INITIAL ASSESSMENT AND STABILISATION OF THE COLLAPSED FOAL

Celia M. Marr
Rossdales Equine Hospital, Newmarket, Suffolk, UK

RISK FACTORS

- MATERNAL & PRE-PARTUM FACTORS
  - Any form of maternal illness
  - Placentitis
  - Placental Insufficiency
  - Early lactation

- PARTURITION
  - Foaling in adverse environment
  - Dystocia
  - Caesarian Section

- POST-PARTUM FACTORS
  - Orphan
  - Any form of illness
  - Rejection of the foal
  - Poor quality colostrums
  - Poor milk supply

ASSESSING THE PLACENTA

- Check it has all been expelled
- Identify
  - focal areas of thickening - oedema/inflammation
  - loss of villi - villous atrophy

- Weight
  - normal 4.5 - 6.75 kg
  - infection: > 6.75 kg
  - villous atrophy < 4.5 kg

PLACENTESIS

- Usually ascending infection
- Lesions may be most obvious around cervical star
- May be associated with:
  - fever, anorexia, malaise, and premature mil secretion in the mare
  - septicaemia in the perinatal foal

PLACENTAL INSUFFICIENCY

- Pathology: villous atrophy
- Dysmaturity
- Azotaemia in the neonate - returns to normal concentrations within 24-48 hours provided the foal's renal function is adequate

PREMATURITY & DYSMATURITY

- Prematurity - a foal born at a gestational age of < 320 days that displays immature physical characteristics
- Dysmaturity - a full-term foal that displays immature physical characteristics

PHYSICAL CHARACTERISTICS

- low birth weight
- weakness, prolonged time to stand
- short, silky hair coat
- floppy ears

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• domed head
• flexor tendon laxity
• incomplete ossification of tarsal and carpal bones

COLOSTRUM

PRODUCTION
• contains essential immunoglobulins that will confer immunity until the foal’s own immune system is mature
• also contains complement and lactoferrin (proteins essential to immune function)
• The mammary gland concentrates immunoglobulins from the mare’s blood during last 2-4 weeks of pregnancy: IgG, IgG(T), IgA and IgM
• Secreted into milk on one occasion
• if mare has run milk prior to foaling the quality of colostrum may be poor

ABSORPTION OF COLOSTRUM
• Specialist enterocytes absorb the immunoglobulins by pinocytosis
• These cells have a lifespan of a maximum of twenty-four hours
• Maximum absorption occurs within eight hours of life
• foal must ingest around 1 litre of colostrum within the first six hours of life
• Half-life of maternal IGG = 20-23 days
• Autogenous IGG detectable at 2 weeks, adult levels reached at 4 months
• Serum IGG concentrations lowest at 1 - 2 months of age

ASSESSING COLOSTRAL INTAKE
• best guide to colostral quality is specific gravity
• measure IgG concentration in foal’s blood at 18 hours of age
• Radioimmunoassay
• ELISA tests
• Zinc sulphate turbidity

COLOSTRUM STORES
• up to 250 mls from a nursing mare without affecting her own foal
• freeze at -20oC for 18 months
• thaw at room temperature or in a waterbath not exceeding 38oC.
• Bovine colostrums can be used if equine is not available

FAILURE OF PASSIVE TRANSFER
• most common predisposing cause of infection in foals of less than 2 weeks of age

DEFINITION
< 400 mg/dl - total
400-800 mg/dl - partial
>800 mg/dl - complete

TREATMENT OPTIONS
• Administer plasma intravenously until IGG concentration is adequate
• Administer antimicrobials - for a few days, until umbilicus has closed?
• Do nothing - but monitor foal carefully for signs of infection

CLINICAL ASSESSMENT OF THE NEONATE
EXAMINATION FROM A DISTANCE
• Is it breathing normally?
• Is it displaying normal suckling behaviour?

DETAILED EXAMINATION
• What are the localising signs?

