



WorldHorseWelfare
the new name for the ILPH

Emergency Medicine

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Emergency Medicine

- Common Equine Emergencies
 - Severe Lameness
 - Wounds
 - Abdominal pain
 - Respiratory Distress





Emergency medicine

- Distressed/painful horses are inherently dangerous
 - Control situation
- Assess horse as fully as possible given the conditions
 - Airway
 - Breathing
 - Circulation
 - Complete clinical examination
- Appropriate decision on whether it is possible to treat or euthanase immediately
 - Financial considerations
 - Available treatment
 - Prognosis



Severe Lameness

- **Sub – solar abscess**
- Synovial sepsis
- Soft tissue injury
- Fracture





Sub-solar abscess

- Most common cause of severe/non-weight bearing lameness in the horse
- Mostly acute onset, occasionally chronic – owners can mistake for fracture
- Any age/breed and occurs in shod and unshod horses
- Bacteria penetrate the hoof wall or sole, set up infection adjacent to the sensitive laminae/corium
- **PRESSURE** between sensitive structures and solid hoof capsule leads to lameness





Sub-solar abscess

- **Diagnosis**
 - Unilateral moderate to severe lameness
 - Increased digital pulse to ONE foot
 - Heat in the foot
 - Mild pyrexia (usually $<39.0^{\circ}\text{C}$)
 - Occasionally distal limb swelling – chronicity?
 - Hoof tester application

- **DDx**
 - P3 #
 - Laminitis
 - Severe soft tissue injury





Sub-solar abscess

- Treatment
 - Abaxial sesamoid nerve block if very painful
 - Remove shoe/examine nails
 - Pare the foot to establish drainage
 - Poultice the foot until no further drainage
 - Iodine/sugar to dry hoof/sole following poulticing
 - Occasionally the abscess will exit at the coronary band
 - Phenylbutazone 2.2mg/kg per os twice daily



Sub-solar abscess

- Possible sequelae
 - Chronic abscessation
 - Hoof paring, dremmelling
 - Septic pedal osteitis
 - Quittor
 - (Tetanus)
- Prognosis is excellent if treated quickly
- Prevention
 - Improved hoof care/trimming
 - Dry stabling conditions



Pedal osteitis

- Sequel to chronic abscess or nail penetration
 - Chronic discharging sinus
 - Radiography
- Easily treated in the field
 - Abaxial seamoid nerve block
 - Tourniquet at the level of the fetlock
 - Pare tract down to level of P3
 - Curette – noise indicates when healthy bone reached
 - Antibiotic impregnated PMMA beads
 - Foot bandaging to protect wound
- Good prognosis
 - Wound granulates, keratinizes
 - Persistent discharge – inadequate treatment



Synovial sepsis

- Infection of any of the bursae, joints or tendon sheaths will often cause severe lameness
- Microorganisms colonise the synovial structure via:
 - Wounds
 - Joint injection
 - Surgery
 - Haematogenous spread (predominantly foals)
- Pathogenesis
 - Rapid changes that cause effusion and lameness
 - Cytokines, autolytic enzymes
 - Pressure within a closed anatomical space contributes to pain and lameness



Synovial sepsis

- **Diagnosis**
 - Effusion of the joint (unless open draining wound)
 - Heat in the region
 - Generalised oedema/swelling
 - Mild pyrexia, leucocytosis
- **Fluid sample obtained from the synovial structure using aseptic technique**
 - Gross appearance – turbid/haemorrhagic/reduced viscosity
 - WCC $>20 \times 10^9/l$ (Normal $<1 \times 10^9/l$)
 - Neutrophils $>90\%$ (Normal $<10\%$)
 - Total protein $> 40g/l$ (Normal $<20g/l$)
 - Cytology – bacteria identified in approx 25% cases
 - Bacterial culture positive (time delay/contamination)
- **Radiography**
 - Osteomyelitis, fractures etc



Synovial sepsis

- Aims of therapy
 - Treat as soon as infection is identified – delay makes the infection harder to treat and the likelihood of secondary joint disease higher
 - Reduce viable bacterial numbers to a level which the horses' own defences can successfully clear
 - Systemic antibiotics
- Gold standard therapy arthroscopic lavage
 - General anaesthesia required
 - Expense
 - Expertise
 - Equipment



