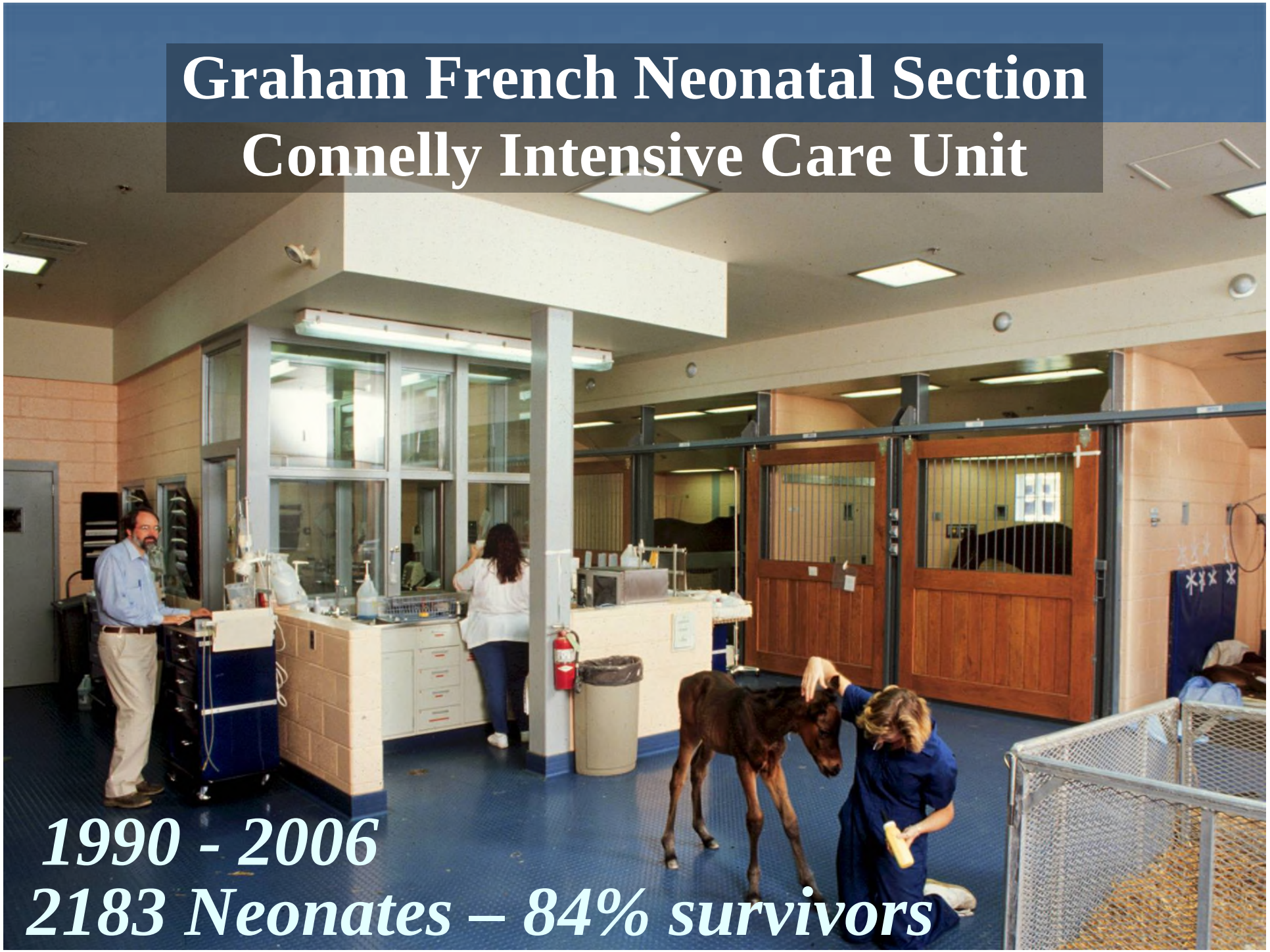
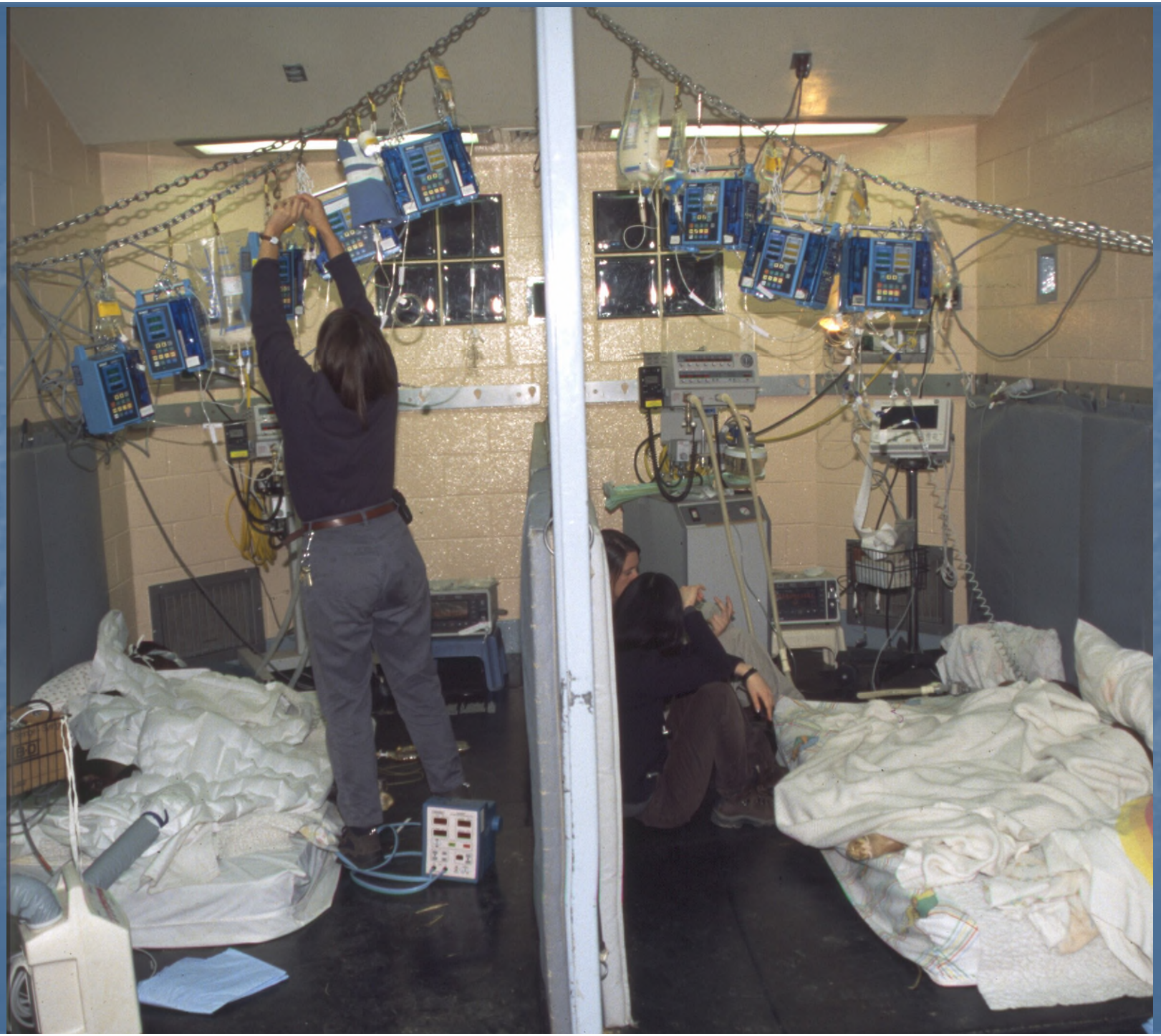
HIGH RISK PREGNANT MARE BIRTH RESUSCITATION

Jonathan Palmer, VMD, DACVIM
Director of Neonatal/Perinatal Programs
Graham French Neonatal Section
Connelly Intensive Care Unit
New Bolton Center, University of Pennsylvania


Graham French Neonatal Section
Connelly Intensive Care Unit
1990 - 2006
2183 Neonates – 84% survivors



High Risk Pregnancy

- History of previous problems
- Development of new problems


Perinatology
• What is the threat to the fetus?
• How can the threat be eliminated?

