Resuscitation of the Critically ill Foal

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Chester

- *Tb Colt*
- *Birth date: May 7, 6 PM*
- *Admission date: May 8, 8:53 AM*
- *15 hours old*
Chester

History

- Born on May 7 at 6 PM
- On day 338 of gestation
- Normal gestation
- Parturition - normal

Placenta

- Edematous
- Meconium stained
Chester
History

- Fetal diarrhea
- Assisted to stand after 1.5 hours
  - Nursed
- Farm manager left the barn
  - Foal watcher
Chester History

- **During night**
  - Foal was never vigorous
  - Got up once unassisted
    - but only for short time and did not nurse

- **Next morning**
  - Weak, inability to stand, only nursed once
  - Foal needed medical attention
  - Called her veterinarian, requesting referral
  - Waited transport
    - Bottle-fed Chester 8 oz. of colostrum milked from the mare
Chester
Admission 15 hours old

- Arrived recumbent
  - required transport to the NICU
- A rapid evaluation
  - Vital signs
  - Essential organ function
  - As initial therapy begun
Critical Neonate

Admission Procedure 1\textsuperscript{st} 10 minutes

- Team approach
- Vital signs and essential organ function
  - Rapid physical assessment of essential organ function
  - ABG/electrolyte sample
  - PCV, TP, dextrose (venous and arterial)
  - Blood for culture/laboratory analysis
  - Indirect blood pressures recorded
- Initial therapy
  - Intranasal oxygen
  - Jugular catheter
  - Intravenous fluid administration
    - Resuscitation strong ion balanced crystalloids
    - Glucose containing fluids begun
Chester

Vital Parameters

- Temp 38.0°C (100.4°F)
- HR 96 bpm
- RR 24 bpm
- BP 88/50 (61) 91
- PCV 44%
- TP 6.1
- Venous Dextrose 44 mg/dl
- Arterial Dextrose 59 mg/dl
- Wt 54 Kg (119 lb)
Chester

Essential Organ Function

- **Injection, Icterus**
- **Oral petechiae**
- **Cardiovascular**
  - Ice cold hooves
  - Cold lower legs
  - Cool nose, ears and upper legs
  - Weak pulses, poor arterial fill, poor arterial tone
  - Relative bradycardia (90-96)
  - Evidence of recent urination
Chester

Essential Organ Function

- Fetal diarrhea - subsiding
- CNS
  - Somnolent
  - Meaningful struggling
  - Arousable, responsive
  - Subdued
Chester

Initial Supportive Therapy

- Intransal oxygen insufflation - within 3 min
- Fluid therapy begun within 10 min
  - Dextrose 5% in water
    - Rate to deliver 4 mg/kg/min (8 mg/kg/min)
  - Balanced replacement crystalloid
    - Normisol R®
    - 20 ml/kg boluses over 10-20 minutes
    - Reevaluation of cardiovascular status
- Plasma transfusion
  - Colloid value
  - Biologically active proteins
- Intravenous Timentin®
  - Bolus q6h
  - CRI
Resuscitation of the Critical Foal

- Critical signs
  - Inconsistent or lack of nursing behavior
  - Weak or develop progressive weakness
  - Recumbent
- Immediate intervention
- In our practice
  - 70-80% neonates admitted < 48 hrs
  - Fatal outcomes
    - 70% die within initial 48 hrs
- Essential for success
  - Rapid referral
  - Assessment of essential organ function
  - Immediate directed, supportive therapy
- Coordinated care delivery team
Resuscitation of the Critical Foal
Cardiovascular Examination

- Effective perfusion
  - Macroperfusion
  - Microperfusion

- Signs of hypoperfusion
  - cold extremities
  - pulse quality - pulse pressure
  - arterial tone
  - arterial fill
  - Blood Pressure
  - Urine output
  - Mental status
  - GI function
Resuscitation of the Critical Foal

Blood Pressure

- Blood pressure
  - Vital sign
  - Measure in all critical neonates

Technique

- Direct
- Indirect
  - Oscillometric technique
  - Minimize errors
    - Cuff size
    - Cuff placement
    - Measure during a quiet or sleep state
    - At least 3 measurements
    - Mean value is least likely erroneous
Resuscitation of the Critical Foal