CLINICAL OBSERVATIONS
• Time to begin suckling reflex: within 20 minutes
• Time to stand: 57 (15 - 165) minutes
• Time to nurse from mare: 111 (35 - 420) minutes
- Body Temperature: 99 - 102oF; 37.2 - 38.9 oC
- Heart Rate: Birth 40 -80 bpm; First week 60 - 100 bpm
- Meconium - dark brown pellets or paste all passed within 24 hours
- Urine - dilute and large volumes first passed by six hours (colts) or ten hours (fillies)
- Average weight (Thoroughbred): 45 - 55 kg
- Weight gain: 0.5 - 1.5 kg/day
- Daily consumption of mare’s milk: 20 - 28% of body weight
- Respiratory rate newborn: 45 to 60 bpm with small abdominal component and no nostril flaring or exaggerated rib movement. Periods of tachypnea may be present during REM
- 7 days of age - 35 to 50 bpm
- Auscultation of lung fields
  - caution, not a sensitive diagnostic tool, extensive parenchymal disease may be unremarkable
  - normal foal lungs have harsh bronchovesicular sounds and crackles are present in the ventral dependent side if in lateral recumbency
- Thoracic symmetry and rib fractures
- Mucous membrane colour – caution not very sensitive
  - Cyanosis: < PaO2 30 - 40 mmHg
  - Signs of sepsis: congestion/injection, petechiae
- Loud murmurs are common in foals
  - Patent ductus arteriosus
  - Foramen ovale
  - Forward flow through great vessels
  - Congenital heart disease is uncommon, and with the exception of VSD will lead to cardiac failure in the first few days or weeks of life
  - When a loud murmur is detected in an otherwise healthy foal - monitor over the next few weeks and the majority with disappear
- Entropion and consequent corneal ulceration is common in debilitated foals and it is important not to overlook and fail to treat this
- Assess other body systems carefully, particularly
  - umbilicus
  - joints

**CLINICAL IMPERATIVES IN EQUINE NEONATOLOGY**
- Most illnesses in the neonatal period are life-threatening and can deteriorate rapidly
- Rapid recognition of abnormal behaviour and signs
- Quick action when the foal does not behave normally, are necessary
- May not need to reach specific diagnosis
- More important to:
  - Determine need for antimicrobial therapy (i.e. identify sepsis)
  - Determine need for respiratory support
  - Determine need for intensive care
  - Nutrition, blood pressor support, anti-oxidant therapy etc

**IDENTIFICATION OF SEPSIS**
- Blood culture & antimicrobial sensitivity
  - obtain blood in sterile manner
  - prior antimicrobial administration reduces likelihood of positive culture
  - delay before results available
  - provides information on pathogens in your area
- Sepsis Score
  - Developed to overcome limitations of blood culture
  - Results should be acted on quickly
  - Scores assigned to clinical and historical features
    - Neutrophil numbers and morphology
    - Fibrinogen concentration
- blood glucose
- serum IGG
- Sensitivity 93%; Specificity: 88%

- Localised sepsis
  - Umbilical infection
    - Ultrasonography
  - Pneumonia
    - Radiography, blood gas analysis
  - Osteomyelitis/arthritis
    - Synovial fluid analysis, radiography

DETERMINE THE NEED FOR RESPIRATORY SUPPORT
- Inspection of respiratory effort
- Blood gas analysis
- Generally all recumbent foals will have some degree of respiratory compromise

DETERMINE THE NEED FOR INTENSIVE CARE
- Clinical history and signs
- Blood glucose concentration
- PCV/TP and creatinine

MAJOR DIFFERENTIAL DIAGNOSES FOR THE COLLAPSED NEONATE

GENERALISED SIGNS
- Septicaemia
- Prematurity/dysmaturity
- Perinatal asphyxia syndrome (hypoxic-ischaemic encephalopathy)
- Neonatal Isoerythrolysis
- Uroperitoneum
- Congenital cardiac disease

NEUROLOGICAL SIGNS
- Perinatal asphyxia syndrome (hypoxic-ischaemic encephalopathy; neonatal maladjustment syndrome)
- Septicaemia
- EHV1
- Bacterial meningitis
- Neonatal Isoerythrolysis

COLIC
- Meconium impaction
- Perinatal asphyxia syndrome
- Enteritis (septicaemia)
- Surgical lesions - e.g. jejunojejunosal intussusception, intestinal atresia
- Ruptured bladder

DIARRHOEA
- Perinatal asphyxia syndrome
- Foal heat diarrhoea
- Enteritis (Septicaemia)
- Clostridia
- Campylobacter
- Rota Virus

LAMENESS
- Septicaemia
- Fractures
- Soft tissue injury
- Angular limb & flexural deformities
NEONATAL SEPTICAEMIA

COMMON PATHOGENS
- E coli, Actinobacillus, Salmonella, Proteus, Klebsiella, other gram negative spp.
- beta-haemolytic Strepococcus, Staphylococcus, Clostridia
- mixed infections possible

