

# Neonatal Diseases of Foals



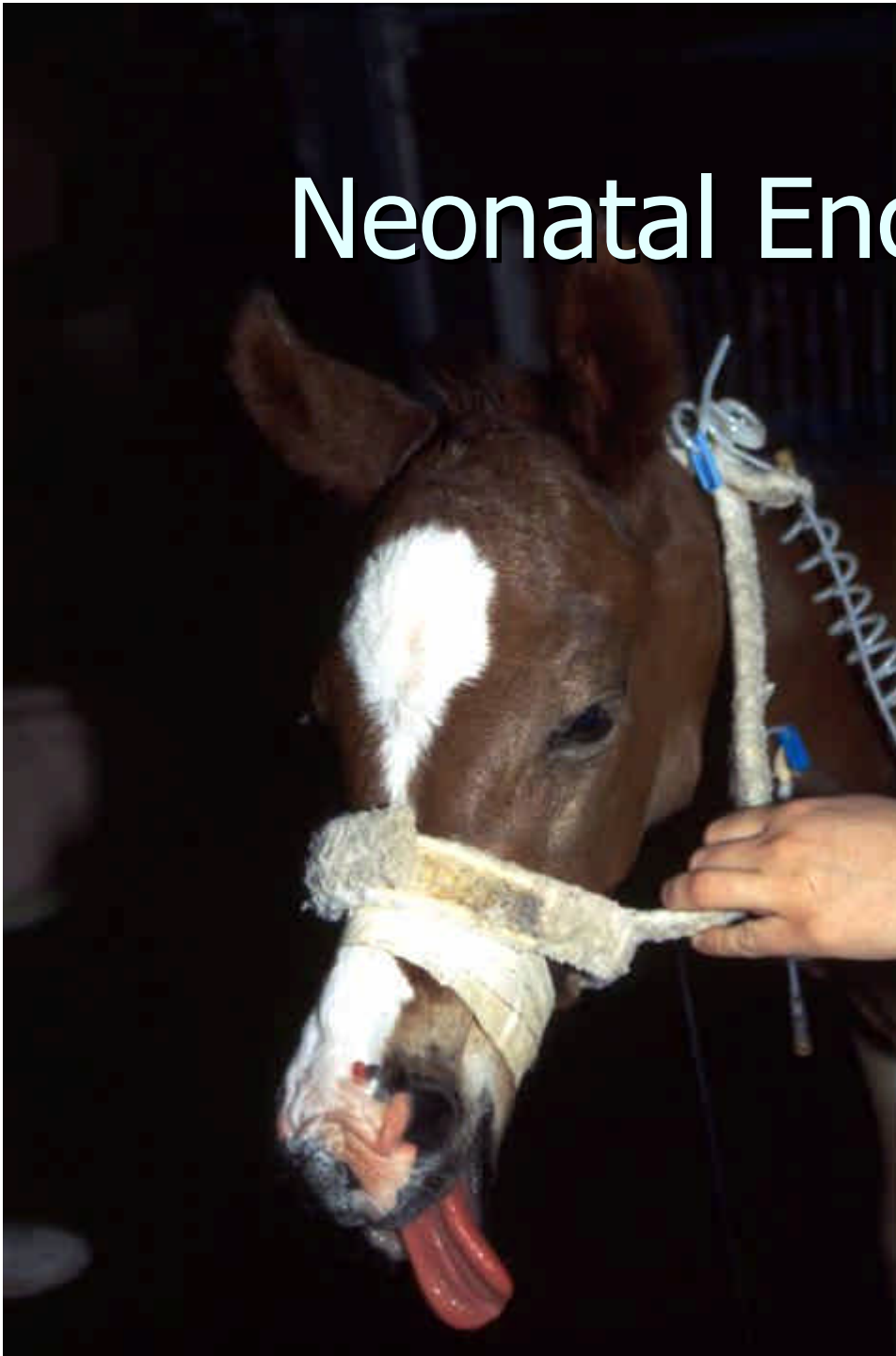
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# Neonatal Diseases

- Neonatal Encephalopathy
- Neonatal Nephropathy
- Neonatal Gastroenteropathy
- Sepsis/septic shock
- Prematurity
- IUGR



# Neonatal Encephalopathy



- Neonatal Maladjustment Syndrome (NMS)
- “Dummy foal”
- Barkers
- Hypoxic Ischemic Encephalopathy (HIE)



# Neonatal Encephalopathy

- Changes in behavior
- Changes in responsiveness
- Changes in muscle tone
- Brain stem damage
- Seizure-like behavior