Needle lavage

- Can be performed under general anesthesia or under standing sedation
- If standing then consider local anaesthesia techniques
- Clip limb and prepare surgically circumferentially
- Large volumes sterile fluid (2-15l dependant on the joint and size of the horse)
- DO NOT add iodine/chlorhexidine to lavage solution – detrimental to the joint
- 14G, 16G or 18G needles dependent on the joint affected



Synovial sepsis

- Intra-articular medication
 - End of lavage/sole treatment
 - Highest concentrations achievable in synovium (10-100x systemic)
 - Will cause inflammation of the synovium as well
 - Gentamicin, Ceftiofur – minimal
 - Dose should be subtracted from the total systemic dose – foals especially
- IVRA
 - Delivers high local concentrations of drug
 - Osseous infections
 - Multiple synovial infections





Synovial sepsis

- **Aftercare**

- Bandage the limb/treat concurrent wound
- Phenylbutazone (Low dose? Necessary? Masks lameness?)
- Repeated medication of joint/centesis of fluid

- **Prognosis**

- Fair if the infection is treated in the acute phase





Chronic synovial sepsis

- Failure of treatment of acute sepsis/delay in treatment
- Fibrin clots form within the joint
 - Cannot be removed with needle lavage
 - Excellent medium for bacterial growth
- Constant infusion systems
 - Deliver high antibiotic concentrations directly into structure
- Open drainage
 - 3-5 cm arthrotomy incisions made into the joint
 - Lavage of the joint performed with sterile fluids
 - Repeated intra-articular antibiotics
 - Systemic antibiotic therapy
 - Arthrotomy incisions kept open to encourage drainage
 - Incisions **MUST** be protected with sterile dressings



Foot penetration

- Common injury in the horse
- Vital structures run very close to the sole of the foot
- Position on foot, direction and DEPTH of penetration important
- Potential sequellae
 - Sub-solar abscess
 - Septic pedal osteitis
 - Septic navicular bursa/distal interphalangeal joint
 - Deep digital flexor tendon/Impar ligament injury
 - Ideal environment for *Clostridium tetani*



Foot penetrations

- **Diagnosis**
 - Increased digital pulse/heat
 - Penetrating wound/foreign body identified
 - Radiography
 - Synoviocentesis – DIP, navicular bursa
- **Treatment**
 - Debride tract
 - Antibiosis – systemic and intra-synovial
 - Tetanus antitoxin
- **Prognosis**
 - Synovial sepsis – poor
 - DDFT/Impar ligament injury – fair (no sepsis)
 - Sub-solar abscess – good/excellent



Fractures

- Many equine fractures are not amenable to fixation
 - Size of animal
 - Strength of implant
 - Immediate weight bearing
- Expense, time and equipment needed to treat many fractures is prohibitive
- Various fractures are amenable to conservative management
 - Standing surgery
 - External coaptation
 - Cross tying
 - Box rest
 - Time +++





Non-displaced radial fractures

- Severe lameness
- Often following kick to the medial aspect of the antebrachium
- Variable amount of swelling
- Pain on palpation/percussion of the radius
- Radiography (+ 7-10days)
- Often spiral and comminuted fracture



Non-displaced radial fractures

- Treatment
 - Sedation
 - Appropriate wound management (?open fracture)
 - Antibiosis if open
 - Phenylbutazone initially (1.1 – 2.2 mg/kg po BID)
- External coaptation
 - 3-4 layer support bandage placed
 - Caudal splint (fetlock to olecranon)
 - Lateral splint (ground to mid scapula)
- Prevent the horse from lying down!
 - Splints (most won't lie down)
 - Cross ties





Non-displaced radial fractures

- Healing will take 3-4 months
- Splints removed at 2 months depending on healing
 - Repeat radiography
- Begin walking exercise (2-3 months)
- Small paddock turnout prior to return to work
- Excellent prognosis if no complications
- Tibial fractures – less common but similar management