Blood Pressure

- Blood pressure is related to perfusion
  - Flow \approx \text{pressure/resistance}
  - Objective numbers are obtained
  - Used as a surrogate for perfusion
  - Dangerous assumption
    - Changing peripheral resistance
Blood Pressure
Neonate Transition State

- From a low pressure fetal circulation
  - Low systemic blood pressure vital for fetal physiology
    - Low precapillary tone
    - Low baroreceptor set point
- To a normal pressure pediatric circulation
  - Near birth
    - Increase peripheral resistance - increase in precapillary tone
    - Increase in blood pressure to maintain tissue perfusion
      - Shift in baroreceptor sensitivity
      - Progressive increase in BP matching the increase in peripheral resistance
  - Begins before birth, slowly progresses during neonatal period
    - Most evident during the first week of life
    - Transition does not proceed simultaneously in all tissues
Resuscitation of the Critical Foal

Blood Pressure

- Normal blood pressure values a moving target
  - Normal neonatal foal with good perfusion
    - 59/35(45)
  - Another may have hypoperfusion (shock)
    - 73/55(61)

- Low BP should not be treated alone
  - Unless there are coexisting signs of hypoperfusion
    - Cold extremities
    - Poor arterial pulses, fill and tone
    - Oliguria
    - Metabolic acidosis
    - Organ hypoperfusion
Resuscitation of the Critical Foal

Blood Pressure

- BP numbers should not be given more weight in directing therapeutic interventions than any other physical examination finding
Resuscitation of the Critical Foal

Initial Therapeutic Interventions

- Insuring tissue oxygen delivery
  - Maximizing pulmonary loading of hemoglobin
  - Guaranteeing sufficient blood oxygen content
  - Returning perfusion to normal

- Intranasal oxygen insufflation
  - Immediately, before ABG results
  - ABG

- Blood oxygen carrying capacity
  - Birth changes in PCV
  - Anemia
    - PCV < 20% trigger point, sliding trigger
  - Hemoglobin based blood substitutes
    - Significant chance adverse effects
    - Microcirculation key to survival
Chester
Fluid Therapy

- **Intravenous line established < 10 min**
  - BP 88/50 (61) 91
- **Normisol boluses – 1 l over 10 min**
  - Reassess signs
  - BP 65/45 (50)
- **Total of 5 liters (90 ml/kg) + 1 l of plasma - 110 ml/kg**
  - Temp gradient periphery to core decreased
  - Nose, ears and upper legs were warm
  - Lower legs cool and hooves cold
  - No urine
  - Minimally responsive
  - Pulses moderately strong, good arterial fill, poor arterial tone
  - BP was 58/35 (45)
Chester
Inopressor Therapy

- **Dobutamine CRI 10 µg/kg/min.**
  - Leg temperature improved but still cool
  - other signs not changed
  - BP was 69/35(48)

- **Vasopressin CRI 0.5 mU/kg/min.**
  - Hooves and legs were warm
  - Peripheral pulses strong
  - Excellent arterial fill, very good arterial tone
  - BP 75/40(55)
  - Sternal recumbency, head up, vocalized
  - Began to urinate
Resuscitation of the Critical Foal

Inopressor Therapy

- Adrenergic agonists
  - Dopamine
  - Dobutamine
  - Norepinephrine
  - Epinephrine
- Physiologic doses vasopressin
- Physiologic doses of corticosteroids
- Naloxone
- NOS blockers (methylene blue)
Resuscitation of the Critical Foal

Inopressor Therapy

- Pharmacologic doses of adrenergic agonists
  - Increase in perfusion
  - Increase in the maldistribution of that perfusion

- Goal
  - Return perfusion to minimally acceptable levels
  - Not achieve normal or supranormal perfusion
Resuscitation of the Critical Foal