# Neonatal Encephalopathy

- Changes in behavior
  - Loss of suckle response
  - Loss of tongue curl
  - Loss of tongue coordination
  - Disorientation especially relative to the udder
  - Aimless wandering
  - Blindness
  - Loss of affinity for the dam
  - Abnormal vocalization ("barker")

# Changes in behavior



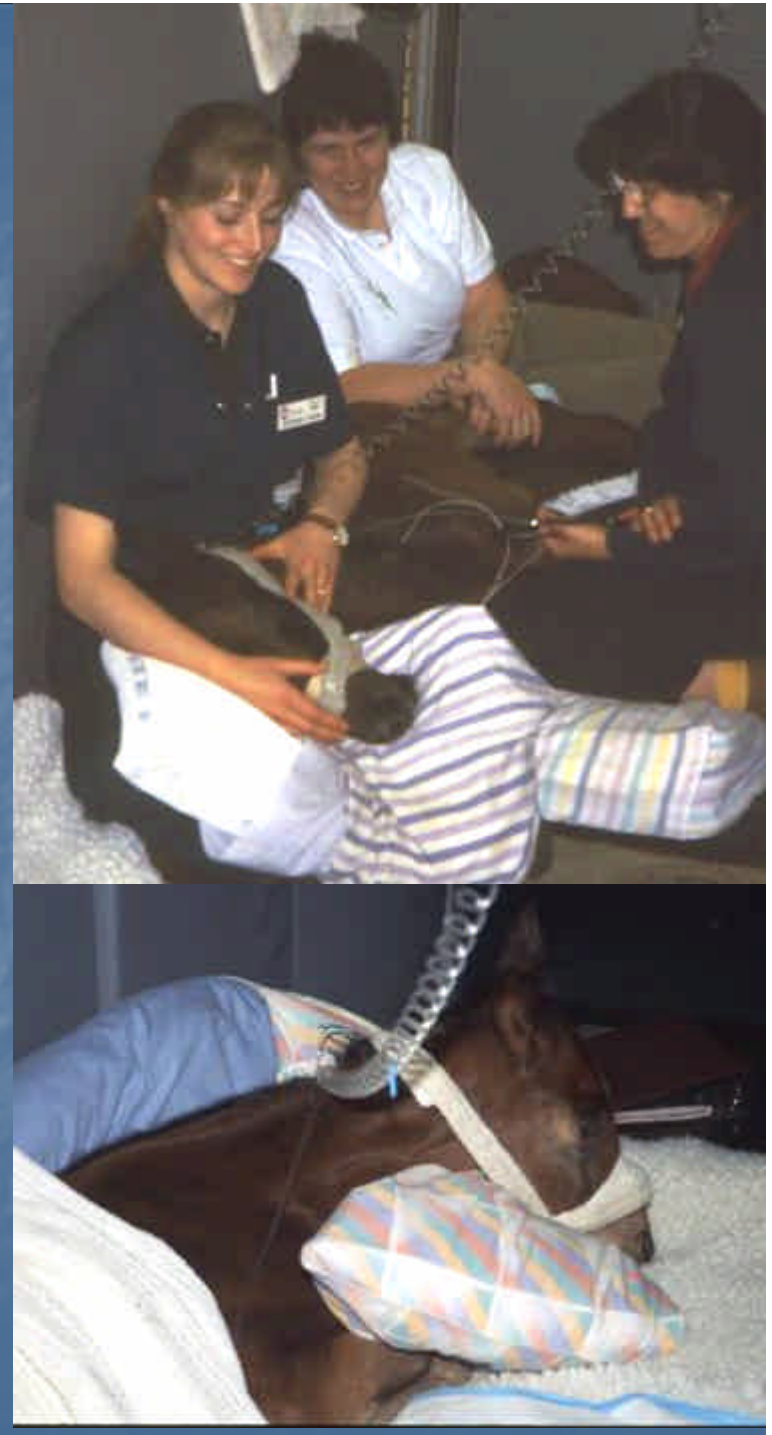


# Changes in behavior



# Neonatal Encephalopathy

- Changes in responsiveness
  - Hyperesthesia
  - Hyperresponsiveness
  - Hyperexcitability
  - Hyporesponsiveness
  - Periods of somnolence
  - Unresponsiveness
- Changes in muscle tone
  - Extensor tonus
  - Hypotonia
  - Neurogenic myotonia
  - Failure to protract legs





# Neonatal Encephalopathy

## Signs of Brain Stem Damage

- Changes in respiratory patterns
  - Tachypnea, apneusis
  - Apnea, cluster breathing
- Central hypercapnia
- Loss of thermoregulation
- Weakness
- Hypotension
- Vestibular signs
- Facial nerve paresis
- Loss of consciousness