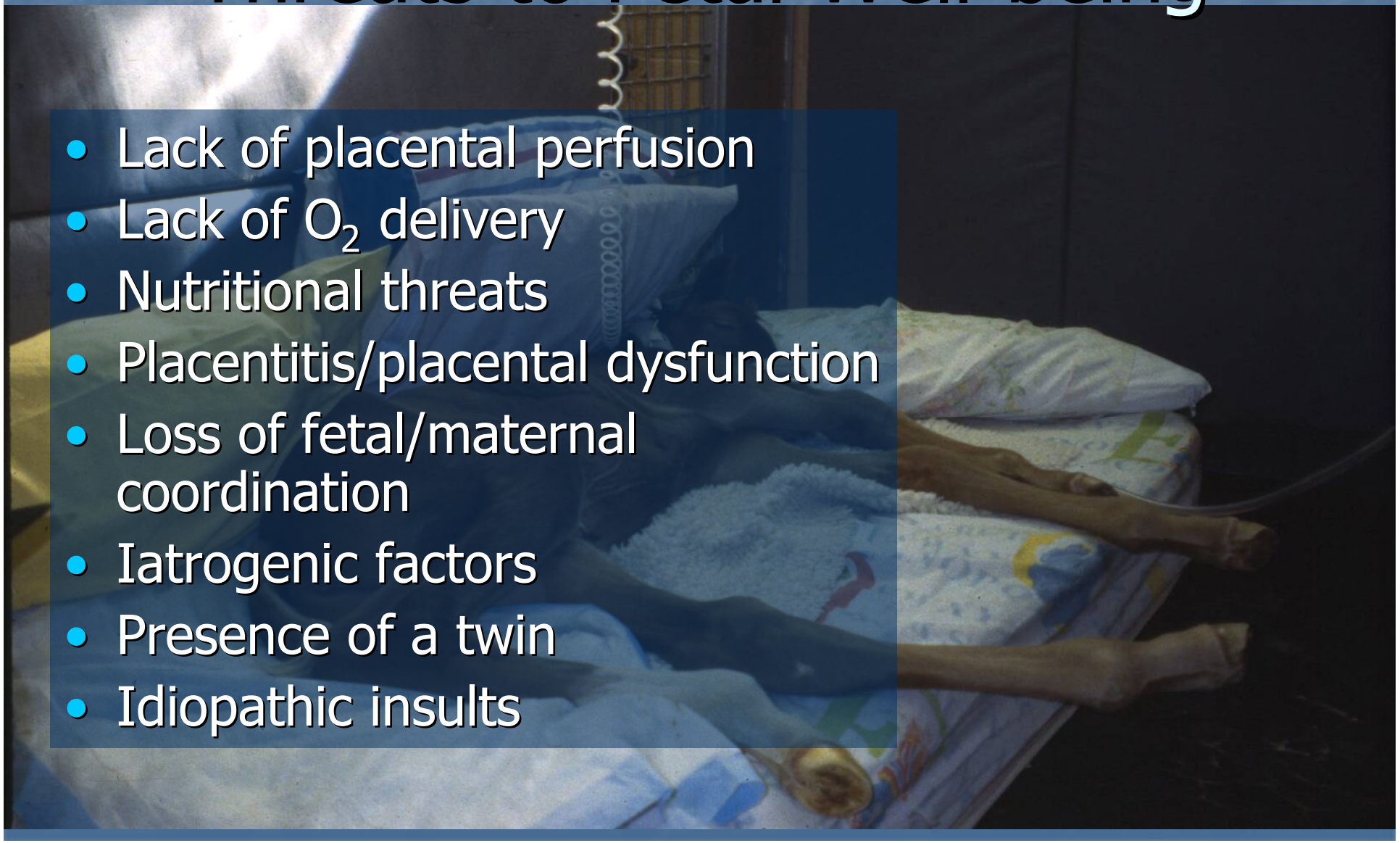
Fetal Resuscitation

- Identify the fetal problem
- Direct therapy at the problem's source
- Support the fetus – fetal resuscitation

Fetal Environment

- Fetus dependent on mare
  - Everything comes from mare
  - No communication of changing needs
- Insufficient resources delivered
  - Survival depends on redistribution of limited resources
    - Sophisticated compensatory responses
    - Response to challenges
- Fetus approaching term
  - Metabolic demands at limit of placenta's ability
  - Any disruption - devastating results

High Risk Pregnancy
Threats to Fetal Well-being



- Lack of placental perfusion
- Lack of O_2 delivery
- Nutritional threats
- Placentitis/placental dysfunction
- Loss of fetal/maternal coordination
- Iatrogenic factors
- Presence of a twin
- Idiopathic insults

Threats to Fetal Well-being
Lack of Placental Perfusion

- Late term fetus
  - High oxygen demand
  - Must receive constant perfusion
  - Margin of safety in late pregnancy small
- Maternal compromise
  - Dehydration/Shock
  - Decreased perfusion for any reason
- Placental response limited
- Compromised placental circulation

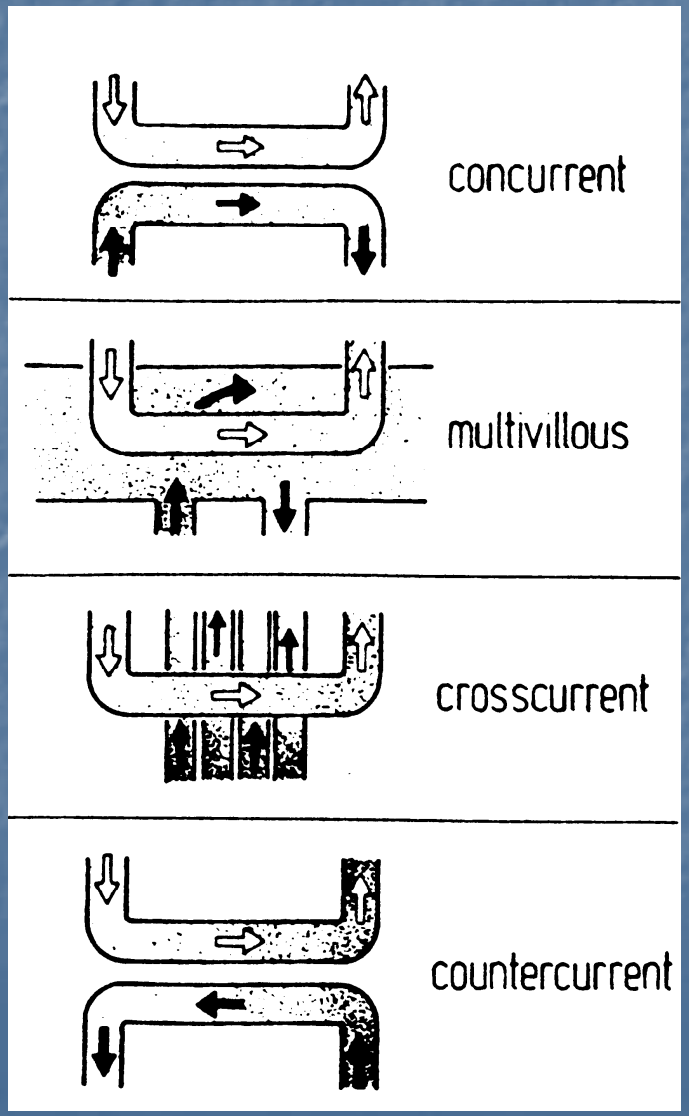
Fetal Resuscitation
Maintenance of Placental Perfusion