Vasopressin

- Septic shock
  - Vasoplegia
  - Vasopressin deficiency
  - Death
- Vasopressin replacement therapy
  - Returns responsiveness to adrenergics
  - Reverses vasopoplegia
- Vasopressin receptors
  - V1 vascular receptor
  - V2 renal receptor
  - V3 pituitary receptor
  - OTR - oxytocin receptor
  - P2 purinergic receptors
Resuscitation of the Critical Foal

Vasopressin

- Vasopressin in low, physiologic doses
  - Refractory vasodilatory shock states
    - Restore vascular tone
    - Potentiation of endogenous and exogenous vasoconstrictors
  - Directed perfusion
    - Selective vasoconstriction
    - Selective vasodilation
  - Supporting increase in cardiac output
    - Inotropic effect
    - No chronotropic effect – minimizing O₂ consumption

- Vasopressin in larger doses
  - Negative cardiac effect
Resuscitation of the Critical Foal

Vasopressin

- Dose used in foals
  - 0.25-1.0 mU/kg/min
Chester
Dextrose Therapy

- **Glucose levels on admission**
  - Venous glucose 44 mg/dl
  - Arterial glucose 59 mg/dl
- **5% dextrose in water**
  - 259 ml/hour - 4 mg/kg/min
- Within an hour - blood glucose 74 mg/dl
- Within 2 hours – blood glucose 89 mg/dl
- **10% dextrose** - 8 mg/kg/min
Therapeutic Interventions
Dextrose Therapy

- All compromise neonates
  - will benefit from exogenous glucose support
- Blood dextrose levels
  - Not a gas gauge
  - summation of glucose mobilization and glucose utilization
- Placenta transfer rate of glucose
  - Between 4 and 8 mg/kg/min.
  - Fetal blood level 50-60% of maternal
Therapeutic Interventions
Dextrose Therapy

- Fetal distress
  - Develop active glucogenesis
  - Born with a high resting glucose

- Normal fetus
  - Born before glucogenesis begins
  - Birth blood glucose - 25-45 mg/dl
  - Continues to drop for the first 2-4 hours

- Neonate suffering from perinatal disease
  - Not make the transition to glucogenesis
  - Hypoglycemic

- Chester at 15 hours - hypoglycemic
Chester
Glucose Intolerance

- Chester became hyperglycemia
  - on 8 mg/kg/min glucose
- Blood glucose levels
  - 6 hr - 176 mg/dl
  - 12 hr - 225 mg/dl
  - 18 hr 326 mg/dl
    - Glucosuria 500-1000 mg/dl
- CRI of regular insulin
Therapeutic Interventions
Glucose Intolerance

- Failure to adapt to the exogenous glucose load
- Glucose regulation
  - Not needed in fetus
  - Transition at birth
- Exogenous glucose therapy
  - Spare endogenous reserves – prevent catabolism
  - Glucose intolerance
    - Neonate may continue glucogenesis despite exogenous glucose
    - Glucose administration in excess of utilization – insulin problem
    - Iatrogenic glucose overload – calculation errors or bolus therapy
Therapeutic Interventions

Glucose Intolerance

- Dangers of moderate hyperglycemia
  - Without an insulin response
    - Cellular dehydration,
  - Glucose diuresis
    - Fluid and electrolyte wasting

- Advantages of mild hyperglycemia
  - Neonate develop its own innate insulin response

- Dangers of mild hyperglycemia
  - Glucose and insulin have many modulating influences
  - Tight glucose control - intensive insulin therapy
  - No studies in pediatrics or neonatology
  - Should we use tight glucose control?
Therapeutic Interventions

Insulin Therapy

- Continuous infusion of regular insulin
- Respond to surprisingly low insulin levels
  - suggesting insulin deficit
  - Not resistance
- Dose
  - 0.00125-0.2 u/kg/hr
  - Began at 0.0025 u/kg/hr
  - Double rate every 4 to 6 hr
  - if > 0.04 u/kg/hr more slowly
- Special care in preparing and delivering
Resuscitation of the Critical Foal

Lactate

- Origin of lactic acid
  - Traditionally linked to
    - Oxygen debt
    - Magnitude of hypoperfusion
    - Severity of shock
  - Therapy focused
    - Tissue hypoxia
    - Hypodynamic shock
    - Organ ischemial
Resuscitation of the Critical Foal
Lactate