# Neonatal Encephalopathy

Seizure-like behavior





# Neonatal Nephropathy

- Spectrum of disease
  - Incomplete fetal transition
  - Water/Na retention
  - Mild tubular dysfunction
  - Acute tubular necrosis
- Usually transient
- Occasionally
  - Irreversible acute damage
  - Chronic renal disease





# Neonatal Nephropathy

- Decreased GFR
  - Slow decrease birth Cr
  - Decrease  $Cl_{cr}$
- Decreased  $H_2O$  excretion
  - Edema formation
  - Weight gain
  - Slow response to fluid challenges



# Neonatal Nephropathy

- Abnormal electrolyte handling
  - Na loss
  - Abnormal Na balance
- Abnormal excretion of drugs
  - High amikacin trough levels
- Oliguria/Anuria



# Neonatal Gastroenteropathy

- Wide spectrum of disease
- Mild indigestion – functional deficits
  - Dysmotility
  - Enema dependence
- Moderate disease
  - Colic
  - Abdominal distension/ileus
  - Diapedesis of blood into the lumen
  - Mucosal edema





# Neonatal Gastroenteropathy

- Severe
  - Epithelial necrosis
  - Intussusceptions
  - Structures
  - Hemorrhagic gastritis or enteritis/colitis
  - Pneumatosis intestinalis
- Predisposition to sepsis
  - Translocation of bacteria

