 hours. **Strong pain relief, can mask signs of pain and endotoxaemia**
- Phenylbutazone –2.2 – 4.4 mg/kg iv q12 – 24h. Weaker analgesic for intestinal pain.
- Dipyrone - 5 – 22 mg/kg iv, im q12 – 24h – weak, limited pain relief.

Anti-spasmodics

- Buscopan compositum (hyoscine-butyl bromide and dipyrone) 0.5 mg/kg iv once. Causes intestinal stasis so avoid with impactions

Control of Pain

Sedative analgesics (α2 agonists)

Very good short acting analgesics, ideal for allowing safe rectal exam and nasogastric intubation

- Xylazine ‘rompun’ 0.3 – 1.1 mg/kg iv, im as required
- Detomidine ‘domosedan’ 10 – 20 µg/kg iv as required
- Romifidine ‘sedivet’ 40-120 µg/kg iv

Opioids

- Butorphanol ‘torbulgesic’ –0.1 mg/kg iv, im as required. 0.01 – 0.02 mg/kg iv, im in combination with with α2s

Fluid Therapy

Assessing Percentage Dehydration

%	Clinical Signs
<5	not detectable
5-8	loss of skin elasticity, slight↑ CRT
10-12	skin tenting, CRT >2s, sunken eyes, dry mms, mild signs of shock (pale/congested membranes, ↑ HR, low temperature, weak pulses)
12 -15	definite signs of shock

Volume required = FLUID DEFICIT = %dehydration x body weight

e.g.10% x 300 = 30 litres

(give one half of deficit over 6 hours, then the remainder over the following 12 hours)

Oral Fluids for treating large colon impactions

- Water – large volumes frequently. For 300kg horse 4-5l water by nasogastric tube q 3-4 hours.
- Oral electrolyte solutions if available
- Mineral oil/liquid paraffin – (laxative) lubricates and reduces water loss from intestine so hydrates obstruction. 5 – 10 ml/kg PO q12 – 24h (1.5-3l for 300kg horse)

Combination of above e.g. 4l water plus 1l liquid paraffin 4x day. Alter depending on progress.

Decision making with colic cases

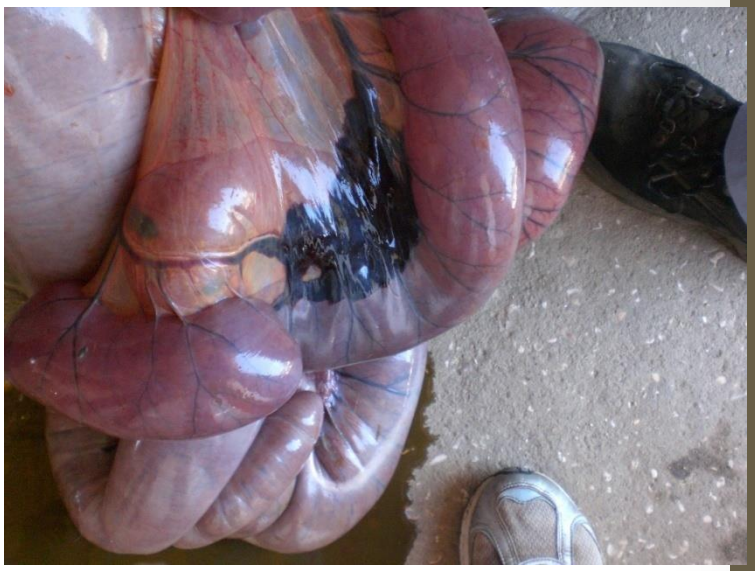
Medical management

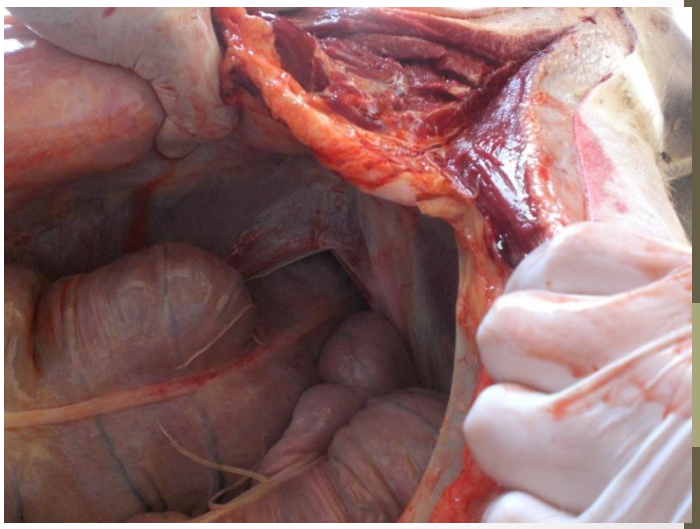
- Bright and alert
- Mild to moderate pain
- HR <60, CV system stable
- Gut sounds present
- Reflux <2l
- Rectal exam Normal or improving
- Good response to analgesia and fluid therapy