- Other sources
  - Hypermetabolism without lack of oxygen
    - Aerobic glycolysis (from epinephrine)
    - Protein catabolism
  - Increased muscle activity (shivering)
  - Decreased lactate clearance
    - Liver failure
    - Liver hypoperfusion
  - Inhibition of pyruvate dehydrogenase
    - Thiamine deficiency
    - SIRS (secondary to cytokine enzyme inhibition)
  - Activation of inflammatory cells
    - ARDS
    - Liver failure
    - NEC
  - Placenta source
Resuscitation of the Critical Foal

Lactate

- Increased lactate
  - Sign of the critical state of the neonate
  - Cannot be equated to poor perfusion and anaerobic metabolism
    - One possible cause
    - Correcting hypoperfusion is vital
    - Super resuscitation in pursuit of normalizing lactate
      - More harm than good
  - Multiple reasons for an elevated lactate
    - During different disease stages - causes have different importance

- Lactate clearance
  - Important in predicting survival
  - Absolute lactate value not as important

- Chester’s Lactate Clearance
  - 1 hour - 25%
  - 10 hrs - > 60%
  - 24 hr 75% clearance – to 1.6 mmol/l
Chester
Initial Hospital Course

- 4 hours
  - **Cardiovascular status** – good perfusion
  - **Responsive to his environment**
    - **Sternal recumbency**
    - **Hold his head up and look around**
Chester
Neonatal Encephalopathy

- **4.5 hours**
  - Respiratory effort decreased
  - Apneustic breathing (breath holding)
  - Progressive hypercapnic acidosis
    - $Pco2 = 82.3$ torr, $pH = 7.284$
  - Treated with caffeine (10 mg/kg – oral)

- **6 hours**
  - $Pco2 = 62$ torr, $pH = 7.354$
Chester
Neonatal Encephalopathy

- **10 hours**
  - Apneic respiratory pattern
    - 40 second apneic period
    - *Cluster breathing in-between*
  - *Simultaneously*
    - $P_{CO_2} = 85$ torr, $pH = 7.276$
  - *Second dose of caffeine*

- **12 hours**
  - $P_{CO_2} = 45.0$ torr, $pH = 7.451$
Neonatal Encephalopathy

- Respiratory centers are a common target
- Abnormal respiratory patterns
  - Central tachypnea
  - Apneusis
  - Periodic apnea
  - Cluster breathing
  - Ataxic breathing
  - Cheyne-Stokes breathing
- Central hypercapnia
  - Independent of abnormal respiratory patterns
  - Caffeine
  - Mechanical ventilation.
Chester

Neonatal Encephalopathy

- **12 hours**
  - *Periods of somnolence and nonresponsiveness*
  - *Apneic respiratory pattern with cluster breathing*
  - *Facial nerve paresis*
    - right ear lower and slower to respond
    - ears are not synchronized

- **21 hours**
  - *Seizure-like activity*
    - Opisthotonus
    - Tonic/Clonic marching activity
  - *Treated with intravenous phenobarbital*
Chester

Neonatal Encephalopathy

- 27 hours
  - Chester was again hypercapnic
    - \( \text{Pco2} = 74.7 \text{ torr, pH} = 7.313 \)
  - Treated with a third dose of caffeine

- 29 hours
  - \( \text{Pco2} = 84.6 \text{ torr, pH} = 7.269 \)
  - Placed on a volume cycled positive pressure partial ventilatory support
    - SIMV/PS
    - Easily ventilated
    - \( \text{pH} = 7.401, \text{Pco2} = 53.8 \text{ torr, Po2} = 95.7 \text{ torr,} \)
    - \( \text{HCO3} = 33 \text{ meq/l, BE} +8.3, \text{SAT} 99.0\%, \text{O2 Cont} = 14.4, \)
    - alveolar dead space ventilation of 10.4%,
    - peak airway pressure 24 cm H2O and plateau pressure of 18 cm H2O
    - Weaned from the ventilator 48 hours later.
Chester