Non-displaced fractures of the ulna

- Displaced fractures give typical 'dropped elbow' appearance
 - Poor prognosis without internal fixation
- Non-displaced fractures can respond to conservative management
 - Radiography
 - Caudal splint if the horse is having trouble weightbearing
 - Cross tie
 - Better prognosis if non-articular
- Prolonged healing time
 - Possibility of elbow joint OA in the long term
 - Continued lameness



Small metacarpal/metatarsal fractures

- Common injuries following trauma
- Easily identified on palpation/radiography due to superficial location
- Distal $\frac{1}{2}$ of all splint bones can be removed surgically
 - Concurrent soft tissue injury
 - Callus causing impingement
- Proximal $\frac{1}{3}$ of MC IV and $\frac{1}{2}$ of MT II and IV
 - Don't remove
 - Stability of carpus/tarsus
 - Open – curettage, lavage bandaging
 - Closed – conservative beware callus formation



Pelvic fractures

- Often due to trauma such as a fall
- Sequel to intense exercise intensity in racehorses
- External landmarks asymmetrical
- Rectal examination (crepitus?)
- Muscle atrophy in chronic cases
- Ultrasonography useful, radiography less so.



Pelvic fractures

- Treatment
 - Stall rest 3-4 months
 - Analgesia
 - Support bandaging, frog support
- Non-displaced fractures often heal without complication
- Displaced fractures in mares
 - Severe compromise of the birth canal can occur – dystocia
 - ‘High risk’ mares



Lamintis

- Common cause of moderate to severe bilateral/quadrilateral lameness
- A wide range of conditions will result in the development of the disease
 - Colitis/enteritis
 - Pneumonia
 - Metritis
 - Endocrine/metabolic disorders
 - Severe concurrent musculoskeletal disease
- More common in forelimbs (60% weightbearing)



Laminitis

- **Pathogenesis**
 - Laminitis occurs secondary to systemic inflammatory response syndrome
 - Cytokines, neutrophil activation, COX-2 expression
 - Vascular endothelial cell dysfunction
 - Venous vasoconstriction
 - Lamina oedema
 - Insulin resistance - EMS
 - Impairs glucose uptake into lamina cells
 - Oxidant stress
 - Matrix metalloproteinases 2 and 9
 - Responsible for normal growth of hoof past P3
 - Over activation leads to basement membrane dissolution
 - Mechanical/traumatic theory
 - Vasospasm/oedema formation
- **Interconnected and complex theories**
 - Likely mechanical and mediator component to all cases



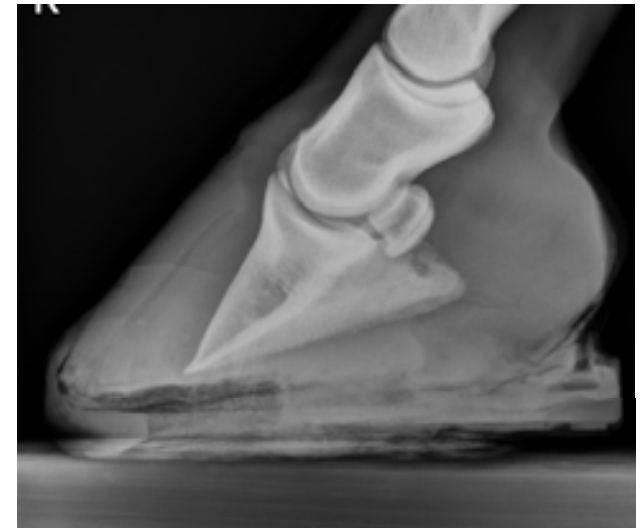
Laminitis – hoof anatomy

- **Dermal laminae**
 - Attached to the sub-cutis and periosteum of the distal phalanx
 - Blood supply from laminar arteries- branches of circumflex artery
 - Multiple AVA – reduce blood flow through laminar capillaries
- **Epidermal laminae**
 - Interdigitate with dermal laminae to support P3
 - Primary and secondary EL
- **Laminitis causes a loss of support of the distal phalanx within the hoof capsule**
 - Rotation of P3 wrt dorsal hoof wall
 - ‘Founder’ or sinking of P3