ROUTE OF INFECTION
- In utero
- Inhalation
- Ingestion
- Via umbilicus

SEPTIC SHOCK
- Vasoactive inflammatory mediators lead to vasodilation
- Increased metabolic rate and oxygen consumption
- Cardiac output is increased initially (hyperdynamic phase)
- Microvascular permeability leads to volume maldistribution
- Increased cardiac output can no longer be maintained (hypodynamic phase)
- Culminates in:
  - Multiple organ failure
  - CNS depression
  - Renal failure
  - Autonomic exhaustion and decompensation of circulation
  - Gastrointestinal dysfunction

EARLY CLINICAL SIGNS
- Foal goes off-suck and is lethargic
- Increased respiratory rate and effort
- Acute severe lameness
- Discharge or swelling of umbilicus
- Fever is not consistent
- Petechial haemorrhages

ANTIMICROBIAL SELECTION
- Cefquinome, ceftriaxone, ceftiofur
- Aminoglycosides
  - care in very young foals - nephrotoxicity
  - used in conjunction with gram positive cover
- Penicillins and other beta-lactams
- Trimethoprim-sulpha
  - organisms may not be sensitive

POLY ARTHRITIS & OSTEOMYELITIS
CLASSIFICATION
- S Type: synovitis with no bony involvement
- E Type: in joint and adjacent epiphysis
- P Type: bone infection adjacent to physis
- T Type: infection of the small tarsal bones

POLY ARTHRITIS & OSTEOMYELITIS
TYPICAL SIGNALMENT
- S Type: a few days or older
- E Type: any age
- P Type: 9 - 90 days

CLINICAL SIGNS
- S Type: acute onset, one or several distended joints, systemic signs
- E Type: acute onset, one or several distended joints, systemic signs
• P Type: variable lameness, + swelling or pain over physis, joint distension & systemic signs frequently not present

TREATMENT
• S Type: aggressive and frequent joint lavage
• E Type: joint lavage, arthroscopy
• P Type: curettage

PROGNOSIS
• S Type: good if few joints affected, also depends on degree of other systemic involvement
• E Type: fairly poor, also depends on degree of other systemic involvement
• P Type: fair to good

UMBILICAL INFECTION
• The umbilicus consists of two arteries, the urachus and one vein
• It can act as a portal of entry of infection
• Infection can localise there following haematogenous spread
• Arteries and urachus tend to become infected more commonly than vein (in contrast to calves)

CLINICAL SIGNS
Fever, malaise, lethargy
• Swelling and discharge from umbilicus
• Localising signs are not present in every case, particularly when there is generalised septicemia

ULTRASONOGRAPHIC SIGNS
• Enlargement (normal vein and arteries measure <1cm)
• Accumulation of anechoic or hypoechoic material and/or gas echoes within umbilical structures

TREATMENT
ANTIMICROBIAL THERAPY
• Monitor response with ultrasonography

SURGICAL RESECTION
• Removes risk of spread of infection to other sites
• If there is generalised septicaemia, the foal may not be a good candidate for anaesthesia

BACTERIAL MENINGITIS
uncommon manifestation of neonatal septicaemia

CLINICAL SIGNS AND DIAGNOSIS
Seizures, bizarre behaviour, central blindness, ataxia
CSF analysis and culture
Culture of blood or aspirates from tachea or joints may also reveal specific pathogens

TREATMENT
ANTIMICROBIAL SELECTION
• Aminoglycosides do not cross blood-brain barrier
• Penicillins and other beta-lactams only cross the blood-brain barrier when meninges inflamed
• Cefquinome, ceftriaxone
• Other cephalosporins

PERINATAL ASPHYXIA SYNDROME
• Damage to foal’s brain, kidneys, intestine and other organs due to lack of oxygen
  o In utero hypoxia
  o Interruption of oxygen supply during birth
• Clinical signs may not be apparent until the foal is 12-24 hours old

PATHOLOGY
• Oedema
• Ischaemia
• Haemorrhage

CLINICAL SIGNS
• Signs appear within first 12 - 24 hours after birth
• + history of dystocia or other hypoxic event
• Often have dysfunction of other organs systems, particularly renal and gastrointestinal dysfunction
• Dummy foal, Barker, Wanderer
  o Off suck, lethargic
  o Does not recognise or follow mare
  o Seizures, sometimes with bizarre vocalisations
  o Colic, diarrhoea
  o Anuria, oliguria or polyuria
  o Occasionally presents as other forms of neurological dysfunction
    ▪ dysphagia
    ▪ muzzle and feed via nasogastric tube
    ▪ urinary retention and overflow
      ▪ urinary catheter to keep bladder small and prevent irreversible damage to muscles
      ▪ avoid bethanecol?