Summary of Problems

- Bacteremia/Sepsis
  - Admission blood culture - *Pantoea agglomerans*
- Neonatal Encephalopathy
  - HD 9 - nursing mare
  - HD 18 - resolved.
- Neonatal Enteropathy
  - HD 10 – dysmotility, not passing fece
  - HD 19 – complete resolution
- Neonatal Nephropathy
  - Glucosuria
  - Slow drop in plasma creatinine, rising creatinine
  - High F<sub>xNa</sub>
  - HD 18 - resolved.
- Other problems
  - Urachitis, hepatomegaly
  - Linear dermal necrosis, patent urachus
  - Angular limb deformity.
Sick Cell Syndrome

- Foal: Wishful
- Warm Blood filly
- DOB: March 25 1 AM
- Admission Date: March 25 11:25 AM
  - 10 hours old
Wishful
History

- **Born at 1 AM on March 25**
  - Foal began to breathe with nostril flaring
    - As soon as the nostrils cleared the canal
  - Stage II 10 minutes
    - Foal was pulled
  - Stage III
    - Placenta came with the foal
    - Placental horn retained
- **Foal “appeared slow”**
  - From the beginning...but normal
  - Able to stand with help
  - Not searching the mare
  - Became weaker
  - Developed periods of somnolence
Wishful Admission

- **Recumbent on arrival**
  - Transported to the NICU

- **Rapid assessment of essential organ function**
  - **Severe sepsis**
    - Poor pulse quality
    - Cold legs and ice cold hooves
    - Temperature 99.6
      - dropped during initial hospitalization 97
    - HR 104 bpm
    - RR 18 bpm
    - BP 73/30(37)
Wishful Admission

- **Rapid, directed interventions**
- **Treatment of shock**
  - $\text{INO}_2$
  - Crystalloid boluses
    - Responded after 3 X 1 liter boluses
  - BP after fluids
    - 90/58(65)
  - PE – good perfusion
Wishful Admission

- Further examination after initial resuscitation
  - Bilateral entropion
  - Extreme scleral injection
  - Oral drying injuries
  - Icterus
  - Pseudopetechia
  - Moderate coronitis
  - Normal body condition
  - Neonatal skin wrinkling
  - Normally responsive
  - Searches, inducible suckle
  - Can stand with support with good balance
  - Somnolent periods
Wishful
Initial Laboratory Analysis

- PCV = 50
- TP = 7.4
- Fibrinogen = 370 mg/dl
- WBC = 7000
- Segs = 5110
- Bands = 210
- Lymphs = 1680
Wishful
Initial Laboratory Analysis

- Venous Dextrose = 20 mg/dl
- BUN = 24 mg/dl
- Total Ca = 16.38 mg/ml
- Ca++ = 6.84 mg/dl
- Mg++ = 2.79 mg/dl
- IgG = 776 mg/dl
- Total Bili = 4.5 mg/dl
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**Wishful**

**Initial Laboratory Analysis**

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Major finding
- Hyponatremia
- Hypochloremia
- Hyperkalemia

Magnitude of changes
- May require urgent intervention
- Vital to understand the origin of the abnormalities
  - Direct rational therapy
  - Wrong choices – severe consequences
  - Many clinicians assume ruptured bladder
    - easily rule out
    - age
    - lack of fluid intake
Hyponatremia

- Spurious Hyponatremia
- Dilutional Hyponatremia
  - Ruptured bladder
  - Fenestrated ureters
  - Renal failure
  - Delayed renal transition from fetal to neonatal physiology
  - Water overload
- Depletional Hyponatremia:
  - Diarrhea
  - Sodium wasting nephropathy
  - Diuretics
- Redistribution Hyponatremia
  - Other osmoles in the blood
    - Hyperglycemia
    - Iatrogenic addition of osmoles (e.g. mannitol)
    - Sick Cell Syndrome
Wishful Hyponatremia

- Spurious hyponatremia
- Dilutional hyponatremia
  - No intake since birth
- Depletional hyponatremia
  - Not begun to urinate
  - Has not past meconium yet
- Redistribution hyponatremia
  - Water diluting Na come from cells
  - Some osmolyte other than sodium
    - Drawing water from cells
- Source of osmoles?
  - Hypoglycemic
  - Not received exogenous substances
  - Presence of endogenous osmolytes
    - Leaked from cells
Wishful
Hyponatremia