Laminitis

- Obel grading system
 - Grade I – Weight shifting, stilted gait at trot
 - Grade II – Willing to lift feet, stilted gait at walk
 - Grade III – Resist lifting feet, reluctant to walk
 - Grade IV – Refuses to move unless forced
- Clinical signs
 - Increased digital pulses ++
 - Increased hoof temperature
 - Hoof tester sensitivity
 - Dropped sole
 - Palpable depression at the coronary band
- Radiography
 - Useful as a diagnostic tool and prognostic indicator





Laminitis - Treatment

- Controversy over different treatments – clinician preference
- IF possible instigate treatment before clinical signs develop
 - Mineral oil via NGT (grain overload)
 - Endotoxaemic therapy
 - IVFT
 - NSAIDs
 - Cooling of the distal limbs
 - Experimentally 0°C
 - Practical implications



Laminitis – Medical therapy

- **Anti-inflammatory therapy**
 - Reduce oedema, pain and inflammation
 - PBZ – best musculoskeletal analgesic – 2.2 – 4.4mg/kg i/v, p/o
- **Vasodilators**
 - ACP 0.03-0.06 mg/kg i/m q8hrs, higher doses orally
 - Isoxoprine hydrochloride – no proven efficacy
- **MMP inhibitors**
 - Doxycycline – weak effect in the horse, GIT side effects
- **Aspirin – inhibition of platelet aggregation/vasoconstriction**
 - 10–20mg/kg orally once in every 48 hour period
- **Complete box rest – dependent on level of pain**



Laminitis – foot care

- Reduction of mechanical forces
 - Rest – do not force to walk
 - Sand (best) or deep bedding
 - Frog supports
- Shoeing
 - Heart bar shoe
 - Glue on support shoes
- Partial dorsal hoof wall resection





Exertional Rhabdomyolysis

- Common cause of severe lameness/stiffness
 - Single event of over exertion
 - Recurrent events – underlying muscle disorder
- Can be difficult to differentiate from
 - Colic
 - Laminitis
 - Peritonitis/rupture
 - Pneumonia
- Clinical signs
 - Increased heart rate/respiratory rate
 - Reluctance to move
 - Sweating
 - Muscle fasciculation
 - Myoglobinuria



Exertional Rhabdomyolysis

- **Diagnosis**
 - Haematology (PCV/TP minimum),
 - Biochemistry (CK/AST)
 - Urinalysis
- **Treatment**
 - IVFT/Oral fluid therapy
 - NSAIDs
 - ACP?
 - Rest/exercise restriction
 - Keep horse warm
- **Prevention**
 - Diagnosis or underlying myopathy
 - Daily exercise/slow changes in exercise intensity



Wounds of the distal limb

- Traumatic injuries common
- First aid - owner
 - Rough decontamination
 - Arrest haemorrhage
- Structures involved
 - Skin
 - Tendon/ligament
 - Osseous
 - Synovial
- Treatment
 - Financial and prognosis considerations





Treatment of distal limb wounds

- Sedation and local anaesthesia
- Debridement and lavage
- Partial closure
 - Rarely completely close
 - Drain placement
- Bandaging
 - Robert Jones
 - Splints
 - Bandage casts
 - Casts
- Antibiosis
 - Systemic and local



Treatment of distal limb wounds - chronic

- Sequestra
- Movement
- Skin grafting





Soft tissue injuries of the distal limb

- Lacerations and ruptures of the flexor/extensor tendons and suspensory ligament in the distal limb are common
- Superficial location
- Synovial sheaths over areas of movement
- Gait abnormalities allow which ligament is injured to be identified
 - Hyperextension of fetlock only - SDFT
 - Hyperextension of fetlock and toe lift – SDFT + DDFT
 - Toe lift and palmar fetlock to floor – SDFT+ DDFT + SL



Extensor tendon injuries

- Synovial infection of extensor tendon sheaths less serious
 - Respond better to conservative management
- Complete lacerations don't require repair
 - Little or no long term consequences
 - Often 'knuckle' at the fetlock initially
- Partial resections of extensors can be performed with minimal long term consequences