Anticonvulsants
• diazepam - 5 mg iv, to effect (up to 4 times)
• phenobarbitol - 10-20 mg/kg over 15 to 30 min then 5 - 10 mg/kg iv, bid, tid
• midazolam - Infusion: add 45 mls 0.9% saline to 5 ml midazolam (5 mg/ml) administer at 2 – 6 ml/hour for a 50 kg foal.

Anti-inflammatories and antioxidants
• Magnesium sulphate - 50 mg/kg/hr for first hour then 25 mg/kg/hr for next 35 – 47 hours
• Vitamin E – 10 iu/kg, SID, po
• DMSO - 1 gm/kg in 20% solution over several hours for three days
• Mannitol - 0.25 - 1 gm/kg q4 hrs iv, 20% solution slowly

Stimulants
• Naloxone - 0.01-0.02 mg/kg iv

Prognosis
• fairly good providing nursing can be provided unless multiple organ failure is present

NEONATAL ISOERYTHROLYSIS

Pathogenesis and Clinical Signs
• Qa and Aa blood groups are major cause
• Degree of haemolysis and consequent clinical signs are dependent on dose of antibody ingested
  o Small amounts - mild malaise and lethargy, 1 to 3 days after birth
  o Large amounts - severe jaundice, depression, seizuring (bilirubin is toxic to CNS)

Diagnosis
• Anaemia
• Increased haemoglobin
• PVC:Hb < 3:1 confirms haemolysis
• Increased indirect (unconjugated) bilirubin
• Coombs positive

Treatment
• Washed RBCs from mare
• Blood from a cross-matched horse
• Blood from a gelding of the same breed
• Volume required:
  o Body weight (kg) x blood vol (ml/kg) x (PCV desired-PCV observed) / PCV of donor blood
Prevention

- Blood volume, 2 days = 150 ml/kg
- Prevent “at risk” foals from ingesting colostrum from their dam by identification of:
  - Mares that are Aa and Qa negative by blood typing (specific stallions may then be selected for breeding)
  - Rising anti-RBC antibody concentrations in late pregnancy
  - Minor cross match (foal RBC with mare serum)
- Jaundice Foal agglutination simple “foal side” test

**RESPIRATORY DISTRESS SYNDROME**

Pathogenesis

- Atelectasis due to
  - Inadequate surfactant function
  - Structurally immature lung and muscles of respiration
  - Associated with prematurity and dysmaturity

**MECONIUM ASPIRATION**

Pathogenesis

- Stress in utero or during parturition leads to defecation,
- Meconium enters airways with fetal fluid,
- Aspirated when foal is born and starts breathing
- Chemical pneumonia
- Secondary bacterial pneumonia

Clinical signs
- Respiratory distress, nasal discharge

Diagnosis
- Meconium staining

**ASPIRATION PNEUMONIA**

- Dysphagia
- Neurological - uncommon, transient, manifestation of NMS
- Cleft palate
- Inappropriate bottle feeding
  - Very common in sick foals with inexperienced nursing staff

**PNEUMOTHORAX & HEMOTHORAX**

- Trauma at birth
- Rib fractures are common and affected foals are often asymptotic

**NON-RESPIRATORY DISEASE WITH RESPIRATORY SIGNS**

- Excitement
- Fever
- Poor thermoregulation
- High ambient temperatures
- Pain
- Acute abdominal crisis (colic)

**UROPERITONEUM**

**AT PARTURITION**

- Colts - narrow pelvis and long urethra
- Dorsal aspect of bladder
- Present with progressive lethargy and abdominal distension from 1-3 days of age

**SECONDARY TO INFECTION**

- Urachus, bladder or ureters
- From birth to around two months of age
- Usually history of other illness
- Easy to miss because deterioration in clinical status can be mistakenly attributed to pre-existing disease

**UROPERITONEUM: DIAGNOSIS**
- Peritoneal: Serum creatinine ratio > 3:1
- Ultrasonography

**ACID-BASE AND ELECTROLYTE DERANGEMENTS**
- Hyperkalaemia
  - Failure to excrete
  - Leads to cardiac arrhythmias
- Hyponatraemia
  - Loss of renal regulation
  - Continued intake of water
- Metabolic acidosis
  - Loss of renal regulation
  - “Third spacing” leads to hypovolaemia and to poor perfusion
- Respiratory acidosis
  - Thoracic compression

**PRE-OPERATIVE STABILISATION**
- Bicarbonate, dextrose and insulin to promote intracellular movement of potassium
- Restore circulating volume - 0.9% NaCl or Hartman’s solution
- Drain abdomen
- Intranasal oxygen
- Insulin (0.1 units/kg) and 0.5 gm/kg dextrose in 500ml saline over 30 -45 min
- HCO₃ (mEq/l) = Body weight (kg) x Base deficit x 0.4
  - Give one half in one hour, recheck HCO₃ and recalculate.