- Significant therapeutic implications
  - No sodium deficiency
  - Not water overloaded
  - Not hyposmotic
    - May be hyperosmotic
- Don’t give sodium
- Don’t induce an unsupported diuresis
Hyperkalemia

- **Mechanisms**
  - High intake
    - Dietary
    - Parenteral
  - Blocked excretion
    - Must have continued intake
  - Leak from cell

- **Wishful**
  - No intake
  - Must be cell leak
Sick Cell Syndrome

- Global loss of integrity of cell membranes
- Acute, severe hypoxic ischemic insult
  - Globally affect cells
  - Loss of cell wall integrity
    - Transient or permanent
    - Allowing solutes to leak
    - Drawing fluid with them
    - Dilution of extracellular sodium
- Redistribution hyponatremia
  - Osmolar Gap (OG)
    - Unmeasured osmolytes
    - \[ OG = Osm_m - Osm_c \]
    - \[ Osm_m = (2X [Na]) + (\frac{glucose}{18}) + (\frac{BUN}{2.8}) \]
    - BUN - not part of effective plasma osmolarit
Sick Cell Syndrome

- OG > 10 mOsm
  - osmoles other than Na or glucose
  - Associated with
    - MODS
    - High fatality rate

- What are the osmoles?
  - Organic phosphate
  - Pyruvate
  - Lactate
  - Amino acids,
  - Unidentified middle molecular weight substances
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Regulatory Volume Decrease

- Another explanation
- Regulatory Volume Decrease (RVD)
  - Fluid overloaded cells
  - All mammalian cells
  - Protective mechanism
    - Limits cell swelling
- Reasons cells swell
  - Hyponatremia
    - Hyposmotic interstitium
  - Initial stages of hypoxic ischemic insults
    - Hyperosmotic cell interior
Regulatory Volume Decrease Mechanism

Voltage-independent, volume-sensitive channels
- Activated by cell swelling
- Allow outflow of
  - K+
  - Cl-
  - Amino acids
  - Other organic molecules
- Water follows
  - restoring cell volume.
Redistribution Hyponatremia
Neonatal Foals

- Both SCS and RVD are involved
- Mild insults
  - Compromise cellular function
  - Allow fluid to leak
  - RVD - protective mechanism
- More severe damage
  - Initially result in RVD
  - Evolve into SCS
Sick Cell Syndrome

- Other cell constituents also leak
  - K+ leak
    - Both RVD and SCS
    - High intracellular levels of K
    - Mild increase in efflux globally
      - Increase plasma K levels significantly
  - CPK
  - AST

- Outcome
  - About 60% of SCS cases do not survive
  - Identification of SCS - guarded to poor prognosis.
Sick Cell Syndrome Therapy

- Don’t treat hyponatemia
  - Not sodium deficit
    - Osmolarity high normal
  - Not water overload
- Hyperkalemia
  - If ECG changes
    - Mg
  - Enhance cell entry
    - Insulin/dextrose
    - B₂ adrenergic
      - Albuterol
    - Na HCO₃ – not recommended
- Enhance excretion
  - Osmotic diuresis
    - Support
  - Furosemide
  - GI t
### Wishful Outcome

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<td>312</td>
<td>295</td>
</tr>
<tr>
<td>Osm&lt;sub&gt;c&lt;/sub&gt;</td>
<td>240</td>
<td>270</td>
<td>275</td>
</tr>
<tr>
<td>Osm Gap</td>
<td>72</td>
<td>43</td>
<td>20</td>
</tr>
</tbody>
</table>
Wishful Outcome

- **Intrauterine Insult – catabolism, SIRS**
- **Sepsis**
  - High fibrinogen, left shift
  - Inject, icterus
  - Shock, increased lactate, acidosis
  - Admission blood culture
    - Flavobacterium
- **Neonatal Encephalopathy**
  - Inconsistent nursing behavior
  - HD 6 - nursing from mare