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 1st 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 38.0C (100.4F)*
- *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

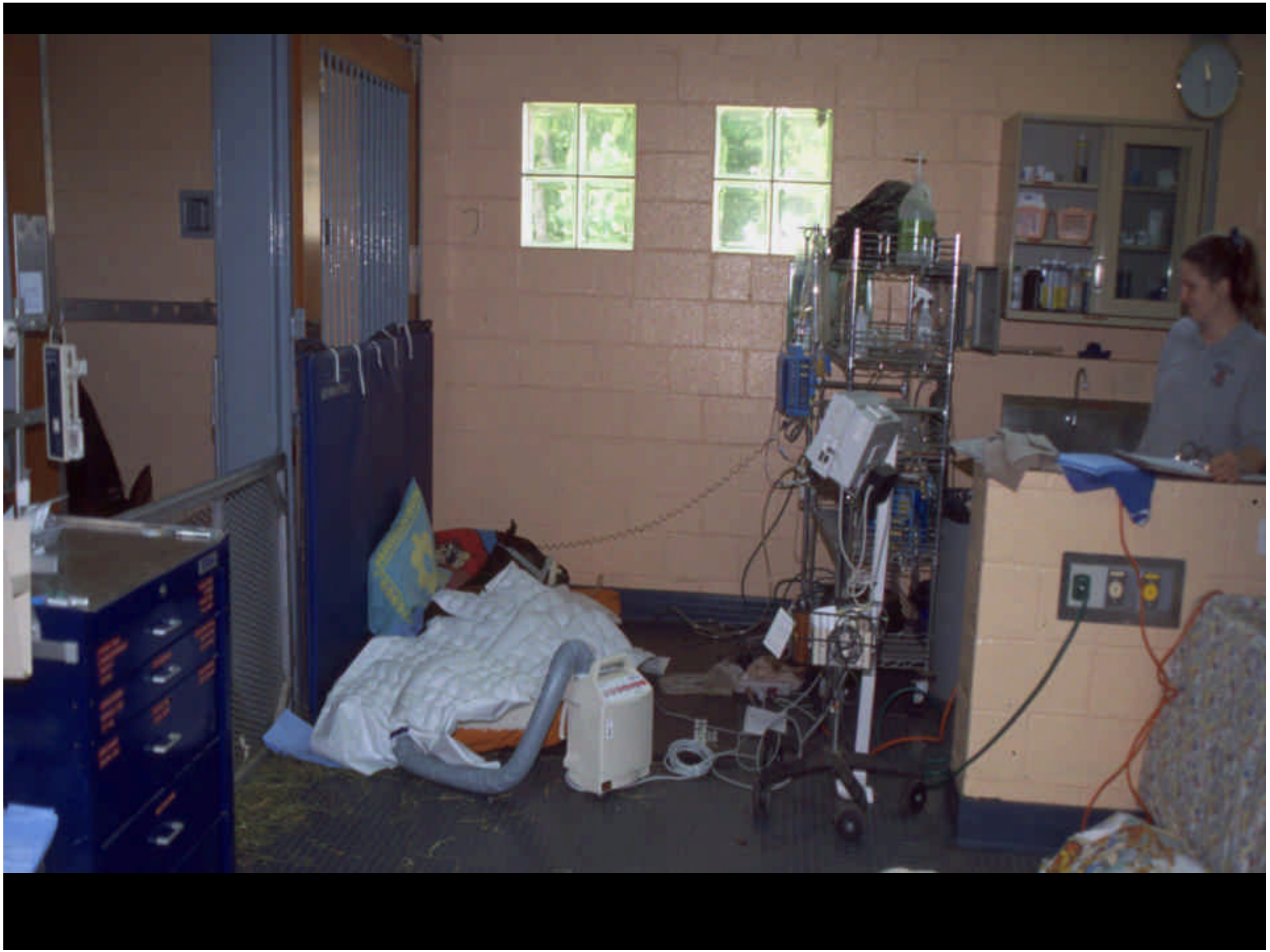
- *Injection, Icterus*
- *Oral petechiae*
- *Cardiovascular*
  - *Ice cold hooves*
  - *Cold lower legs*
  - *Cool nose, ears and upper legs*
  - *Weak pulses, poor arterial fill, poor arterial tone*
  - *Relative bradycardia (90-96)*
  - *Evidence of recent urination*