Euthanasia (surgery)

- Dull and depressed
- Severe, progressive pain
- HR >80, signs of shock
- Ileus (no gut sounds)
- >5l reflux and ongoing reflux
- Distended S.I., progressive distension/rupture of viscus
- Poor response to analgesia. Progressive shock in face of fluids

Post mortem exam





Prevention of colic

- Avoid sudden change in diet
- Allow regular access to water
- Strategic worming
- Dental care
- Avoid ingestion of sand/rubbish
- Avoid overwork/stress

N.B. Even the best looked after horses still manage to get colic!

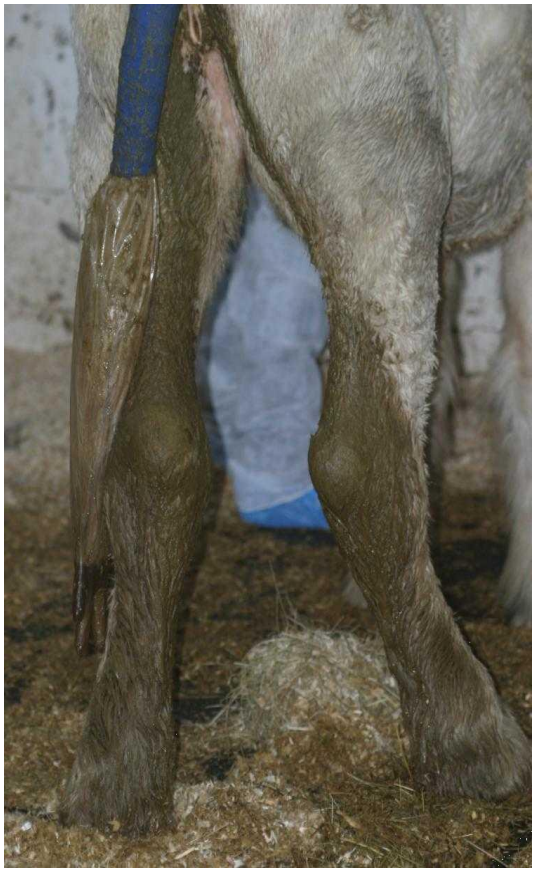
Case 1

- 7 year old stallion, occasional moderate colic signs
- T 38.7, P 48, R 16, mm pink, CRT 1.5s, skin tent –ve, gut sounds hypermotile
- Plan?

Case 2

- 16 yo stallion, dull, moderately painful, T 37.2, P 80, R 40, mms congested, CRT 2.5s, skin tent ++, gut sounds absent, sores on face, eyes slightly sunken and weak pulse.
- Further investigation?
- Plan?

Diarrhea



- Signs can vary from mild, self-limiting change in faecal consistency, to severe colic and profuse watery diarrhoea
- Diarrhoea can be acute in onset or more chronic in nature
- Acute colitis can be fatal in a large percentage (>90%) of horses if not treated
- A definitive diagnosis is made in only 20-30% of cases
- Treatment can be costly due to the fluid therapy
-

Inflammatory diarrhoea

- Majority of acute enterocolitis cases

–Infectious (*Salmonella, Clostridia)* –Toxic (grain overload, NSAIDs, antibiotics)

–Endoparasitic (cyathostominosis)

- Disruption of intestinal barrier ….Absorption of enteric bacterial products -----Lipopolysaccharide (LPS) → hypersecretion, motility disturbance, pain

Acute diarrhoea: non-infectious

1. Grain overload (dietary)

- –Ingestion of excess grain –Colic and abdominal distension followed by diarrhoea and laminitis –Can be acutely endotoxic

2. NSAID toxicity

- –History of administration of NSAIDs, most commonly phenylbutazone
- after prolonged course or excessive doses, although it can occur at recommended drug doses –Lethargy, anorexia, fever, **mild to moderate diarrhoea**, colic, low protein/albumin

3. Endoparasites

3.1. Cyathastominosis (small redworm larvae)

- Young (<5yrs) and elderly horses
- Can have severe diarrhoea or weight loss with ‘cow-pat’ faeces
- Often recent history of administration of anthelmintics
- Seasonal (late winter/spring) –Negative faecal worm eggcount

