**Floppy Kid Syndrome**

Depression, weakness, flaccid paralysis, abdominal distension.
Repeated episodes of spontaneous recovery and comatose state. Could be every 15-20 min.
Results from over-consumption of milk + "infectious agent" (Clostridium/ E. coli) Suckle more often and consumes more milk.
Supplementing the does with corn or a concentrate feed. Irrigated/improved pastures some time before parturition.
Production of lactic acid in GI because of bacterial fermentation (E coli & C perfringens type D)

**Diagnosis/Treatment:**

Clinical signs
Systemic metabolic (lactic) acidosis
Reverse acidosis with isotonic bicarb (156 mEq /liter) over 1-3 hours or calculate deficit (deficit x body weight Kg x 0.4 = mEq deficit. 4.2% bicarb=0.5mEq/ml; 8.2%=1mEq/ml). Give half the calculated deficit over 3 hours then recheck pH
Oral bicarb (1g baking soda= 12mEq bicarb; 1 teaspoon=5gm baking soda)
Antibiotics/Vit E selenium.
Should see improvement over 6-10 hours
Small amount of milk for 2 days then back with doe

**Hypoglycemia: (Presents like Floppy kid)**

**Differentials include Coccidiosis, Sepsis or Exposure**

Blood glucose < 50
Congested mucus membranes, hypopyon, leucopenia, degenerative left shift, high fibrinogen if septic. Dehydration.
Hypothermic, necrosis of extremities with cold exposure
Positive fecal in case of coccidiosis.
Diarrhea may be present with both coccidiosis and sepsis

**Treatment:**
2-10 ml 50% Dextrose IV or 20ml 50% Dextrose mix with 30ml ml water. Inject IP just lateral and caudal to umbilicus with back legs hanging
Continue with 2.5%-5% Dextrose in isotonic fluids
Ceftiofur/Potassium penicillin
Tulathromycin/gamithromycin
Flunixin

**Coccidiosis treatment**

**Oral**
Amprolium solution (Corid® - 9.6%).
If multiple cases treat all adult goats, yearlings and kids.
Kids are very susceptible at weaning.
*Not approved by the US FDA for use in goats.*
Can predispose to polio
Ponazuril 10-20mg/kg once a day for 3 days (E mac in camelids)
Sulfa drugs (sulfadimethoxine-sulfamethazine). Not approved in goats

**Feeds:**
Feeds containing decoquinate (brand name Deccox®) US FDA-approved for prevention of coccidiosis in non-lactating goats.
Monensin (brand name Rumensin®) US FDA-approved in feed for prevention of coccidiosis in non-lactating goats.
Lasalocid (brand name Bovatec®) has US FDA approval for sheep but not goats.

**Drinking water.**
Amprolium.
Limit access to any other water source.

**Salt toxicity**

Pet lamb/calf on milk replacer (2.6% salt). No free water

**Signs:**

- Blindness
- “Star gazing”
- Vocalization
- Ataxia
- Head pressing
- Nystagmus
- Muscle twitching
  - Diarrhea may be hemorrhagic
- Seizures (paddling; loss of consciousness)
- Hemoglobinuria

**Other differentials for hemoglobinuria:**

- Rapid intake of water following deprivation
- Psychogenic polydipsia
- Lepto

**Pathophysiology:**

Acute or chronic
Treatment: Example

20kg animal with a CSF sodium of 165 mEq/L

Sodium correction rate of not more than 0.5 mEq/hr = 12 mEq in 24 hours

Use 0.45% NaCl (77 mEq Na/L)

Change in Na conc. caused by 1 liter 0.45% NaCl = \[ \text{Na} \text{0.45%} - \text{Na present} \quad \text{[body weight Kg x 0.6]} + 1 \]

\[
\frac{77 - 165}{(20 \times 0.6) + 1} = \frac{98}{13} = 7.5 \text{ mEq}
\]

1 liter of 0.45% NaCl will reduce the total sodium of the 20kg animal by 7.5 mEq. We need to lower the sodium by 12 mEq/24 hours thus the 20kg animal needs to receive 1.6 L 0.45% NaCl over a 24 hour period

OR

Make a high sodium crystalloid solution (HSCS) that is 10 – 15 mEq less than animal’s blood concentration.

Use dry table salt (17 mEq Na and Cl/gram) and add to 0.9% NaCl (145 mEq/Liter). Give IV at 1-3 ml/kg/h. Monitor neuro signs and blood Na concentrations 24-48 hours
Prognosis for salt toxicity is guarded.

**Polioencephalomalasia: Thiamine deficiency**

**Signs:**
Depressed
Blindness
Star gazing
Head pressing
Ataxia
Opisthotonus
Extensor rigidity/
paddling and seizures

**Pathogenesis:**

Thiamin (B1) is essential for the oxidative decarboxylation of the multienzyme branched-chain ketoacid dehydrogenase complexes of the citric acid cycle
Uncouples mitochondrial energy metabolism
Symptoms more centered on brain because of high metabolic rate
High sulphur increases demand for thiamine and decrease the availability of Zn and Cu

**Causes:**

Idiopathic
Iatrogenic Use of amprolium for coccidiosis treatment particularly camelids
Amprolium is a thiamine analogue
Rumen acidosis. *Bacteroides sporogenes and Bacillus thiaminolyticus* produce thiaminase
High sulphur in feed or water
Thiaminase containing plants