Chester
Essential Organ Function

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

Chester
Initial Supportive Therapy

- *Intranasal 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 ≈ 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

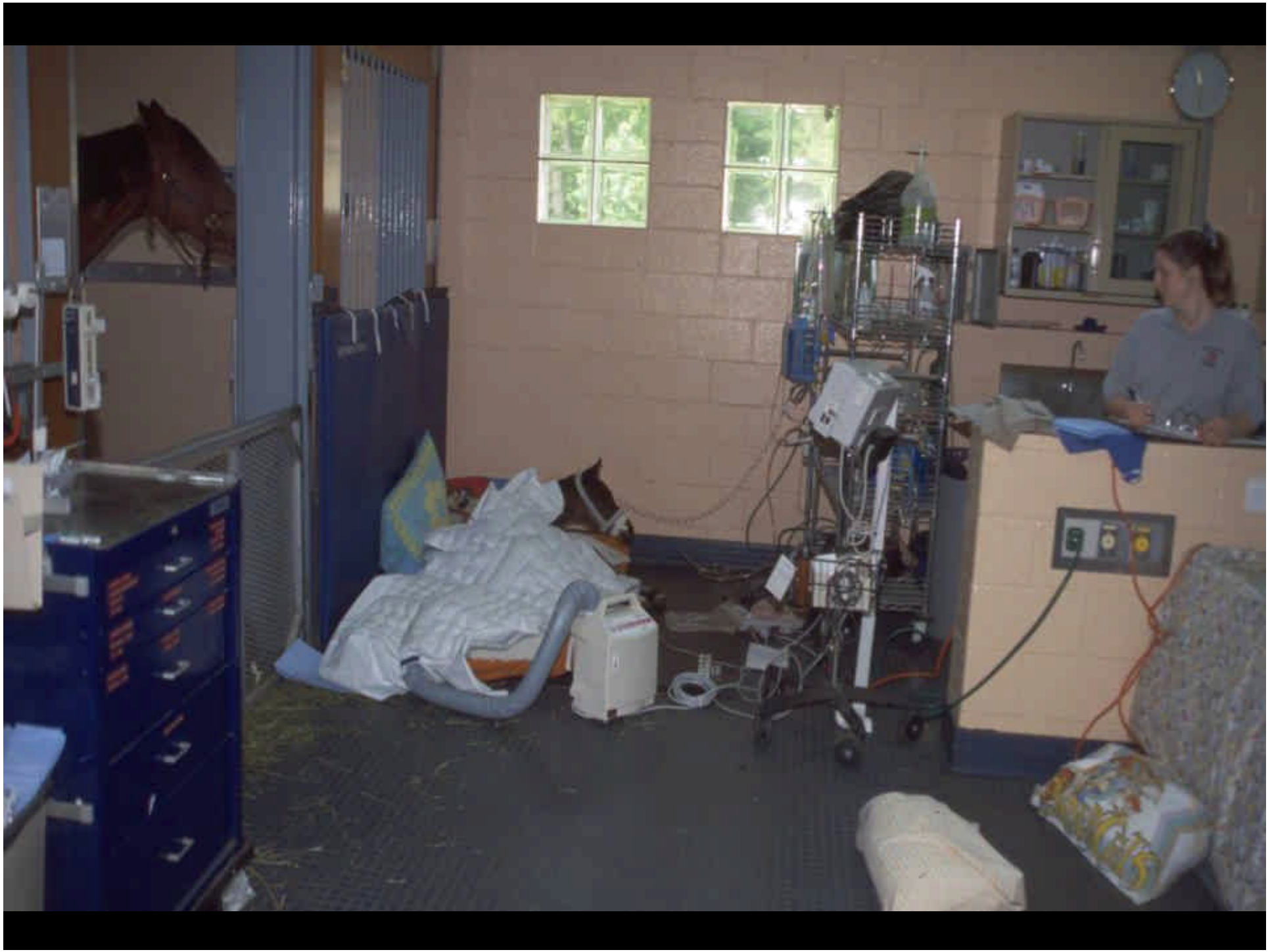
- Insuring tissue oxygen delivery
  - Maximizing pulmonary loading of hemoglobin
  - Guaranteeing sufficient blood oxygen content
  - Returning perfusion to normal
- Intranasal oxygen insufflation
  - Immediately, before ABG results
  - ABG
- Blood oxygen carrying capacity
  - Birth changes in PCV
  - 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 vaspolpegia
- 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 O_2 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*
    - *Pco2 = 85 torr, pH = 7.276*
  - *Second dose of caffeine*
- *12 hours*
  - *Pco2 = 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