Respiratory Distress

- Upper respiratory tract obstructions
 - Bilateral nasal obstruction (rare)
 - Pharyngeal obstruction
 - Strep equi equi
 - Laryngeal obstruction
 - Hepatic encephalopathy - paralysis
 - Physical airway obstruction - arytenoid chondritis, sub-epiglottic cyst
 - Upper tracheal obstruction
 - Anaphylaxis
- Emergency tracheotomy
 - Tracheal tube if possible
 - Or anything to provide a suitable airway



Emergency Tracheotomy

- Junction of cranial and middle third of the ventral trachea
- Clip and surgically prepare (if possible)
- 10ml mepivacaine hydrochloride s/c
- 10cm incision through skin and sub-cutis
- Split paired sternothyrohyoideus
- Incise parallel to the tracheal rings, aim less than 1/3 of the circumference
- When removed allow to heal by second intention



Respiratory Distress

- Lower airway
 - Recurrent airway obstruction
 - Pleuropneumonia
 - Acute respiratory distress syndrome
 - Incorrect NGT placement
 - Smoke inhalation/near drowning
 - Pneumothorax (rare)



Recurrent Airway Obstruction

- Hypersensitivity to environmental pathogens
- Acute episodes of small airway obstruction overlying chronic disease
 - Smooth muscle contraction
 - Accumulations of mucus and neutrophils
 - Can be severe
- Clinical signs
 - Afebrile
 - Spontaneous cough
 - Increased respiratory rate, inspiratory and expiratory lung sounds
 - Ultrasonography – often normal – no gas/fluid in pleural space



Recurrent Airway Obstruction

- **Corticosteroids**
 - Reduce airway inflammation
 - Dexamethasone 0.1mg/kg i/v
 - Approx 24hrs action
 - Follow with course of oral prednisolone/dexamethasone
- **Beta2-adrenergics**
 - Bronchodilator
 - Clenbuterol i/v or orally
- **Atropine**
 - Anticholinergic – smooth muscle relaxation
 - Use if required and available – short duration of action/side effects
 - 0.02 mg/kg short duration of action
- **Long term – environmental management**



Pleuropneumonia

- Usually an extension of pneumonia/lung abscess
 - Less commonly secondary to trauma, oesophageal rupture etc
- Long distance transport, previous viral respiratory disease, dysphagia
- Clinical signs
 - Febrile
 - Lethargic
 - Shallow breathing, increased respiratory rate
 - Painful/stilted gait
 - DDx Rhabdomyolysis/colic
- Diagnosis
 - Absence of lung sounds ventrally
 - Ultrasonography



Pleuropneumonia

- Thoracocentesis
 - Ultrasound guided or blind
 - Diagnostic tool
 - Normal WCC $<10 \times 10^9/l$, 60% Neutrophil, TP $< 25g/l$
 - Therapeutic benefit
- Technique
 - 7-8th IC space, dorsal to costochondral junction
 - 24-32 French chest tube
 - 30-50l of fluid can be removed
 - Leave in situ with Heimlich valve/condom placed over the end



Pleuropneumonia

- **Antibiosis**
 - Broad spectrum Pen/Gent, cephalosporin initially
 - Long courses of oral antibiosis – 2-3months
 - NSAIDs
 - IVFT
- **Prognosis**
 - Better if identified and treated early in the disease course
- **Complications**
 - Severe endotoxaemia
 - Laminitis
 - Pleural adhesions, pulmonary abscess/necrosis



Pneumothorax

- Rare in the horse?
- Absent lung sounds in the dorsal thorax
- Radiography/ultrasonography
- Similar procedure to the chest drain, placed further dorsally
- One way valve placed on the drain, air drained with a 60ml syringe