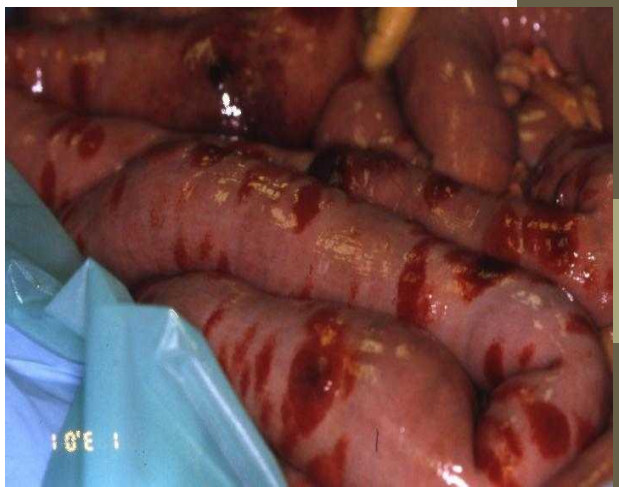
3.2. **Large red worms** (strongyles) associated with colic due to mesenteric emboli

3.3. **Tapeworm** associated with spasmodic colic

3.4. **Ascarids** associated with diarrhoea in young animals and colic due to impaction/perforation

Chronic diarrhoea

1. Infiltrative bowel diseases, including lymphosarcoma
   -Slow onset –Predominantly affect small intestine
   -Malabsorption and weight loss –Thickened intestinal wall palpated per rectum
- Poor prognosis



2. Sand enteritis

–Chronic, intermittent diarrhoea –Impaction colic

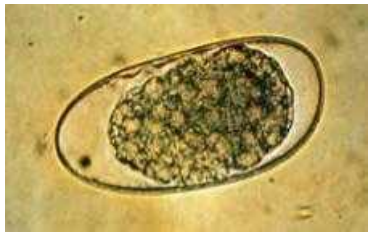
3. Poor dentition
4. Inappropriate nutrition

DX

- Age,
- single/multiple cases
- History – recent
- medication, management, feeding, parasite control, duration, severity of diarrhoea
- Full clinical examination – evidence of dehydration, endotoxaemia
- Faecal analysis :

–Appearance , Fibre length ,consistency

–worm eggs/larva –Sand –culture



- **Faecal culture: when is it significant?**
- **Minimal significance**
- *–Bacteroides fragilis*, *Clostridium perfringens*, *E. Coli*, *Enterococcus*
- *durans*
- **Highly significant**
- *–Aeromonas*, *Salmonella*, *Clostridium difficile* –Formed faeces and serial samples (5) best for isolation of *Salmonella*
- • *C. difficile* very difficult to culture
- • **Toxin detection**

–*C. perfringens* type A enterotoxin; ß2 toxin

–*C. difficile* toxins A and B

Treatment

- Maintain fluid and electrolyte balance
- Oral fluids suitable for mild diarrhoea
- Allow access to fresh drinking water and electrolyte solutions if drinking voluntarily
- Can give fluids via nasogastric tube (indwelling or
- intermittent)

Adsorbents

- Acute, infectious diarrhoea
- Reduces absorption of endotoxin

–Activated charcoal

–Mineral oil

–kaolin

- Administer via nasogastric tube in first 24 hours

Modification of intestinal motility

Codeine phosphate

–60mg tablets. –1-3mg/kg twice daily

–Usually effective within 24hours.

- If no response, increase the dose
- Reduce the dose gradually once the diarrhoea has been resolved

Anti-endotoxic/anti-inflammatory

- •NSAIDs – toxic to colon mucosa, but useful antiendotoxic effect at lower doses (flunixin meglumine :0.25mg/kg every 6 to 8 hours). Good analgesia
- Bismuth subsalicylate – reduces inflammation and secretion. Large volumes for adult (2-3l), more useful in foal

Antibiotic therapy?

- Controversial
- Broad-spectrum cover if low white cell count and septicaemia

Antibiotics themselves can cause diarrhoea

•Metronidazole indicated for clostridiosis: 7.5-10mg/kg three times daily

Corticosteroids

- Inflammatory bowel disease •

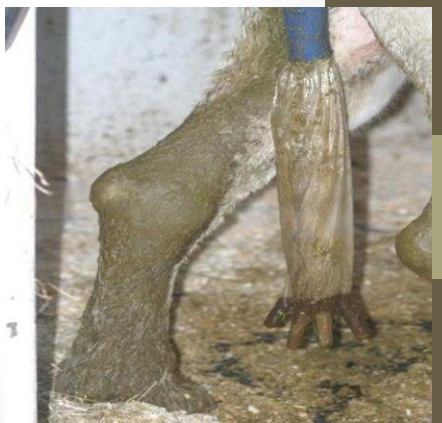
-Prednisolone - 0.5-2.0mg/kg by mouth once daily

