

## Neonatology

# Prematurity, Dysmaturity, Postmaturity

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Prematurity is the result of a preterm birth. Most veterinary texts will define prematurity in the foal by a gestational length of less than 320 days. But in order to truly define a preterm birth, the normal gestational length must be known. Most veterinary texts will say that the average gestational length of a mare is 334 to 340 days. This is true, but it is an average. We never deal with the "average" mare but with an individual mare. Each mare has decided herself what her normal gestational length is, despite what we think. This may vary from 315 days to more than 390 days. Thus a mare who normally carries a foal to 365 days may have a premature foal with a gestational length of 340 days and another which normally carry 315 days may have a term foal at 315 days gestation.

Foals which are born after their expected due date but are small and have the characteristics of being premature are called dysmature. Dysmaturity is commonly thought to be due to placental insufficiency. Dysmature is a poor term because the clinical definition often encompasses 2 etiologic groups: premature foals born to mares with long gestational lengths and small for gestational age foals suffering from intrauterine growth restriction (IUGR) due to placental disease. I rarely use the term dysmaturity, preferring more specific etiologic terms.

Postmaturity should be distinguished from dysmaturity (prematurity or IUGR). A postmature foal is a postterm foal who has adequate axial skeletal size (usually large) for the gestational age but is thin due to emaciated. Postmature foals are basically normal foals retained too long *in utero*. Because of this relative placental insufficiency, they may have hypoxic or inflammatory insults. At least in

theory, the longer they are retained, the more abnormal they become. Their continued skeletal growth may lead to dystocia. A good example of postmaturity is a foal born from a mare with fescue endophyte toxicity.

### **Clinical characteristics of prematurity**

1. Low birth weight.
2. Small frame - may appear thin with poor muscle development.
3. Periarticular laxity.
4. Usually flexor laxity but occasional contracture.
5. Usually hypotonia - occasional hypertonia.
6. High compliance to chest wall - soft ribcage.
7. Low compliance to lungs - stiff lungs - respiratory distress secondary to fatigue.
8. General muscle weakness - delayed time to standing.
9. Short, silky hair coat.
10. Domed forehead.
11. Floppy ears - poor ear cartilage development.
12. Weak suckle.
13. Poor thermoregulation.
14. GI tract dysfunction.
15. Delayed maturation of renal function - low urine output.
16. Entropion with secondary corneal ulcers.
17. Poor glucose regulation.

### **Clinical characteristics of postmaturity**

1. Normal to high birth weight.
2. Large frame but thin with muscle wasting.
3. Often flexor contraction - occasionally flexor laxity.
4. Usually hypertonia - occasional hypotonia.
5. Delayed time to standing because of hyperreactive state and poor postural reflexes.
6. Long hair coat.
7. Fully erupted incisors.
8. Weak suckle.
9. Poor thermoregulation.
10. GI tract dysfunction.
11. Delayed maturation of renal function - low urine output.
12. Poor glucose regulation.

Often hypoxic ischemia insults, fetal inflammatory response syndrome (FIRS) and neonatal sepsis coexist with prematurity/IUGR/postmaturity. It can be

exceedingly difficult to distinguish the signs from these syndromes and determine the relative severity or even presence of each.

## **Causes**

1. Placental problems: twins, placentitis, premature placental separation.
2. Fetal problems: fetal sepsis (Herpes virus, EVA, ehrlichiosis, other bacteria), fetal malformation, hydroallantois, fetal placentitis (FIRS), etc.
3. Maternal problems: severe systemic disease, chronic debilitation, maternal placentitis.
4. Iatrogenic:
  - a. Early induction based on: inaccurate breeding dates, misinterpreting late term colic as ineffective labor.
  - b. Early C-section because of catastrophic medical problem in mare: fatal colic, fracture of major bone, progressive neurologic disease.
5. Idiopathic.