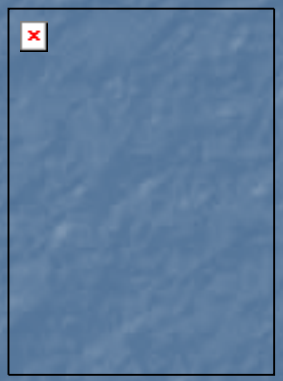
- Aggressively treat hypovolemia in dam
- Aggressively treat hypotension in the dam
- Avoid anesthesia in late term mares

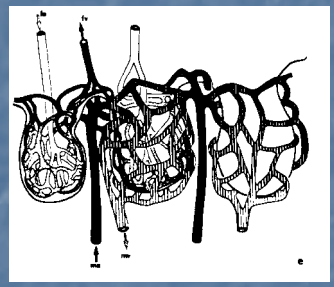
Threats to Fetal Well-being Lack of O_2 Delivery

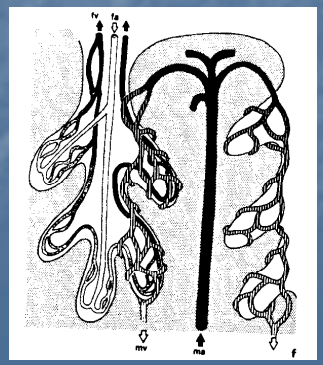
- Maternal threats
  - Maternal anemia
  - Maternal hypoxemia
  - Maternal decrease in perfusion
- Fetal response
  - Unique aspect of placentation
  - Placental oxygen transport mechanisms
  - Fetal physiologic adjustments

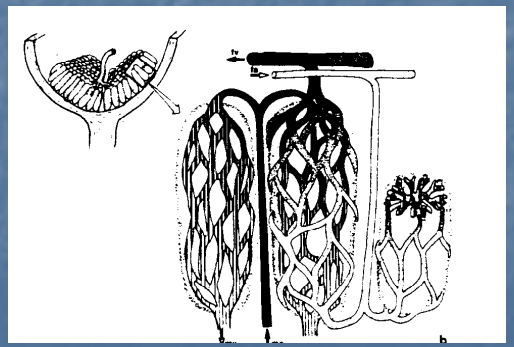
Placental Circulation



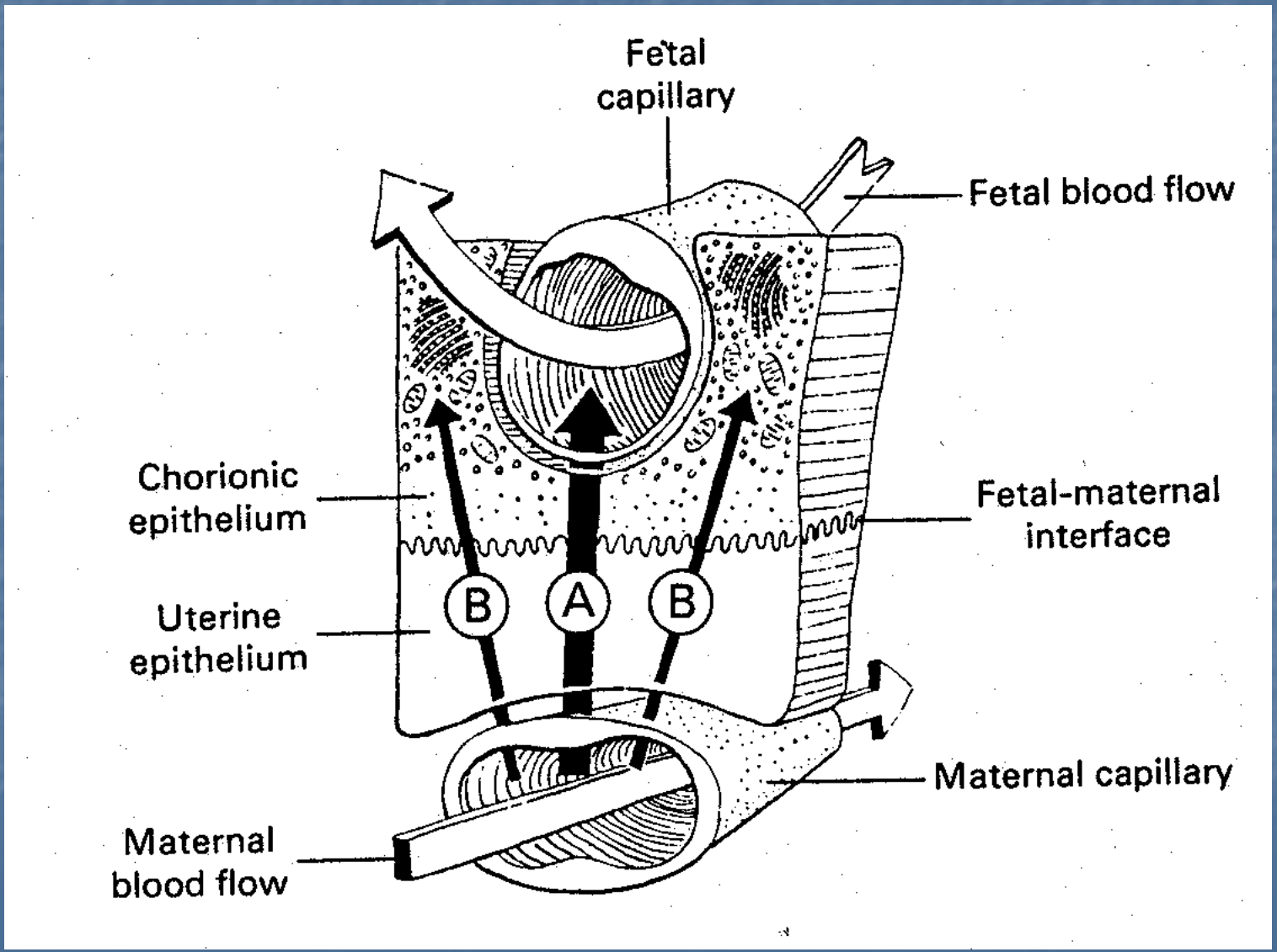








Countercurrent Circulation



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Effect of Maternal Oxygen Therapy

