Potassium imbalance	Cattle eliminate potassium via urine (75%), faeces and milk: only 2% of potassium in ECS K balance and plasma K conc does not always correlate IC & EC K equilibrium affected by: Acid base, Glucose, insulin, catecholamines HypoK: skeletal mm weakness, anorexia, metabolic alkalosis, calves: diarrhoea HyperK: bradycardia, metabolic acidosis	HypoK treatment - Estimate K deficit: 0.4 x K deficit (mEq) x BW - IV – Do not exceed 1mEq/kg/hour with 1g KCl provides 13mEq of K - Oral – 100-200g KCl per os is sufficient to correct hypokalaemia in adult cattle HyperK treatment - Treat acidosis (Sodium bicarbonate)

Botulism	Clinical signs
	- Ataxia/stumbling/knuckling; weakness/ flaccid paralysis of hind limbs, then progress caudally to tail, constipation
	- Paralysis of face, jaw, tongue mm, ☹ eat/drink, tongue hanging out, drooling
	- Shallow abdominal breathing, Paralysis of the eyelids or drooping lids
	- Sudden death
	
	Ingestion of preformed toxin (Types C & D most common) by *Clostridium Botulinum*
	- Source: ensiled feed (e.g. contamination with carcasses), spoiled feed, pica
	- Neurotoxin causes flaccid paralysis progressing to death over a number of days
	- Clinical signs occur 2-6 days after toxin ingestion
	
	Pathogenesis
	- Botulinum toxin absorbed across intestinal wall.
	- Binds to receptors at NMJ on peripheral motor nerves → presynaptic blockage
	- Zinc-binding metalloprotease cleaves specific proteins in synaptic vesicles → inhibits Ach release → flaccid paralysis of striated muscle
	- Death usually due to respiratory or cardiac failure.
		Diagnostics
		- Clinical exam and history (some glucosuric)
		- ELISA C & D toxins/ mouse assay: ☹ sensitivity
		
		Outbreak response & treatment
		- Identify high-risk source: x feeding/ move cattle away from the source
		- Peracute and acute: euthanasia
		- Subacute cattle: expect morbidity >50%.
		  - Supportive: sternal, fluids, electrolytes, nutrition, soft bedding, shelter
		  - Cathartics to remove toxin from GIT
		  - Antibiotics: 2^{o} aspiration pneumonia (procaine penicillin, tetracycline, aminoglycosides x → potentiate neuromuscular block)
		- Vaccination
		
		Prevention
		- Vaccinate (annual in non-endemic areas)
		  - Feed from mixer wagons: definitely vax
		- Avoid applying chicken litter to pasture
		- Walk pasture pre harvest: no animal carcass
		- >1 mth ferment: pH <4.5 x clostridial growth
		- Appropriate storage to prevent contamination
		- Pest/vermin control

Lecture 1: Johne's Disease in Ruminants

Learning outcomes

- Describe how Mycobacterium avium subsp. Paratuberculosis (MAP) establishes endemic infections
- Describe how MAP causes Johne's disease in individual cows: Pathogenesis and Clinical signs
- Remember the basic principles of Johne's disease control
- Contrast methods for diagnosing and monitoring MAP infection in herds
- Interpret test results for MAP / Johne's disease
- Understand the practical implications of test characteristics

Aetiology

- *Mycobacterium avium ss. paratuberculosis* (MAP)
    - Bovine Johne's disease (C strain), Ovine Johne's disease (S strain), Crohn's disease in humans

Pathogenesis & pathology

- Faeco-oral transmission route
    - Makes its way to SI, colonise mucosa and phagocytosed into the system
- As infection progresses, it goes from being localised gut infection to systemic infection
    - Start in mesenteric LN, then widespread infection if she has generalised infection with MAP
- It's a protein losing enteropathy - damages enterocytes

Epidemiology

- Prevalence of infection in Australia: underestimated (notifiable)
    - Endemic in South Eastern Australia; WA, QLD and NT have little/no cases
- Type of infection: lifelong, often subclinical
- Significant impact
    - Cost to farmers: $45 per cow, per year
    - Zoonosis: GIT infections (contaminated milk or drinking water), Crohn's disease
- Reservoir of infection
    - Infected animals
        - Can be shed by clinical and subclinical animals
        - "Super shedders" can exist in the herd: clinical or subclinical
    - Faecal contamination of environment
        - Long survival in environment
        - Milk / Colostrum: poor hygiene when harvesting milk or direct shedding into milk
        - Teats, feeding equipment: poor hygiene when feeding milk
        - Pasture and water

Transmission

- Vertical (transplacental): 50% of clinical dams infect offspring
- Horizontal (faecal-oral): most susceptible in first 12 months of life (highest risk period first 30d)
    - Contaminated calving area or calf rearing facility
    - Dirty teats
    - Contaminated feeding equipment

Clinical signs

Cattle	Small ruminants
- Adults - Weight loss - Reduced milk production - Profuse watery diarrhoea - Submandibular oedema - Hypoproteinemia	- Adults - Progressive weight loss - Unresponsive to supplementary feed or anthelmintics - Diarrhoea unusual