## **Treatment**

Premature deliveries do not just occur. There is always a cause. It is important to try to establish the cause, as often the problem will continue to affect the foal and must be addressed. The same can be said for IUGR and postmature deliveries. When supporting the premature/IUGR/postmature foal, each body system must be examined critically for signs of dysfunction. The intensivist can not focus on one area to the exclusion of others, but must juggle all the problems simultaneously to achieve a successful outcome.

1. *Respiratory system:* Premature lungs fail for a number of reasons. The least common reason is lack of surfactant. Much more important are a compliant chest wall, weak muscles, and stiff lungs resulting in hypoventilation. Also poor control of pulmonary vasculature results in significant mismatching and shunting. Long periods in lateral recumbency also results in atelectasis and

pulmonary edema in ventral regions. Some foals may also have septic or hypoxic lung damage. Failure to make the transition from fetal circulation or reversion to fetal circulation with the persistent pulmonary hypertension is not unusual. All of these can combine to cause respiratory failure. Most premature/IUGR foals require supplemental intranasal oxygen as well as positional support to achieve adequate oxygenation and ventilation. Occasionally they also require more aggressive therapy such as mechanical ventilation and nitric oxide therapy.

2. *Cardiovascular support:* Premature/IUGR foals often have poor or marginal cardiovascular function. This is primarily due to a lack of responsiveness of vessels to pressors. Careful use of fluid support and inotropes and pressors such as dopamine, dobutamine, epinephrine and vasopressin may achieve improvement of cardiovascular function. It should always be remembered that a particular set of blood pressure values is not nearly as important as effective perfusion. The systemic circulation of the fetus is a low resistance system with low blood pressures. During the neonatal period the foal makes a transition to a high resistance/pressure system, but initially the systemic pressures remain low. Despite the low blood pressure, perfusion is normal because of the low resistance. So, mean arterial blood pressures are frequently in the 40's and 50's but does not necessarily indicate a perfusion deficit. Relative changes in blood pressure are important to follow but are not a substitute to evaluation of perfusion.
3. *Renal function:* Premature/IUGR foals often have poor renal function initially. Although foals achieve glomerular filtration rates similar to adult horses within days of birth, the transition from fetal renal function to neonatal renal function is critical. Premature foals appeared to have problems with this transition resulting in poor urine output initially. This may be due to true failure of the transition or due to hypoxic or septic damage during the premature delivery as well as during both prenatal and neonatal periods. The abnormal renal

function is not a reflection of “premature kidneys.” Achieving near normal renal function is important for survival of the neonate. Great care must be taken not to fluid overload these foals. If oliguria or anuria is present, fluid restriction is in order, only using diuretics to adjust unavoidable fluid overload caused by nutritional demands. A bolus dose of furosemide or the use of adrenergic inopressors may reverse the problem but consistent use of diuretics often leads to serious electrolyte imbalances. It is important not to fluid load these foals and then rely on diuretic therapy to rid the body of the iatrogenic fluid overload. This is a formula for physiologic disaster.

4. *CNS function:* The most important steps in supporting central nervous system health are preserving oxygen and nutrient delivery by insuring adequate perfusion. This is done by maintaining intravascular fluid volume and blood perfusion (with appropriate pressor and inotrope therapy). Often these foals have suffered a hypoxic insult during the prenatal, intrapartum, or early neonatal period. This problem is best treated by supportive care. See Neonatal Syndrome notes.
5. *Gastrointestinal System:* The gastrointestinal system may not be ready to function fully. Again this is more commonly caused by a hypoxic or septic insult associated with the prematurity/IUGR, rather than a lack of gastrointestinal tract maturity. Dysmotility is the most common problem although all degrees of severity of necrotizing enterocolitis may be present. Metabolic and cardiovascular stability as well as good respiratory function must be achieved before feeding is attempted. Once feeding is initiated, volumes fed should be slowly increased. During the time between birth and full feeding, parenteral nutritional support is often needed.
6. *Metabolic stability:* Hyperglycemia and hypoglycemia are common during this period. Hyperglycemia is usually associated with stress, high levels of adrenergic hormones, rapid transition to gluconeogenesis and poor insulin

responsiveness. Hypoglycemia may be associated with exhaustion of glycogen stores, failure to develop the ability to perform gluconeogenesis, sepsis, and hypoxic or septic damage. Careful monitoring of blood glucose is necessary and the combination of intravenous glucose and at times insulin supplementation may be required.