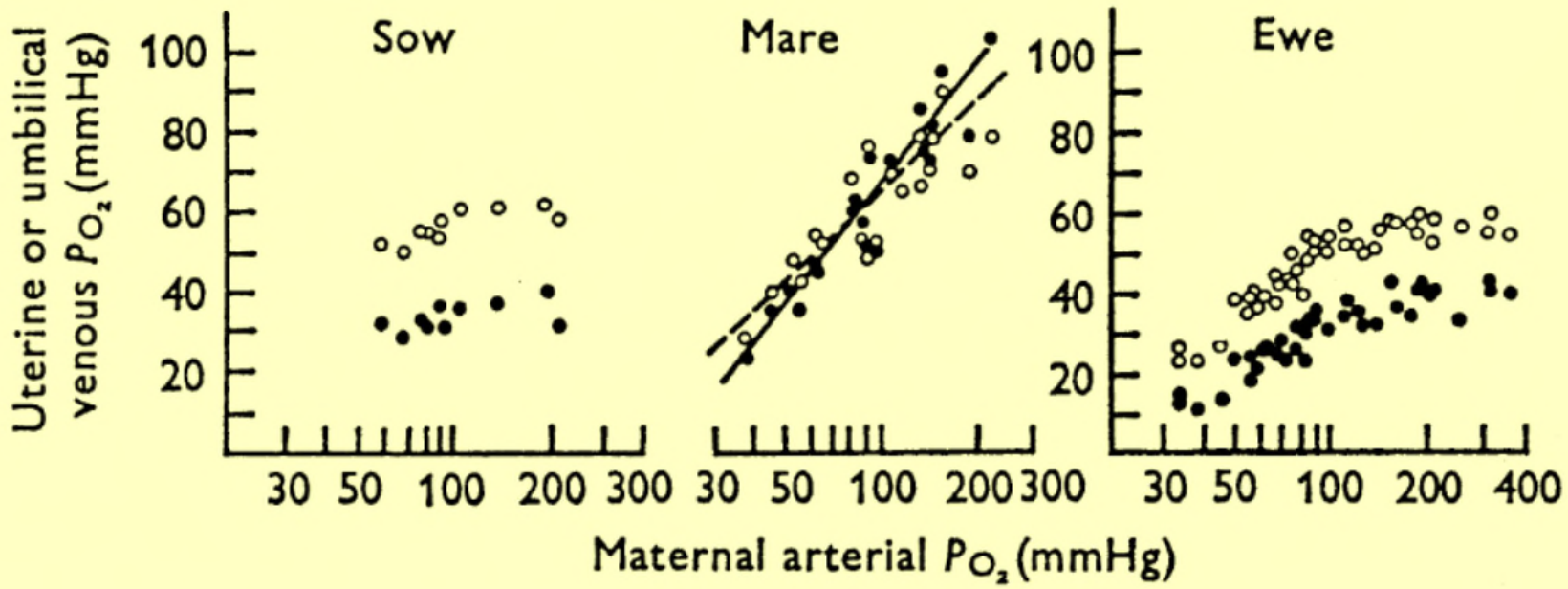
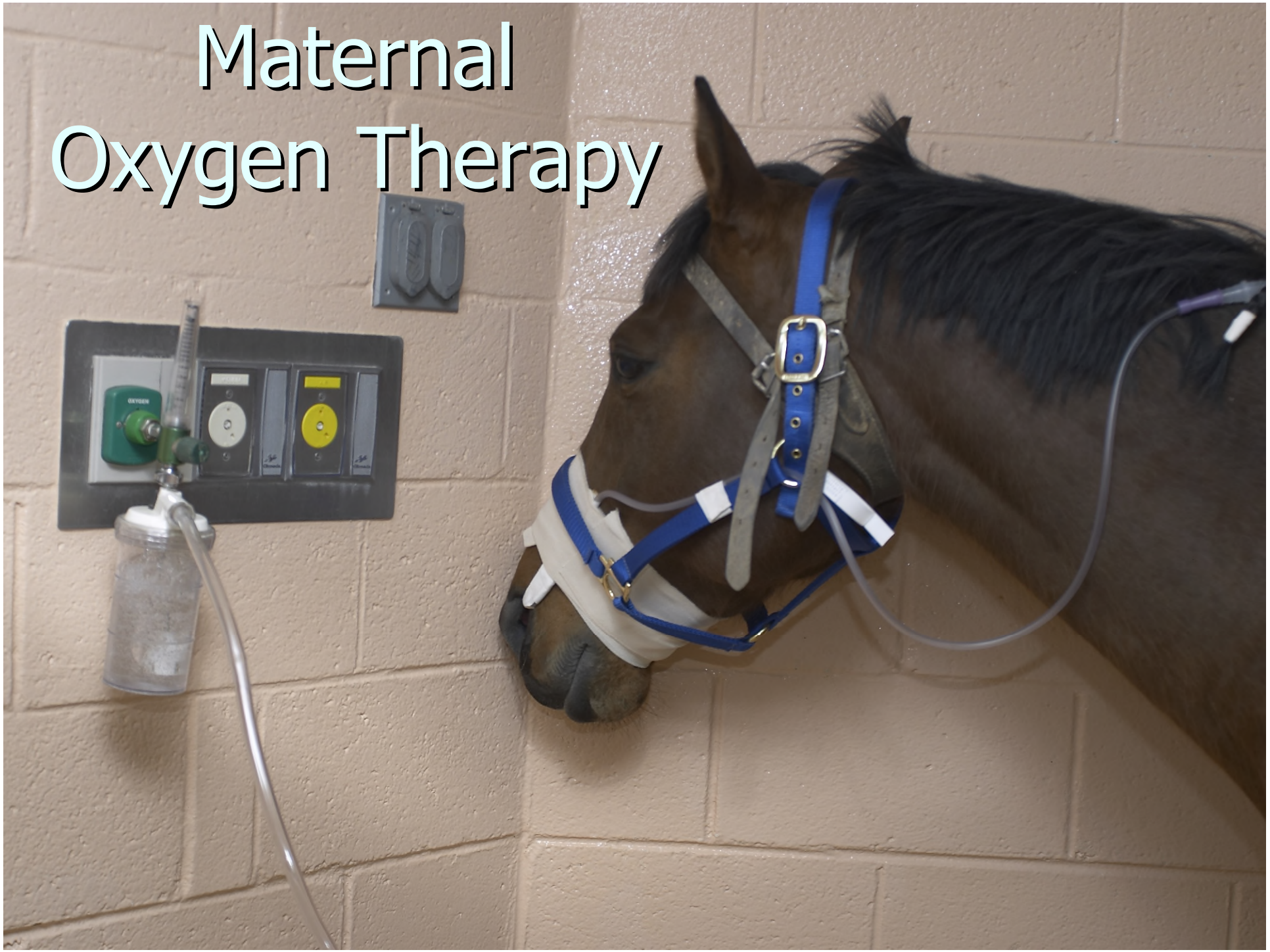


Fig. 4. The relationship between P_{O_2} in maternal arterial blood (log scale) and that in the uterine vein (○) and umbilical vein (●) in seven ewes and seven mares (data from Comline & Silver 1970*b*), and in five sows.

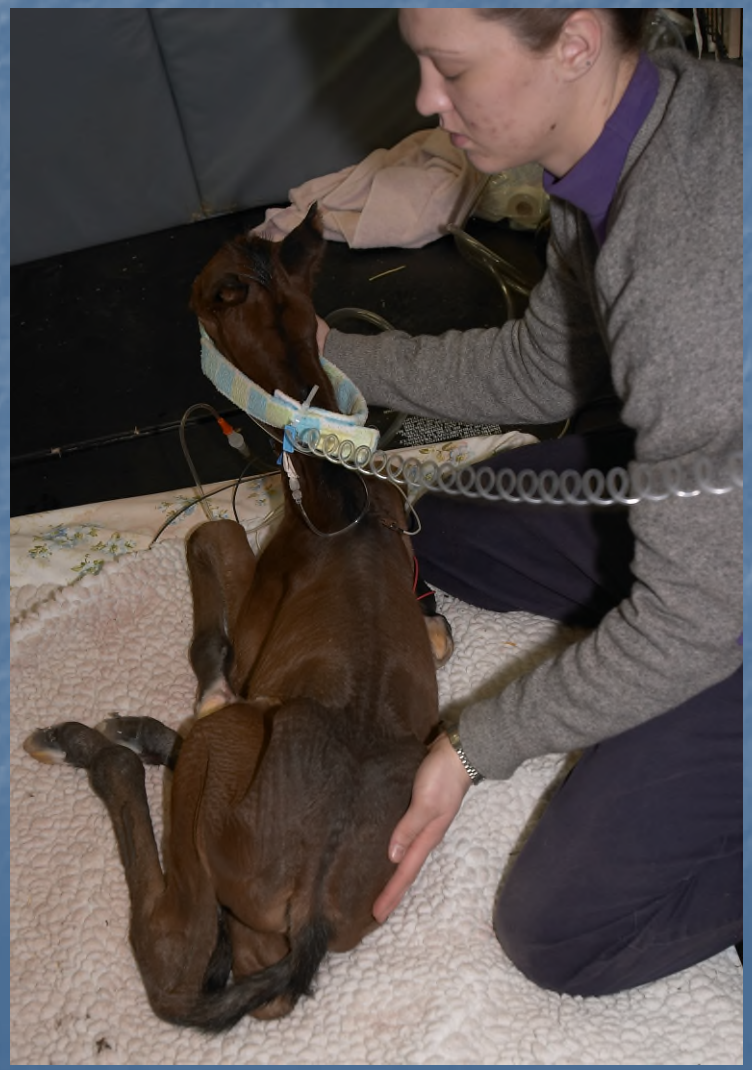
Maternal Oxygen Therapy



Threats to Fetal Well-being
Nutritional Threats

- Chronic malnutrition of the dam
- Placental Glucose Utilization
  - Actively metabolic tissue
  - High glucose utilization - placentitis
  - More glucose delivered from fetus
  - Negative net glucose transport to fetus
- IUGR