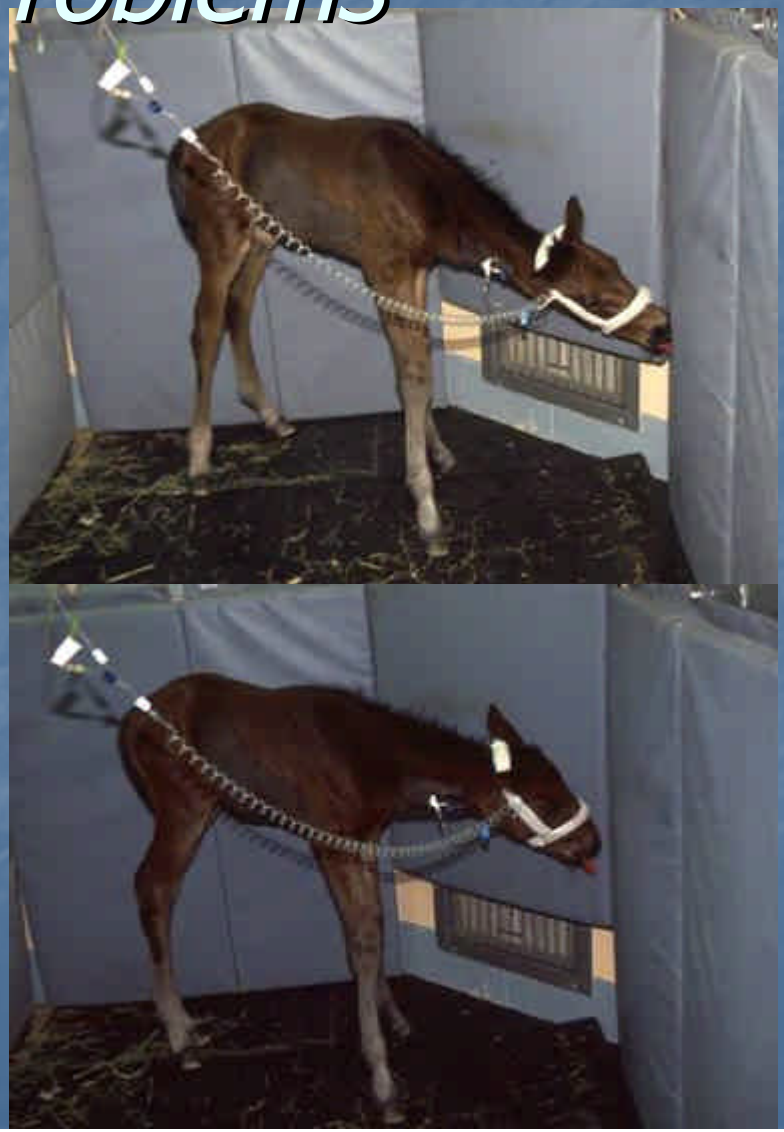
- *12 hours*
  - *Periods of somnolence and nonresponsiveness*
  - *Apneic respiratory pattern with cluster breathing*
  - *Facial nerve paresis*
    - *right ear lower and slower to respond*
    - *ears are not synchronized*
- *21 hours*
  - *Seizure-like activity*
    - *Opisthotonus*
    - *Tonic/Clonic marching activity*
  - *Treated with intravenous phenobarbital*

Chester
Neonatal Encephalopathy

- *27 hours*
  - *Chester was again hypercapnic*
    - *Pco2 = 74.7 torr, pH = 7.313*
  - *Treated with a third dose of caffeine*
- *29 hours*
  - *Pco2 = 84.6 torr, pH 7.269*
  - *Placed on a volume cycled positive pressure partial ventilatory support*
    - *SIMV/PS*
    - *Easily ventilated*
    - *pH = 7.401, Pco2 = 53.8 torr, Po2 = 95.7 torr,*
    - *HCO3 = 33meq/l, BE +8.3, SAT 99.0%, 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_{xNa}
  - 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*
  - *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*