7. *Hypothermia:* Premature/IUGR neonates often have difficulty with thermoregulation. Control of environmental temperature and use of methods to warm the neonate are important therapeutic modalities. The use of heat lamps, hot water bottles and warm air blankets can be valuable. The temperature must be monitored since iatrogenic hyperthermia can occur as thermoregulation is not well developed. Hypothermia has been shown to protect against hypoxic ischemic central damage and surface warming can divert limited perfusion resources, so I don't usually warm hypothermic foals until perfusion and oxygen delivery are normal.
8. *Passive transfer of immunoglobulins:* Although colostrum is important for the development of the intestinal tract, sometimes due to gastrointestinal dysfunction and the predisposition for developing necrotizing enterocolitis, colostrum feeding may be contraindicated. Even when colostrum is given, absorption may not be efficient. In these cases plasma transfusions are indicated. Whole plasma is preferred over products consisting of concentrated IgG, which lack other valuable components found in whole plasma.
9. *Prevention of self-trauma:* Traumatic injuries can be a serious problem since these foals often struggle, cannot assume a sternal position and have poorly developed hair coats which don't effectively protect them. Also, marginal perfusion to the skin can result in dermal necrosis not associated with trauma, although trauma will exacerbate the damage. Decubitus ulcers may develop very quickly if proper care is not taken. Protection can be afforded by deeply bedding the neonate or placing him on blankets, a mattress, etc. Also it is

very important to keep the neonate dry and to dry urine soaked areas completely. Keeping the foal dry is the key to avoiding development of decubitus ulcers.

10. *Support for musculoskeletal problems:* Premature foals generally have significant flexor tendon laxity and poor muscle tone. They benefit from physical therapy in the form of standing and exercise. Care must be taken not to allow them to traumatize their fetlocks if they are lax enough that the caudal aspect touches the ground. Bandaging should be avoided since this will exacerbate the laxity. It should also be remembered that these foals often lack ossification of the carpal and tarsal bones. This predisposes them to crushing injuries if they support their weight in unusual positions. Ossification is stimulated by weight bearing, but as the foal fatigues and stands in abnormal positions they will do damage. So exercise needs be controlled and supervised.
11. *Secondary infections:* These foals are very susceptible to sepsis. Septic shock may occur. Umbilical infections may occur. Pneumonia is also a common secondary sequela.
12. Frequent reexaminations and constant monitoring are necessary to ensure that new problems are caught early and treated appropriately.

## **Complications**

Complications include various infections such as pneumonia, enteritis, osteomyelitis, umbilical infections, corneal ulcers, etc. These foals are also susceptible to fungal infections, which most commonly will be seen first as an oral *Candida* infection (thrush). Self-trauma may also result in corneal ulcers, head and limb injuries, and decubitus ulcers. Limb deformities may occur associated with flexor abnormalities, crushing injuries because of incomplete

ossification of cuboidal bones, and injuries caused by periarticular joint laxity or sepsis. GI problems are very common including colic, indigestion, ileus, and enteritis. Pneumonia is a common sequela of poor lung function, atelectasis, chronic recumbency, and aspiration secondary to poorly coordinated swallowing reflexes and weakness. Over zealous feeding of these foals by naive caregivers can easily result in serious aspiration pneumonia.

## **Prognosis**

With attention to detail and intensive care as needed and in the absence of uncontrolled sepsis approximately 80-85% of these foals will not only survive, but will have a good chance to become a productive athlete. Some will never reach their full stature and are not likely to become successful athletes.