IUGR
Intrauterine Growth Restriction



Threats to Fetal Well-being Nutritional Threat of Acute Fasting

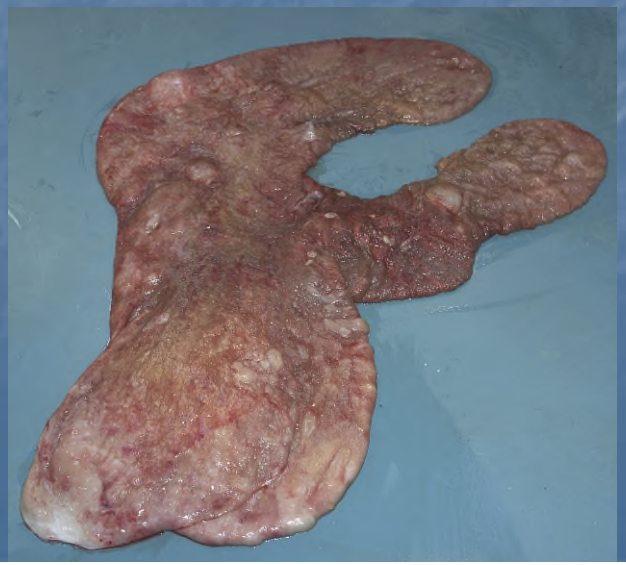
- Fasting the mare for 30-48 hr
  - Decreased glucose delivery
  - Rise in plasma FFA
  - Increased PG's in uterine and fetal tissues
- Increased risk of preterm delivery
  - Within one week of ending the fast
    - Associated with myometrial sensitivity to hormones
- Prevent by intravenous dextrose infusion

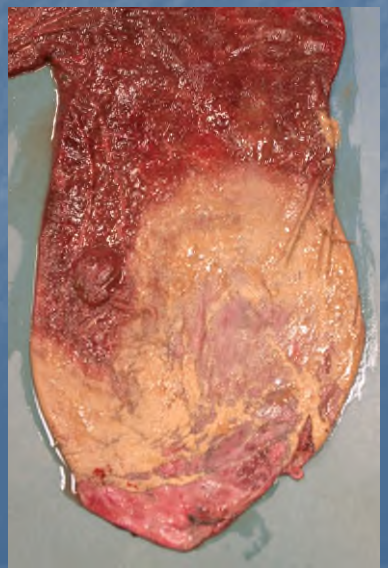
Nutritional Threats
Fetal Resuscitation

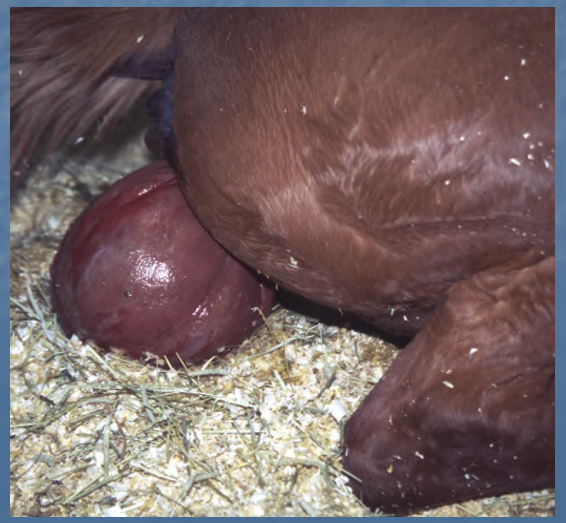
- Support the mare's nutritional needs
- Avoid acute total fasting
- Consider flunixin meglumine therapy

Threats to Fetal Well-being Placentitis

- Percentage of abnormal placenta
  - Not a predictor of fetal outcome
- Presence of abnormal placental tissue
  - Is enough to cause serious problems