Wishful
Initial Laboratory Analysis

Value	Adm	1 hour
pH	7.251	7.305
Pco2	47.3	50.2
Po2	64.0	285
HCO3	20.9	25.1
BE	- 5.8	-0.9
SAT	94.5	100
Cont	17.9	15.9
Lactate	14.9	10.0
	RA	10 lpm

Wishful
Initial Laboratory Analysis

Value	Adm
Na	115
K	7.33
Cl	72
Cr	28
AST	657
CPK	3012

Wishful

- 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
    - $OG = Osm_m - Osm_c$
    - $Osm_m = (2X\ [Na]) + (glucose/18) + (BUN/2.8)$
    - 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

Wishful
Initial Laboratory Analysis

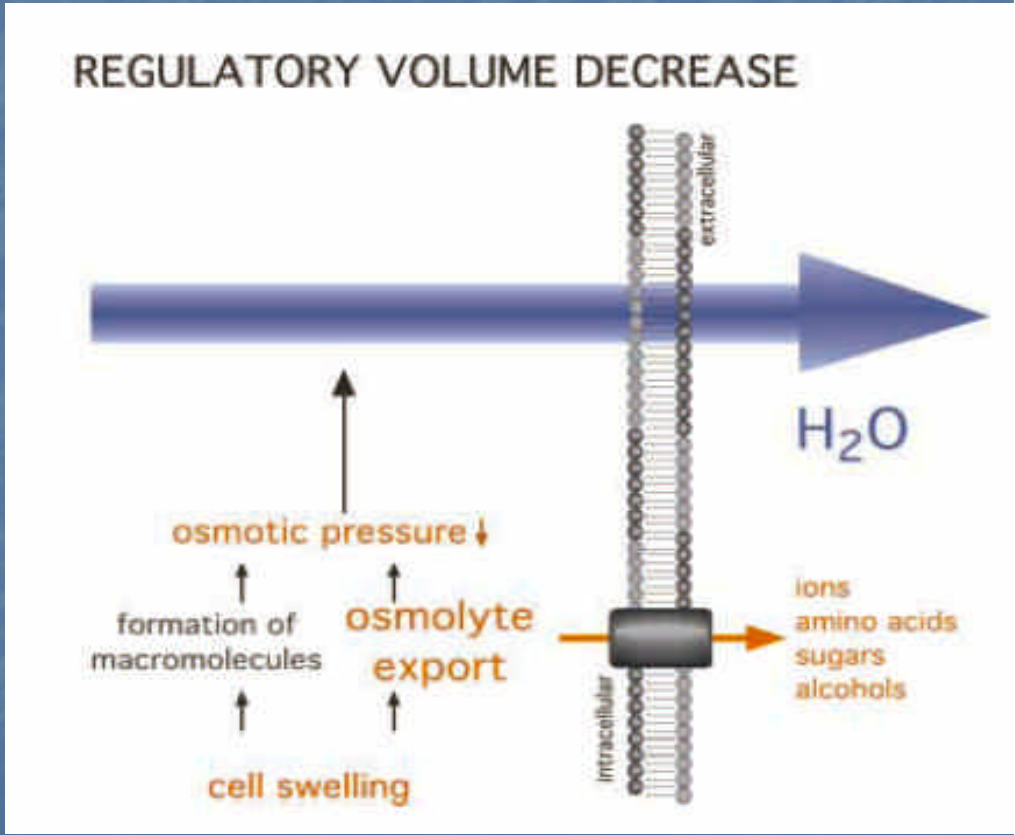
Value	Adm
Na	115
K	7.33
Cl	72
Cr	28
AST	657
CPK	3012
Osm_m	312
Osm_c	240
Osm Gap	72

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
- utcome
  - 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 entery
    - Insulin/dextrose
    - B_2 adrenergic
      - Albuterol
    - Na HCO_3 – not recommended
  - Enhance excretion
    - Osmotic diuresis
      - Support
    - Furosemide
    - GI t

Wishful Outcome

Value	Adm	HD 2	HD 3
Na	115	126	132
K	7.33	4.26	4.76
Cl	72	87	96
Cr	28	9.24	1.74
AST	657	781	534
CPK	3012	625	74
Osm_m	312	312	295
Osm_c	240	270	275
Osm Gap	72	43	20

Wishful Outcome

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