**NEONATAL INTENSIVE NURSING**

**CONTINUOUS FLUID THERAPY**
- More appropriate than bolus administration
- Proportionately, total body water is higher in foals than in adults
- Renal function is marginal and respiratory disease is common, therefore it is important not to over-hydrate foals

**Maintenance Formula** 50 kg foal
- 100 ml/kg for first 10kg 1 l
- 50 ml/kg for next 10 kg 0.5 l
- 25/ml kg for rest of body weight 0.8 l
- Over 24 hours 2.3 l

**REPLACEMENT SOLUTIONS OPTIONS**
- Hartmann’s if acidotic
- 0.9% saline if alkalotic
- Plasma
- Avoid bicarbonate solutions
  - Compromised respiratory function
- Avoid hypertonic saline
  - Marginal renal function

**MAINTENANCE SOLUTIONS**
- Avoiding sodium overload is more important in foals than adults
- Dextrose-saline solutions

**BOLUS FLUID THERAPY**
- Much more convenient
- Introduce as soon as possible
- Particularly helpful if foal is semi-ambulatory
- Only suitable for Hartmann’s, 0.9% saline and plasma - not dextrose solutions
HYPOGLYCAEMIA
- assess with dextrostix and glucometer
- 5 - 10% dextrose at 4 - 8 mg/kg/min
- 50 kg foal, 10% solution, 120 - 240 ml/hr
- Wean off slowly

RESPIRATORY SUPPORT
- postural
  - moving the foal from lateral to sternal recumbency is one of the most important things that you can do to improve respiratory function
- intranasal oxygen
  - in absence of blood gas analysis - assume oxygen is required in all recumbent/semi-recumbent foals - 2 - 5 l/min
  - soft intranasal tubing attached to firmer tubing shaped to head
  - oxygen tank with regulator - care if mare is free in stall
  - humidifier with distilled/sterile water
  - will improve oxygenation but not ventilation
- mechanical ventilation
- drugs such as central stimulants doxapram and caffeine are not as effective as mechanical ventilation

NUTRITION

ORAL ROUTE
- Healthy foals consume 20-28% of their body weight per day and Feed 2-3 times every hour
- Goats’ milk is nearest in composition to mares if powdered milk not available
- Sick foals frequently have reduced GI motility
- do not feed hypothermic foals
- start at 5% of body weight divided into 12 - 24 feeds, increase by 1-2% every 12 hours, if foal tolerating feeding
- commonest mistake - over feeding
- sepsis increases requirement
- ileus common

INTRAVENOUS ROUTE
- iv glucose - only suitable for 24 hours, metabolic acidosis
- total parentral nutrition:
  - solution of aminoacids, lipids and dextrose
  - administered through a central venous line
  - sterility critical - compounding, double lumen catheter

BLOOD PRESSURE
- indirect arterial blood pressure monitor
- aim to keep MAP > 65 mmHg
- inotrophic and pressor support
  - dobutamine 2 – 10 µg/kg/min
  - noradrenaline 0.1- 5 µg/kg/min
  - vasopressin 0.25 – 0.5 mU/kg/min
- must have carefully controlled infusion rates

PASSIVE IMMUNITY
- oral products - adjunctive therapy but should not be used exclusively to treat failure of passive transfer
- septicemia can “use up” antibody
- plasma from mare
  - time consuming
  - antibody to “local” pathogens
  - not suitable if mare is sick?
• commercial plasma
  o expensive
  o convenient
  o guaranteed quality

MANAGING RECUMBENCY
• suitable bed
• warm environment
• keep in sternal
• turn every two hours
• encourage to stand
• keep as dry and clean as possible
• intra-nasal oxygen

COMMON SECONDARY PROBLEMS
• entropion and corneal ulceration
  o correct vertical mattress sutures
  o fluroscein stain frequently
  o artificial tears/gentomycin ointment/drops
• patent urachus
  o iodine
  o silver nitrate cautery sticks
• joint and tendon laxity
• incomplete ossification of cuboidal bones
  o careful clinical and radiographic assessment
  o re-assess when foal becomes more ambulatory
  o exercise restriction
  o

SUMMARY AND KEY-POINTS
• sick foals deteriorate quickly
• septicaemia is common
• most neonatal diseases are multi-systemic and difficult to differentiate on clinical signs alone
• early and aggressive therapy is critical
• supportive therapy is critical