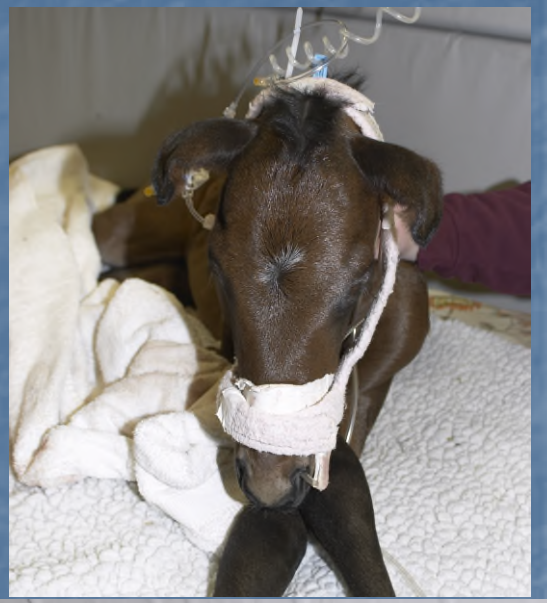


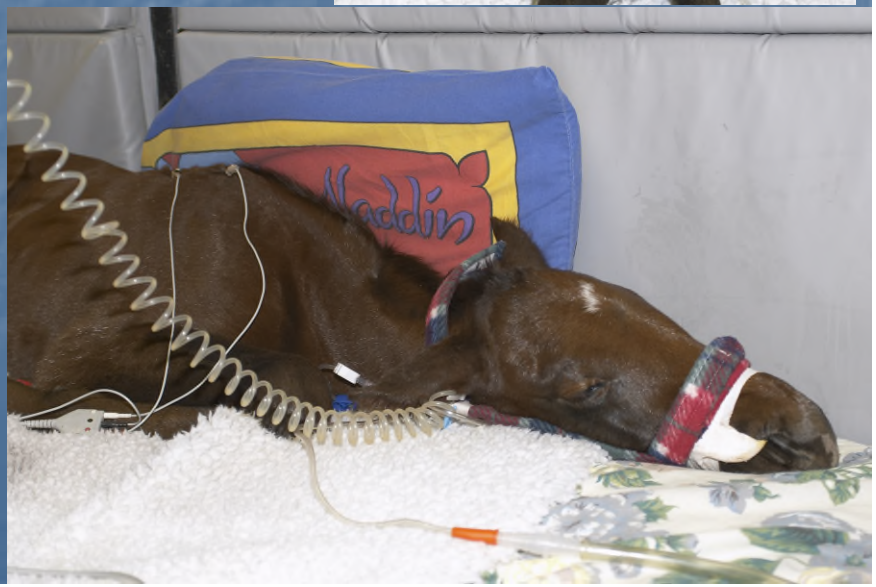




Threats to Fetal Well-being
Placentitis

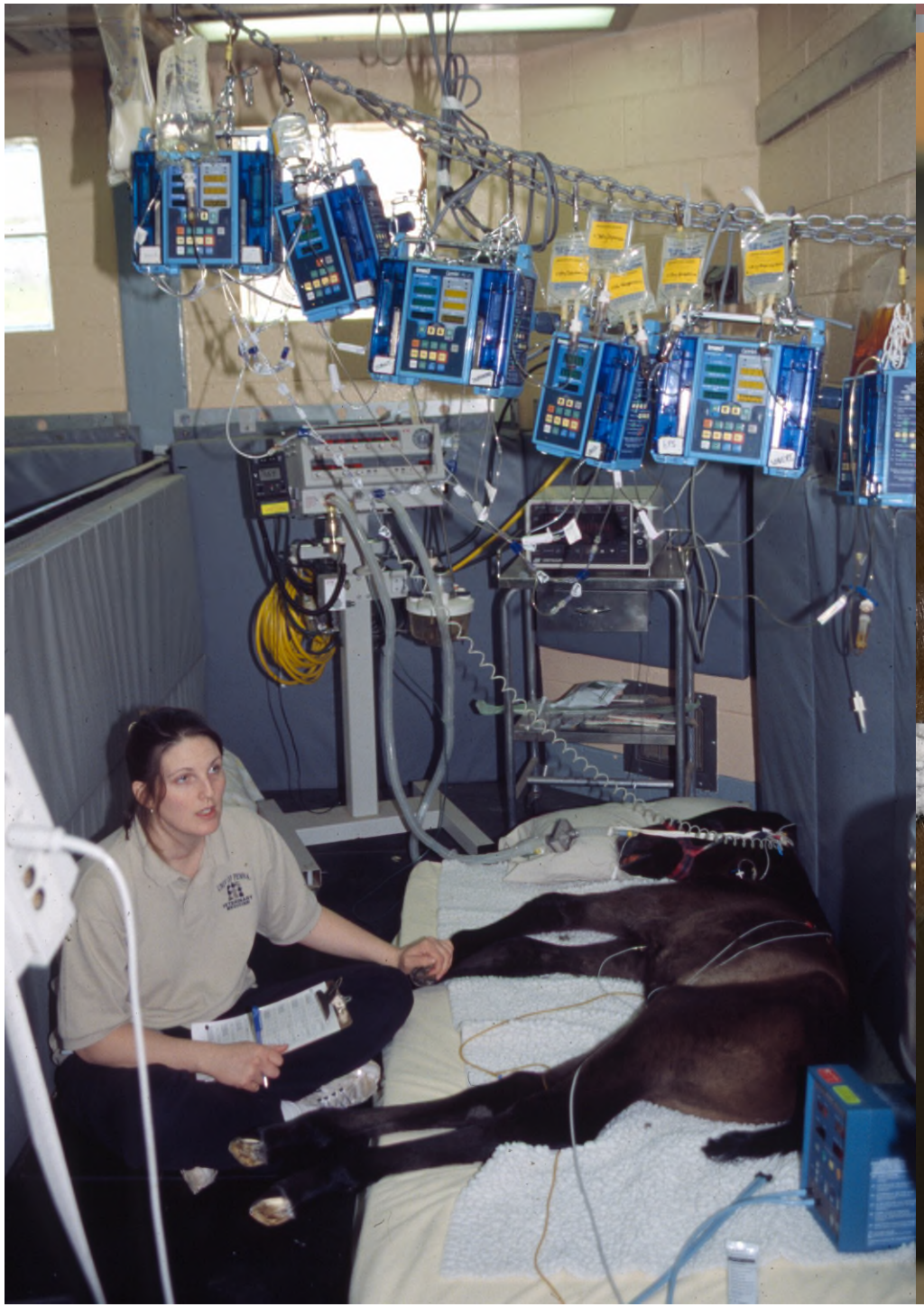
- Fetal foals born with placentitis
  - No prepartum treatment
    - 90% are abnormal
  - Treated mares
    - 50% are abnormal

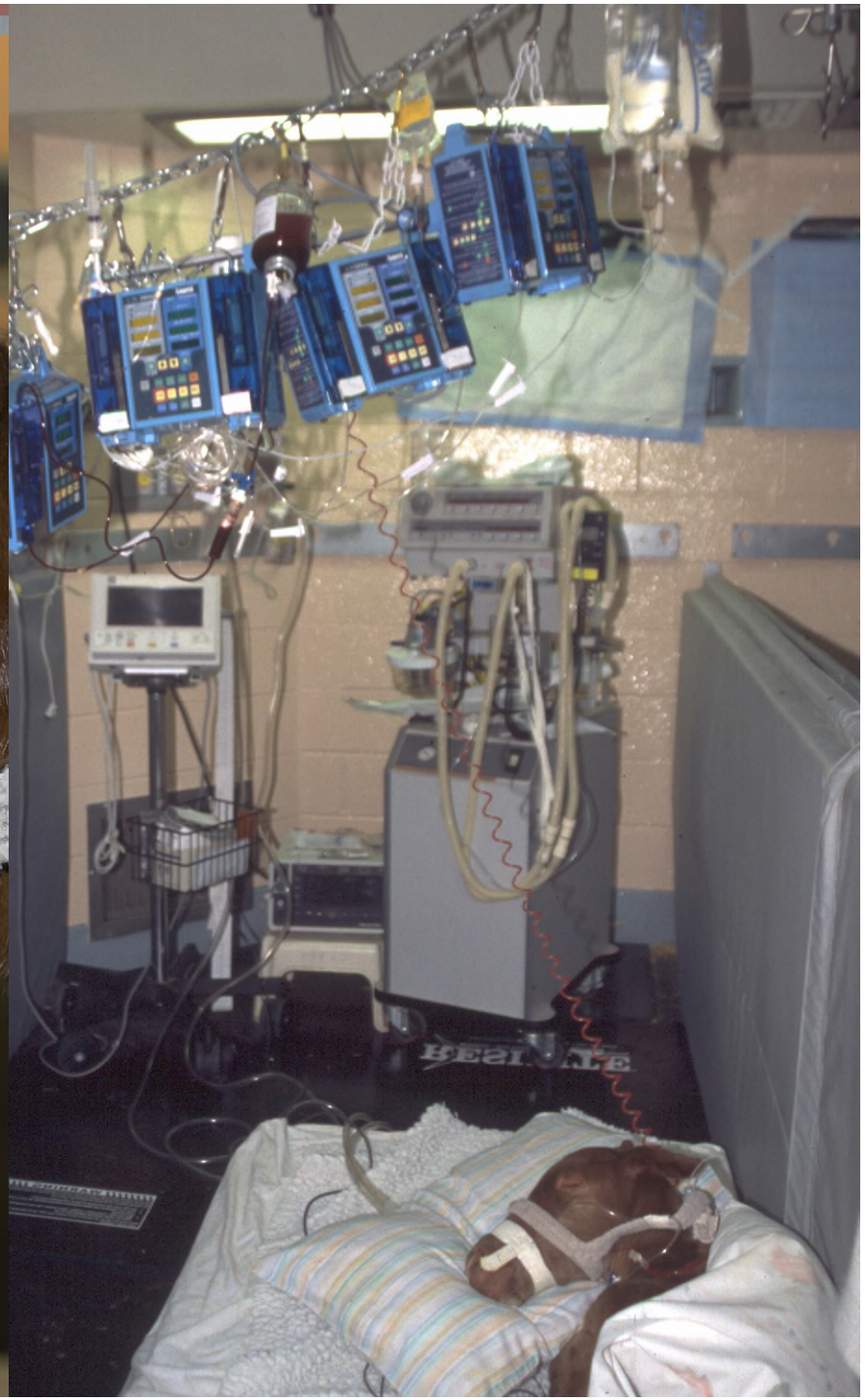




Threats to Fetal Well-being Effect of Placentitis on Foal

- Neonatal Encephalopathy – 75%
  - Neonatal Maladjustment Syndrome
- Neonatal Nephropathy – 67%
- Neonatal Gastroenteropathy – 61%
- Sepsis – 81%
- Bacteremia – 38%
- Survival rate – 73%
  - Overall survival – 84%