Acute respiratory distress syndrome

- Alveolar damage, pulmonary oedema, respiratory failure
- Aspiration/NGT/Smoke inhalation/severe pulmonary infection
- Clinical signs
 - Tachypnoea/respiratory distress
 - Red tinged frothy material at nares
 - Delay in onset following smoke inhalation?
 - Crackles over lung fields
 - Fluid in trachea?
- Treatment
 - Lipid pneumonia (mineral oil) nearly always fatal
 - Symptomatic treatment beta2 agonists, NSAIDs, corticosteroids



Severe epistaxis

- **Guttural pouch mycosis**
 - Most common cause of severe epistaxis
 - +/- dysphagia, Horner's syndrome, parotid pain
- **Diagnosis**
 - Hx , clinical signs
 - Endoscopy –care if recent bleed – leave 2-3 days?
- **Treatment**
 - Antifungal (preferably powder) instilled daily into GP
 - Internal carotid ligation
- **Complications**
 - Fatal epistaxis, ongoing dysphagia, laryngeal hemiplegia, temporohyoid arthropathy



Colic

- Approximately 95% of colic episodes will respond to medical management
- Medical
 - Spasmodic
 - Pelvic flexure impaction
 - Caecal impaction
 - Anterior enteritis
 - Large colon displacements
- Surgical
 - Strangulating lesions of the small or large intestine
 - Ileal impaction, small colon impaction
 - Non-resolving non-strangulating lesions
- ‘False colic’





Colic

- History
 - Recent worming, previous colic, change in management
- Clinical examination
 - Level of pain
 - Environment, breed, age
 - Sweating, muscle fassiculation
 - Cardiovascular parameters
 - HR, mucous membrane colour/crt, PCV/TP, lactate
 - Abdominal auscultation
 - Rectal examination
 - Nasogastric intubation





Colic

- Additional tests
 - Abdominocentesis
 - Gross examination, TP, lactate, PCV?
 - Abdominal ultrasonography
 - Radiography?
 - Laparoscopy??





Colic - Treatment

- NSAIDs
 - Phenylbutazone 4.4mg/kg i/v
 - Flunixin meglumine 1.1mg/kg i/v – anti-endotoxic effects?
 - Ketoprofen, Meloxicam
- Spasmolytics
 - Butylscopolamine
- Fluid therapy
 - Enteral – Water, MgSO₄
 - IVFT
- Light exercise
- Phenylephrine infusion
 - If confirmed nephrosplenic entrapment, u/s?





Colic – PTS/Surgery

- Non-response to analgesia
- Consistent (increasing) HR>60bpm
- Congested MM, delayed CRT
- Deteriorating rectal findings
- Continuing reflux
 - Anterior enteritis vs SI strangulation lesion





Oesophageal obstruction

- Usually caused by impaction of dry feed in the oesophagus
 - Uncommon to be one single foreign body
 - Hx of abnormal feeding
 - Dental disease?
- Clinical signs
 - Saliva from the nostrils/mouth
 - Palpable swelling ventral neck
 - +/- distress
- Diagnosis
 - NGT cannot be passed through into the stomach
- DDx
 - EGS, botulism, colic, dysphagia





Oesophageal obstruction

• Treatment

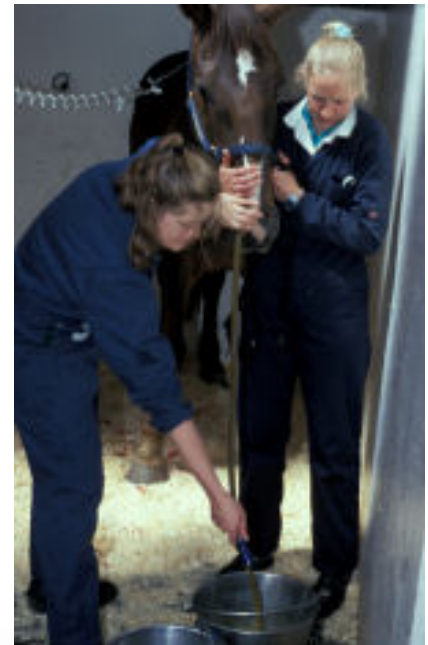
- Sedate the horse heavily enough to lower head carriage significantly
- Detomidine (0.01mg/kg) + butorphanol (0.02mg/kg) intravenously
- Spasmolytics – reduce muscular spasm of the oesophageal wall
 - Butylscopolamine
 - Atropine?
- NSAIDs to reduce the inflammation

- Clearing the obstruction with lavage
 - Passive lavage via gravity
 - Active lavage – need a cuffed tube into the oesophagus

- Not trying to clear obstruction for 12 hours?