**Treatment:**

Remove predisposing cause
Thiamine 20mg/kg IV tid. Give sub cutaneous in camelids
Antibiotics in absence of diagnosis - Listeria
Listeria

Clinical forms include:
Neonates.
  Septicemia. FPT. Crias
  Acute deaths
Abortion
Multifocal brainstem disorder
Diffuse meningoencephalitis
Myelitis

Clinical signs
Anorexia, depression, fever
Asymmetric CNS signs
Conscious proprioceptive deficits
Cranial nerve deficits
Head pressing
Compulsive circling
Recumbent
Torticollis; head tilt; unilateral vestibular signs
Cranial nerves 5-7 usually dysfunctional
CN V - dropped jaw; asymmetric jaw closure; facial analgesia or anesthesia.
CN V1 – medial strabismus on ipsilateral side of lesion
CN V11 – Ptosis; loss of menace; absent palpebral reflex; drooped ear; loss of
  levator nasolabialis function; depressed lip tone; deviated muzzle
Exposure keratitis
Head tilt and circling towards the side of the lesion
CN V111 Nystagmus changes as position of head is altered. Nystagmus can be
  horizontal, vertical or rotatory and is inconstant
CN 1X, X, X11 Dysphagia; stertorous breathing; paresis/paralysis of the
tongue.
  Decreased consciousness, coma, convulsions

Pathophysiology:
Unclear if spread hematogenously or ascendant from cranial nerves. Can be produced by iv inoculation
Produces a hemolysin and microabscesses
Eruption of permanent teeth in younger animals

**Diagnosis:**

Clinical signs/ progression/response to treatment
CSF
  > Protein; mononuclear cells
  Rarely diagnosed on culture

**Epidemiology:**

Silage – aerobic conditions with pH > 5.4
Decaying forages and rotting hay
Asymptomatic carriers. Excreted in feces and milk especially during late pregnancy and parturition
Survives for long periods in environment – up to 2 years in dry soil

**Treatment:**
Poor response to treatment common
Must treat early and continue for up to 1 month
Oxyteracycline IV 10mg/kg twice a day
Potassium penicillin IV 40 – 50,000 IU/kg every 6 hours for first 7 days the procaine penicillin 20,000 IU/kg IM once a day for 3 weeks
Fluid and electrolyte balance should be maintained

**Melting Ulcer**

**Treatment:**

“Drawstring” tarsorrhaphy. Provides protection of the cornea by closing the eye but at the same time allows for multiple treatments per day by opening and closing the eye using the draw string. Pieces of flat plastic is used taking note
that the distance between the perforations on the outer (bottom) piece is more narrow compared to the others. See illustration.

Make sure to go exactly through lid margin otherwise the suture will rub the eye

Treatment:

Topical
  Serum tid. Macroglobulin. Anti MMP
  Fluoroquinolone bid
  Atropine tid

Parenteral
  Oxytetracycline
Pregnancy Toxemia

Patient Profile and History:
Late pregnancy
Obesity
Multiple lambs
Sudden feed changes
Cold Weather

Etiopathogenesis:
High glucose drain from fetuses
High blood cortisol response cause insulin resistance
Some sheep have low insulin response (pregnancy diabetes)
Starvation; stress induced lypolysis
Ketoacidosis and fatty liver

Clinical Findings:
Neurologic signs - ketoacidosis
Head pressing
Circle
“Star Gaze”
Tremors; Convulsions
Other signs:
Recumbent; comatose
Death due to decomposition of fetuses

Etiopathogenesis:
High glucose drain from fetuses
High blood cortisol response cause insulin resistance
Some sheep have low insulin response (pregnancy diabetes)
Starvation; stress induced lypolysis
Ketoacidosis and fatty liver
Pathophysiology: Lipids

Excessive amounts of fatty acids released from adipose tissues – Hormone sensitive lipase (HSL) activated by endogenous steroids; growth hormone; glucagon; catecholamines
Blood nonesterified fatty acid (NEFA) levels rise
The liver takes up fatty acids with the aim of oxidizing them (beta oxidation), but fatty liver develops when it is overwhelmed and fatty acids are converted back into TG

Diagnosis:
Clinical signs
Hypoglycemia < 75 mg/dL
Ketonemia; ketonuria
Hypocalcemia sometimes
Low insulin
Elevated serum NEFA concentrations
Low serum triglycerides (VLDL)
Elevated ammonia (waste product)
Low BUN (product of ammonia catabolism)
Stress leukogram
Standard tru-cut® instrument
Abnormal if triglyceride content exceeds 20% (wet wt.)
Floats in formalin if >34%
Histopathological examination:
   - Number of affected hepatocytes
   - Macrovacuoles (> size of nucleus)

Treatment:
Cesarian section
Glucose iv 5% with balanced electrolyte solutions
Insulin 20-40u protamine zinc insulin
Oral propylene glycol
Alfalfa; grain
Establish a positive energy balance by offering a variety of different feeds
Orogastric intubation: alfalfa meal slurries; electrolytes, propylene glycol (glucose precursor), aminoacids, transfaunation

Prevention:
Increasing plane of nutrition last 2 months
Grain .25 pound 4-6 weeks prior to gestation
Grain 2 pounds last 2 weeks 12% protein
Fat ewes should loose weight first 8 weeks of gestation