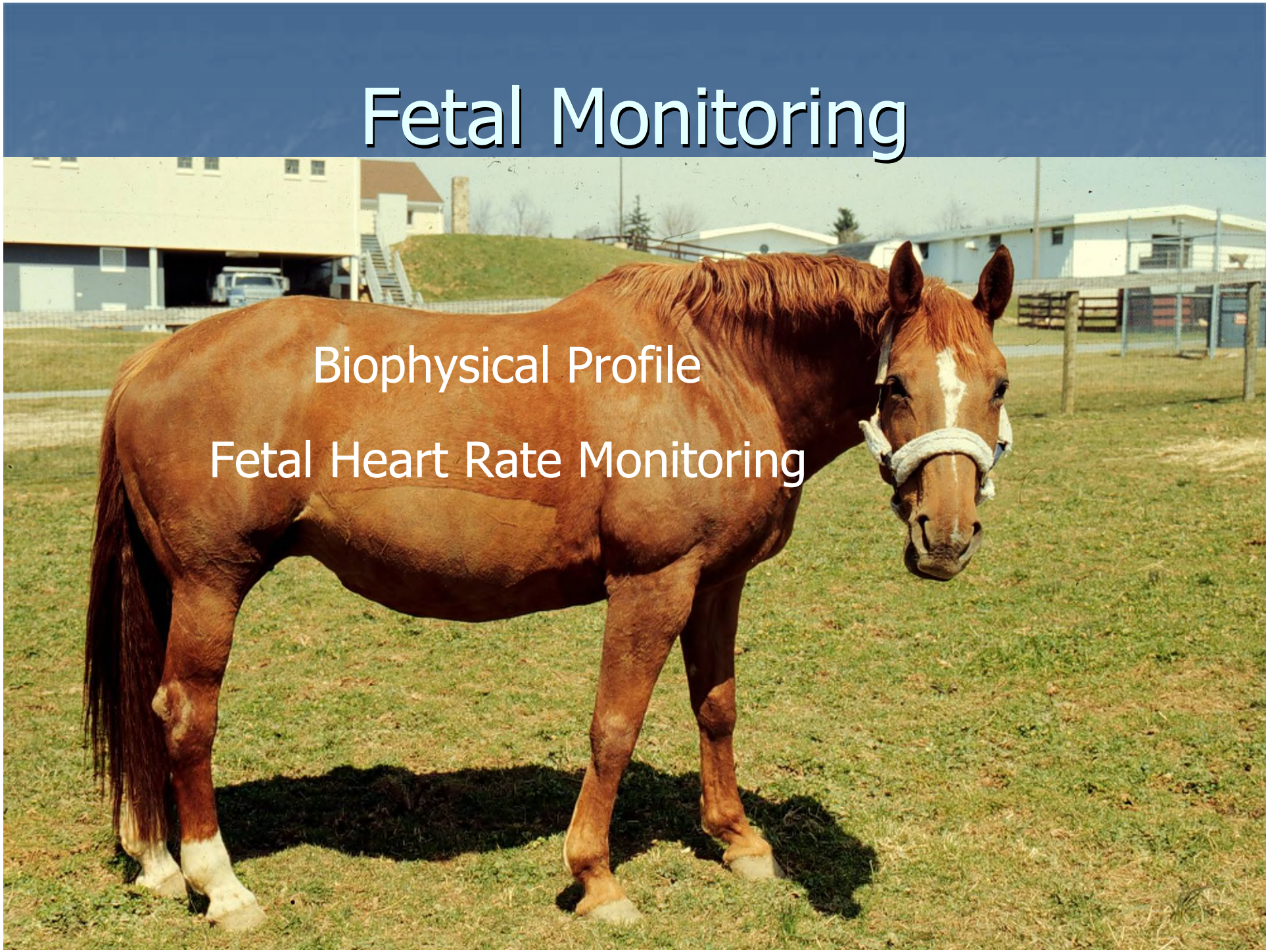




Fetal Resuscitation Placentitis - Therapy

- Antimicrobials
  - Trimethoprim potentiated sulfa drugs
  - p = 0.002, OR 0.04
- NSAID
  - Flunixin meglumine
  - p = 0.004, OR 0.03
- Progestin therapy
  - Altrenogest (ReguMate)
  - p = 0.003, OR 0.03
- Oxygen therapy
- Vitamin E
- Pentoxifylline