# Neonatal Gastroenteropathy



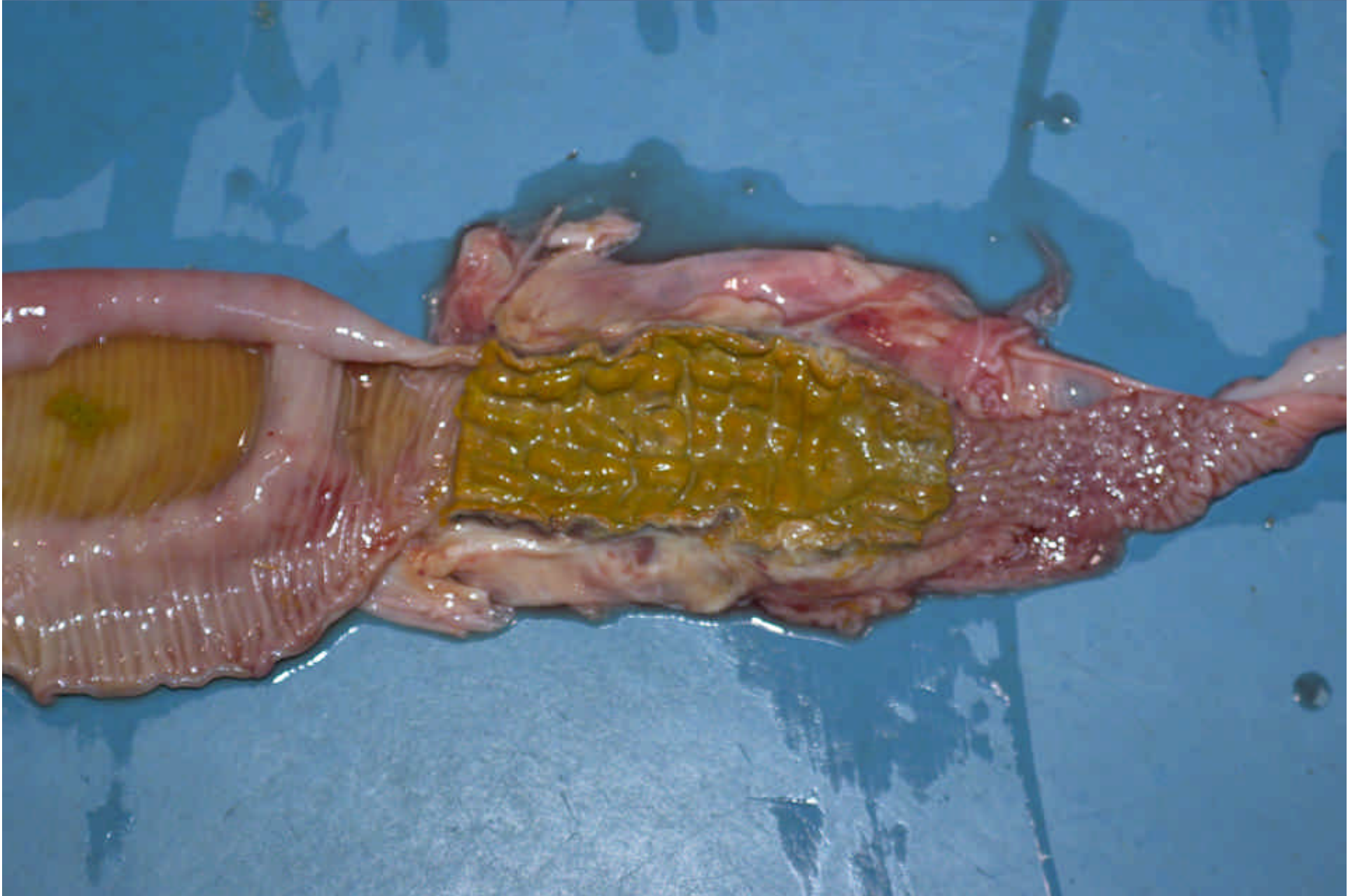


# Neonatal Gastroenteropathy





# Neonatal Gastroenteropathy



# Neonatal Multisystem Maladaptation Syndrome

- Neonatal Encephalopathy
- Neonatal Nephropathy
- Neonatal Gastroenteropathy
- Neonatal Metabolic Maladaptation
- Neonatal Cardiovascular Maladaptation
- Neonatal Autonomic Maladaptation
- Neonatal Endocrine Maladaptation





# Neonatal Multisystem Maladaptation Syndrome Prognosis

- 85-87% of foals with uncomplicated courses recover completely







Prematurity  
Dysmaturity  
IUGR

# Terminology



- Prematurity
  - < 320 days
- Dysmaturity
  - > 320 days
  - Looks premature
- Prematurity
  - Not by calendar
  - Relative to mare's normal gestation
  - Clinical appearance
- IUGR
  - Clinical characteristics
  - May accompany prematurity
  - Precocious development







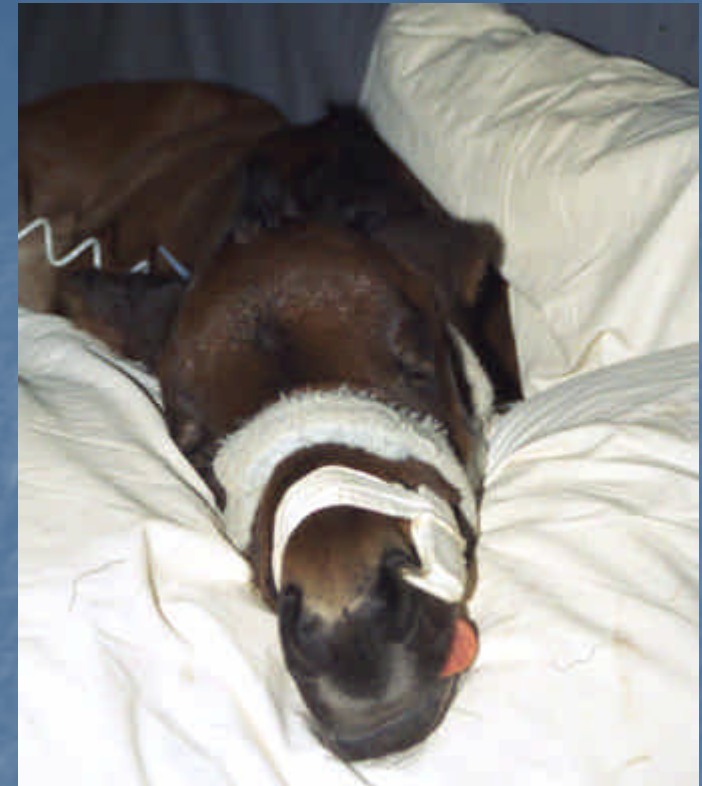






# Sepsis/Infections

- Definitions
  - Clinical diagnosis
  - 1991, 2001 Consensus Conferences
- Infection
  - Bacteremia
  - Pulmonary
  - GI
  - Umbilical
- SIRS
- Sepsis
- Severe Sepsis
- Septic shock







## NEONATAL DISEASES OF FOALS

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Disruption of the intrauterine environment will not only result in fetal distress, but may be a major cause of neonatal diseases recognized in foals. Conditions of neonatal foals which may be directly associated with placentitis include prematurity, IURG and precocious maturation. Other conditions that may be associated with placentitis include the complex of Neonatal Encephalopathy, Neonatal Nephropathy, Neonatal Gastroenteropathy and maladaptation of other systems.

Prematurity in the foal has been defined as birth before 320 days gestation. The term 'dysmature' has been used to describe foals with physical characteristics of prematurity in a foal born after a gestation more than 320 days. These definitions are problematic because of the wide variation in normal gestational length in mares. This has led to confusion in diagnosing prematurity, dysmaturity and IUGR. I feel it is more helpful to define prematurity based on the mare's normal gestational length, use IUGR for foals small for gestational age (which are not constitutionally small) and avoid using the term dysmaturity.