• Complications

- Aspiration pneumonia, oesophageal stricture/rupture





Shock and intra-venous fluid therapy

- ANY event which leads to poor blood supply/oxygen delivery to the tissues will result in shock
 - Progresses to altered metabolism, cell death, organ failure, death
- Classified according to causative factor
 - Cardiogenic
 - Hypovolaemic
 - Maldistributive
 - ENDOTOXAEMIA
 - Metabolic





Shock - Pathophysiology

- Inadequate cellular oxygen delivery leads to
 - Anaerobic metabolism
 - Altered gene expression



- Shortage of ATP and production of lactic acid



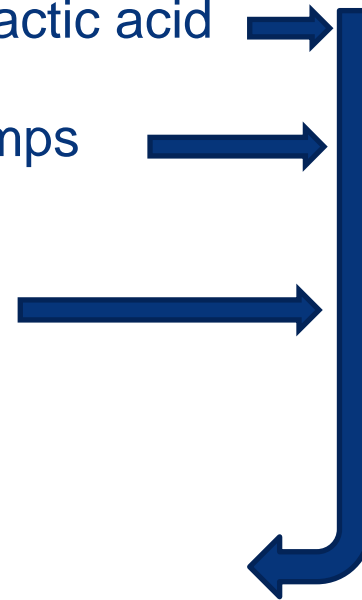
- Derangements in Na/K/ATP-ase pumps
 - Increased Na⁺, Ca²⁺, hypoxanthine



- Increased reactive oxygen species



- Inflammatory response (SIRS)
 - VASODILATION
 - INCREASED CAPILLARY PERMEABILITY





Shock - Pathophysiology

- Inflammatory response then 'drives' the effects of shock
- COX/LOX pathways
 - Prostaglandins, thromboxanes, leukotrienes
- Kinin system, complement activation, coagulation cascade activated
- Inflammatory cells activated
 - Release cytokines, acute phase response
- If SIRS is left untreated then MODS develops **—————>** death



Shock - Pathophysiology

- Body tries to maintain VITAL organ function
 - Sympathetic nervous system, RAAS, ADH
- Compensatory stage
 - Tachycardia, reduced urine output, tachypnoea, congested/pale mm, incr. CRT
 - Hyperglycaemia due to stress response
- Decompensation
 - Vasodilation, incr. vascular permeability, negative inotropy, deranged metabolism
 - Very prolonged crt, bradycardia, hypoglycaemia, DIC, death



Treatment

- Oxygen - rarely available in the clinical setting with horses
- IVFT mainstay of treatment + practical:
 - Crystalloids
 - Synthetic colloids
 - Plasma
 - Blood
- Assess haemoconcentration
 - PCV/TP – slow in hypovolaemic shock
 - Skin tent/mucous membranes
 - Or administer until clinical signs improve ie reduce HR, improve mm colour
- Primary disease process
 - If this is not treated/treatable then pointless treating endotoxaemia – ie colic



Intra-venous fluid therapy

- **Crystalloids – 0.9%NaCl, Hartman’s solution – ECF replacers**
 - ‘shock’ rate (80ml/kg/hr) NOT achievable in the horse
 - Only ¼ of volume administered remains intravascular – redistribution
 - Hypertonic saline (7.2%) 4ml/kg (~2l per horse)
 - Short duration of effect (less than an hour)
 - ‘Pulls’ water from intracellular and interstitial spaces
 - Follow with isotonic fluids to replace loss, DON’T repeat
- **Colloids**
 - Large molecules which should stay intravascular – beware leaky capillaries
 - 10-20ml/kg (per day) ‘plasma volume expanders’
 - Coagulopathies
- **Plasma**
 - Harvested from donor mares – availability/cost?
 - Albumin, globulin, clotting factors etc