-Dexamethasone – 0.1-0.2mg/kg intravenously, orally every day/every other day

- Very gradual reduction once diarrhoea resolved

General nursing

- Isolation and barrier nursing if salmonellosis/clostridiosis suspected
- Vaseline lower limbs
- Tail protection
- Clean perineum and apply petroleum jelly



Prognosis

- Poor if:

–Persistent diarrhoea >48hours

–Liquid faeces –Unable to maintain hydration and electrolyte balance

–Horse ceases eating and drinking

–Secondary complications such as laminitis

–Moderate-severe hypoalbuminaemia

Better if:

–Intermittent diarrhoea or soft/cow-pat faeces

–Mild/no endotoxaemia

–Able to maintain hydration and electrolyte balance

–Horse continues to eat and drink

–Resolution within 48hours

–Mild/no hypoalbuminaemia

–No secondary complication

Diseases of liver

Acute liver disease

- The most common cause of acute hepatic failure is theiler's disease(serum heptitis)
- It occurs only in adult horses and can involve an individual animal or in sporadic outbreaks
- Administration of equine derived biologic products has been frequently reported predisposing agent associated with the onset of the disease
- No infectious agent has been identified
- Other rare causes include acute toxicosis with mycotoxins, liver abscessation, suppurative cholangitits, cholelithiasis, fatty infiltration

Hx and PS

- Signs of depression
- Hepatoencephalopathy
- Hx of administration of equine biological(e.g. TAT)
- Pruritis/photosensetization
- coli

CF and Dx

- Iterus
- CNS derangemnt(head pressing, aimless wandering and yawning)
- Dx is usually based on history, clinical signs and clinicopathologic derangemnts
- Hypoglycemia
- Alteration in serum biochemstry (increased AST, GGT, bilirubin, bile acids)

Treatment

- Control of behavior: xylazine
- Provision of high energy foods:5%dextrose(2L/h)
- Vitamine B -complex

Chronic liver failure

- Horses with this disorder usually are presented because of chronic weight loss or the onset of signs consistent with hepatic insufficiency
- Common cause is exposure to hepatotoxic plants particularly those containing pyrrolizidine alkaloids

CF and Dx

- Annorexia or inapptence is common
- Behavioral changes(hepato encephalopathy)
- Dependent edeoma
- Decreased gut sounds,colic and watery feces
- Skin lesions(photosensitization/pruritis) may be seen
- Profound icterus
- Elevation of liver enzymes
- hupoproteniema

Treatment

- Control of behavior: xylazine
- Provision of high energy foods:5%dextrose(2L/h)
- Vitamine B –complex
- Plasma transfusions(6-10ml/kg) to increase plasma proteins temporarly

Cholelithiasis

- Calculi in the common bile duct are thought to be the result of ascending or heamatogenous infection from deodnum
- Calculi can be single or multiple, and if bilariy stasis occurs, clinical manifestation usually occurs
- Usually occurs in more than 5 years
- Mild colic, inapptence, signs of depression
- Alterations in behaviour(hepato encephalopthy)
- Fever in some cases
- Marked increases in liver enzymes , bilirubin and bile acids

RX

Supportive:

control of behaviour (xylazine)

high energy diet(5%dextrose)

Abs: cotrimoxazole (15-30 mg/kg)

Pancreatic diseases

Acute and Pancreatitis

- Although rare in the horse, diseases of the Pancreas may be evident as either acute episodes of severe inflammation or chronic non specific destruction secondary to abscessation (Streptococcus, corynebacterium spp),eosinophilic granulomas,or fibrosis due to strongyle migrations
- If generalized, chronic hepatitis sometimes results in beta cell destruction and subsequent typ I diabetes

HX and PS

Acute

- Acute severe colic
- Depression and shock
- Abdominal distention

Chronic

- Usually in older horses and in particular in those with an irregular deworming history
- Weight loss and signs of ill thrift
- Depression
- Polyuria and polydypipsia
- polyphagia

CF and DX

Acute:

- Clinical signs of acute pancreatits are non specific and frequently resemble those found in cases of small intestinal obstruction or sever inflammation
- Signs of hypovolumic shock(tachcardia, tachypnea, prolonged capillary refill time, congested MMs and dehydration
- Increase in serum amylase and lipase and hypocalcemia

Chronic :

Polyphagia, polyuria , polydypsia, hyperglycemia, glucosuria and ketonuria

Treatment

Acute

- Supportive therapy
- Large volumes of balanced intravenous fluids with added calcium
- Gastric decompression

chronic

Insulin: 0.5 -1 units/kg