There is a neonatal multisystem maladaptation syndrome which includes neurologic, renal, gastrointestinal and other system malfunctions which may be secondary to placental disease. The most prominent signs are neurologic abnormalities traditionally referred to as Neonatal Maladjustment Syndrome (NMS). Research findings in other species early in the 1990's lead many equine neonatologists to speculate about a hypoxic ischemic or asphyxial origin for this syndrome. The term Hypoxic Ischemic Encephalopathy (HIE) largely replaced NMS. It is clear, however, that despite attractive experimental models showing many similarities, often this disease syndrome occurs in the absence of a detectable hypoxic ischemic insult. Because of this I prefer to use the terms, such as Neonatal Encephalopathy, that do not specify an etiology or pathogenesis but only the organ dysfunction and age group. Most recently there has been speculation that inflammatory mediators secondary to placentitis may (possibly by initiating a hypoxic ischemic insult) be responsible for the multiorgan dysfunction.

Foals with Neonatal Encephalopathy (NE) may show changes in responsiveness, muscle tone, behavior, evidence of brain stem damage or seizure-like behavior. Changes in responsiveness include hyperesthesia, hyperresponsiveness, hyperexcitability, hyporesponsiveness, periods of somnolence or unresponsiveness. Often the foals go through a period of increased responsiveness followed by a period of decreased responsiveness. Changes in muscle tone include increased extensor tonus, hypotonia and other less common changes such as neurogenic myotonia or failure to protract front legs. Changes in behavior are very common and include loss of suckle response, loss of tongue curl, loss of tongue coordination, disorientation especially relative to the udder, aimless

wandering, loss of affinity for the dam and abnormal vocalization. Foal with NE commonly have changes in respiratory patterns with central tachypnea, apneusis, apnea, cluster breathing, ataxic breathing, Cheyne-Stokes breathing or central hypercapnia. Other signs of brain stem damage include loss of thermoregulatory control, generalized weakness, anisocoria, pupillary dilation, pinpoint pupils, central hypotension, decreased responsiveness, difficult to arouse, loss of consciousness, vestibular signs (circling, head tilt), facial nerve paresis and a variety of other signs. Foals with NE have a wide variety of signs and degrees of severity. More than 90% of affected foals are normal within 10 days.

Foals may also develop renal maladaptation referred to as Neonatal Nephropathy (NN). There is a wide spectrum of disease seen in cases of NN including incomplete transition from fetal renal physiology, water/sodium retention, mild tubular dysfunction (sodium wasting), abnormal excretion of drugs (e.g. high amikacin trough levels), acute tubular necrosis or decreased GFR. Often the signs of dysfunction are subtle and easily overlooked unless anticipated. Although almost always transient, on occasion significant acute damage may lead to chronic renal disease. These foals often have a decreased GFR as reflected by a slow decrease birth Creatinine or decreased creatinine clearance, delayed water excretion with edema formation and weight gain and slow response to fluid challenges.

Neonatal Gastroenteropathy (NG) can be manifested by a wide spectrum of signs ranging from mild indigestion with dysmotility and enema dependence to moderate disease with ileus, diapedesis of blood into the lumen and mucosal edema to severe disease with epithelial necrosis, intussusceptions, structures, hemorrhagic gastritis/enteritis/colitis, and pneumatosis intestinalis. Even mild forms predispose to sepsis and SIRS with increased likelihood of translocation of bacteria. Like NN, often the signs of dysfunction are subtle and easily overlooked unless anticipated. The most common manifestation is dysmotility with meconium retention and lack of fecal passage for days (range 2-30 days). Despite fecal retention, an important aspect is lack of discomfort. Classically, foals with classic motility will not return enema fluid or strain associated with rectal distension.

Often, affected foals have the triad of Neonatal Encephalopathy, Neonatal Nephropathy and Neonatal Gastroenteropathy. Other problems seen include metabolic maladaptation, autonomic failure and other systemic problems. Foals born from an environment of placentitis commonly have a generalized inflammatory response as reflected by systemic and hematologic reactions. The activation of inflammatory and anti-inflammatory cascades may support precocious maturation of many body systems and may, in fact, impart some protection from systemic infections.

Prematurity and IURG are easily confused. Clinically prematurity is marked by low birth weight, small frame, thin, poor muscle development, periarticular laxity and general flexibility. IUGR is marked by apparent cachexia and disproportional growth. Either may be a direct result of placentitis and it is common for both to be present concurrently.