Fetal Monitoring

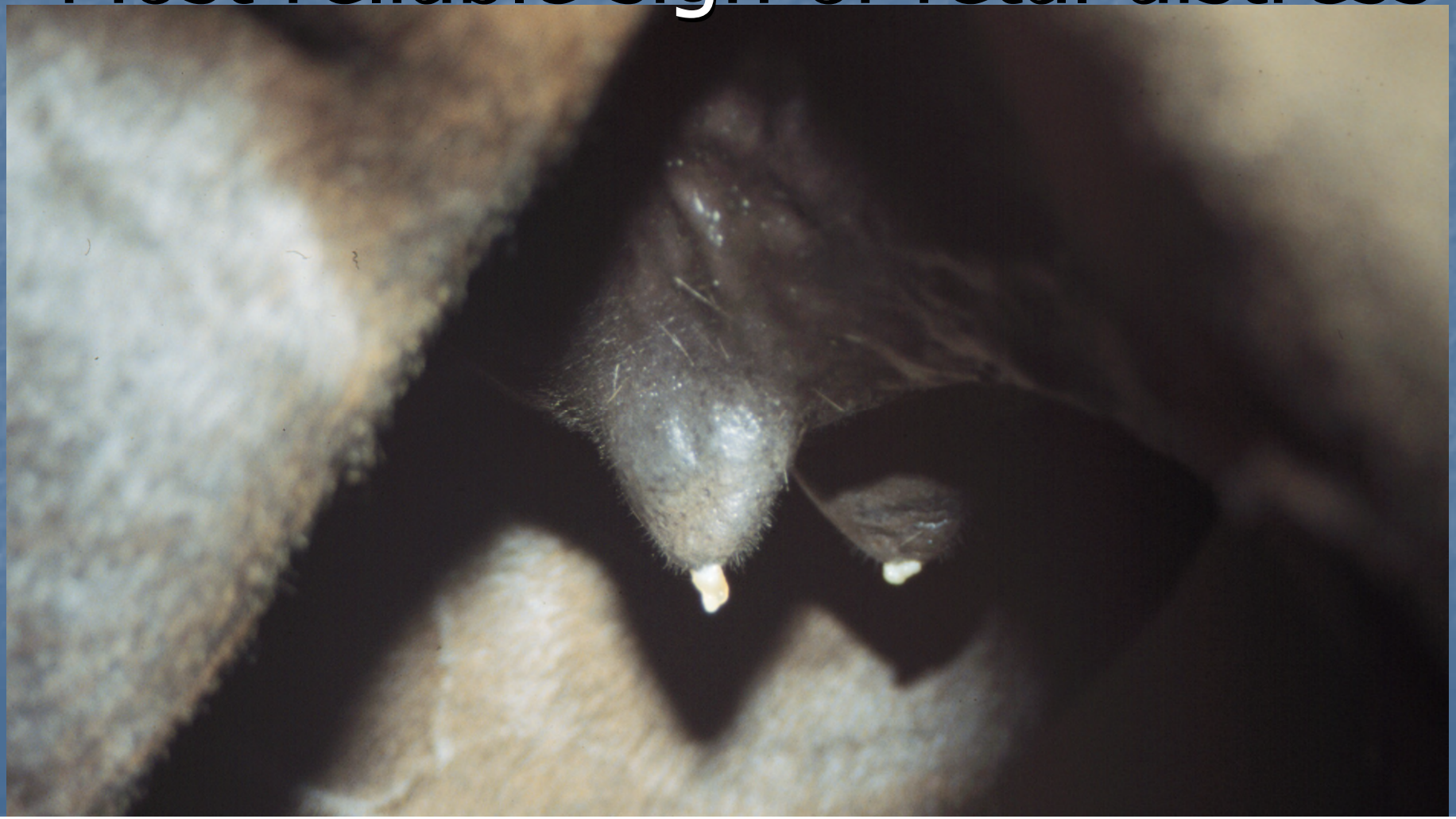


Biophysical Profile

Fetal Heart Rate Monitoring

Early Udder Development
Precocious Lactation

Most reliable sign of fetal distress



Fetal Monitoring

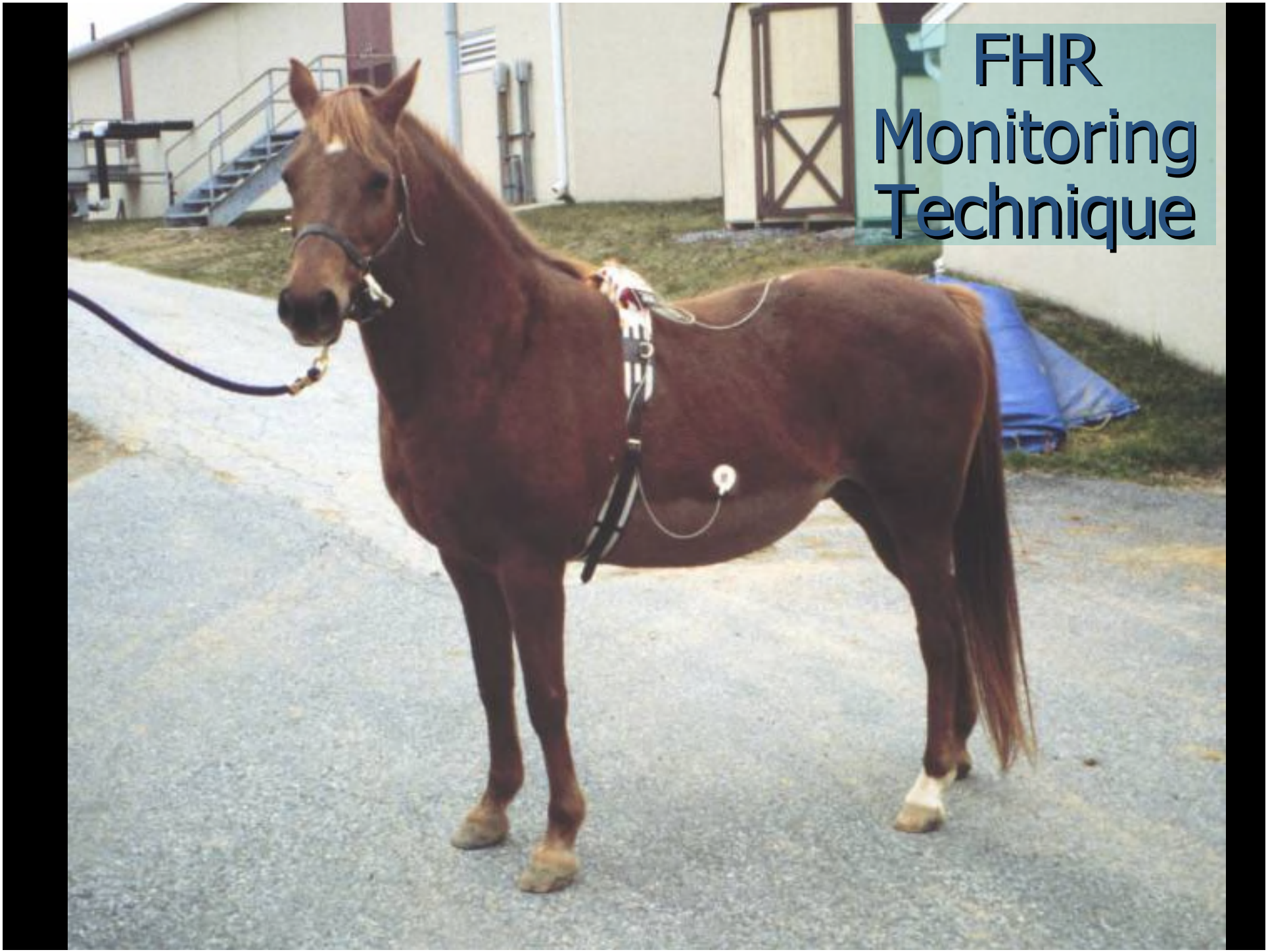
- Means of insuring fetal well-being
- Detect fetal physiologic responses
  - When normal are reassuring
  - When abnormal could justify active intervention
  - Fetal resuscitation
- Hypoxic ischemic asphyxial insult
  - Most likely fatal threat to fetal well-being
  - Fetal monitoring is designed to detect
- Other prenatal insults
  - Intrauterine inflammation
  - Important in pathogenesis of neonatal diseases

Fetal Adaptation to Hypoxia/FIRS

- Distribution of perfusion
  - Fetal hypertension
  - Fetal bradycardia
- Decreased activity
  - Fetal activity stops
  - Fetal breathing stops
  - Fetal swallowing stops
- Decreased growth
  - 50% O_2 consumption

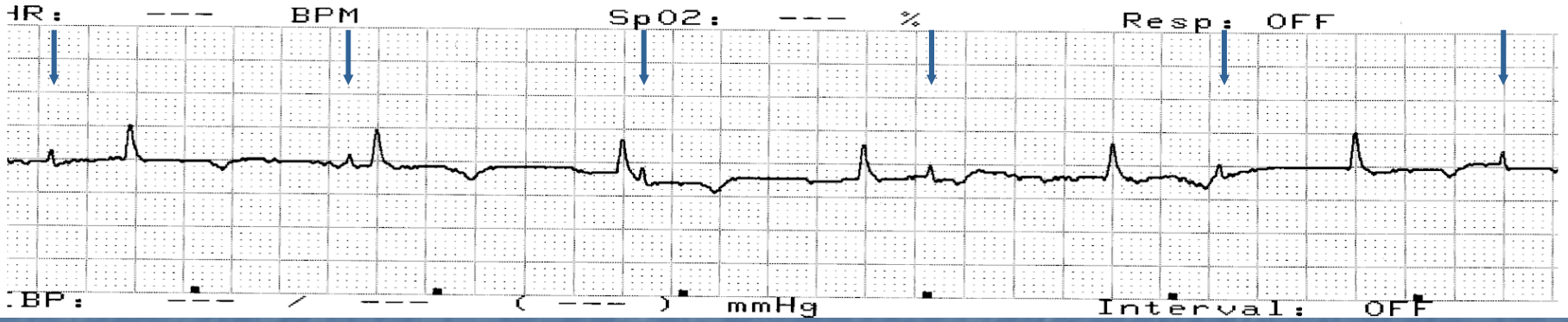
Fetal Physiologic Response to Hypoxia Ischemia

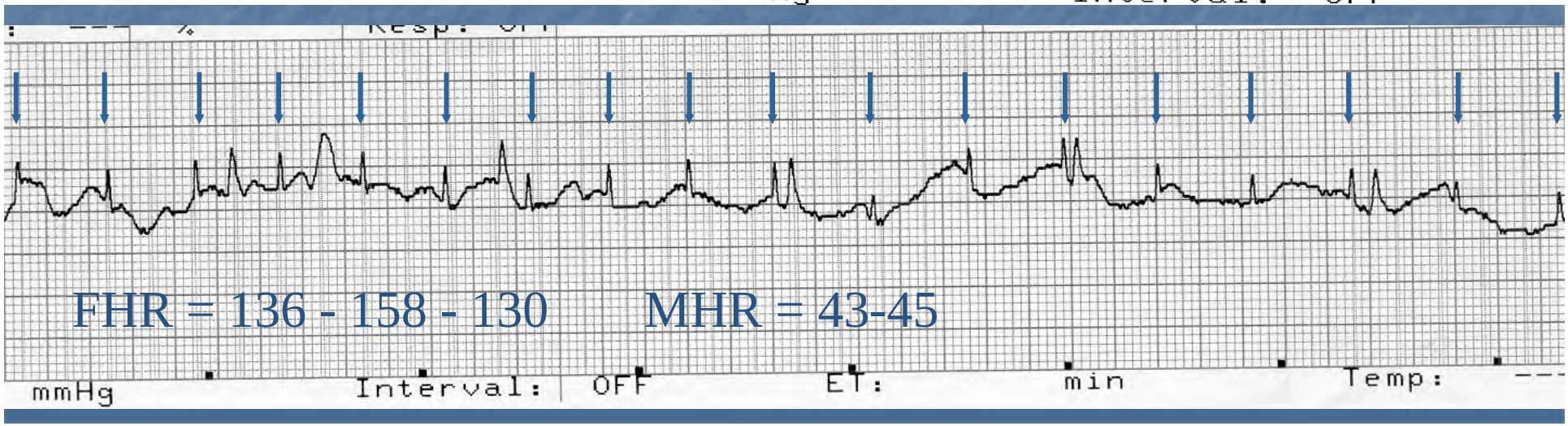
- Insult becomes severe
  - Lactic acidosis
    - Central, myocardium depression
- Compensation will fail
  - Lack intact CNS-adrenergic response
    - Loss of vascular control
  - Bradycardia will become a tachycardia
  - Terminal bradycardia
  - Hyperactive fetus
    - Escapes from central suppression


FHR
Monitoring
Technique

Fetal heart rate measurements
Fetal ECG

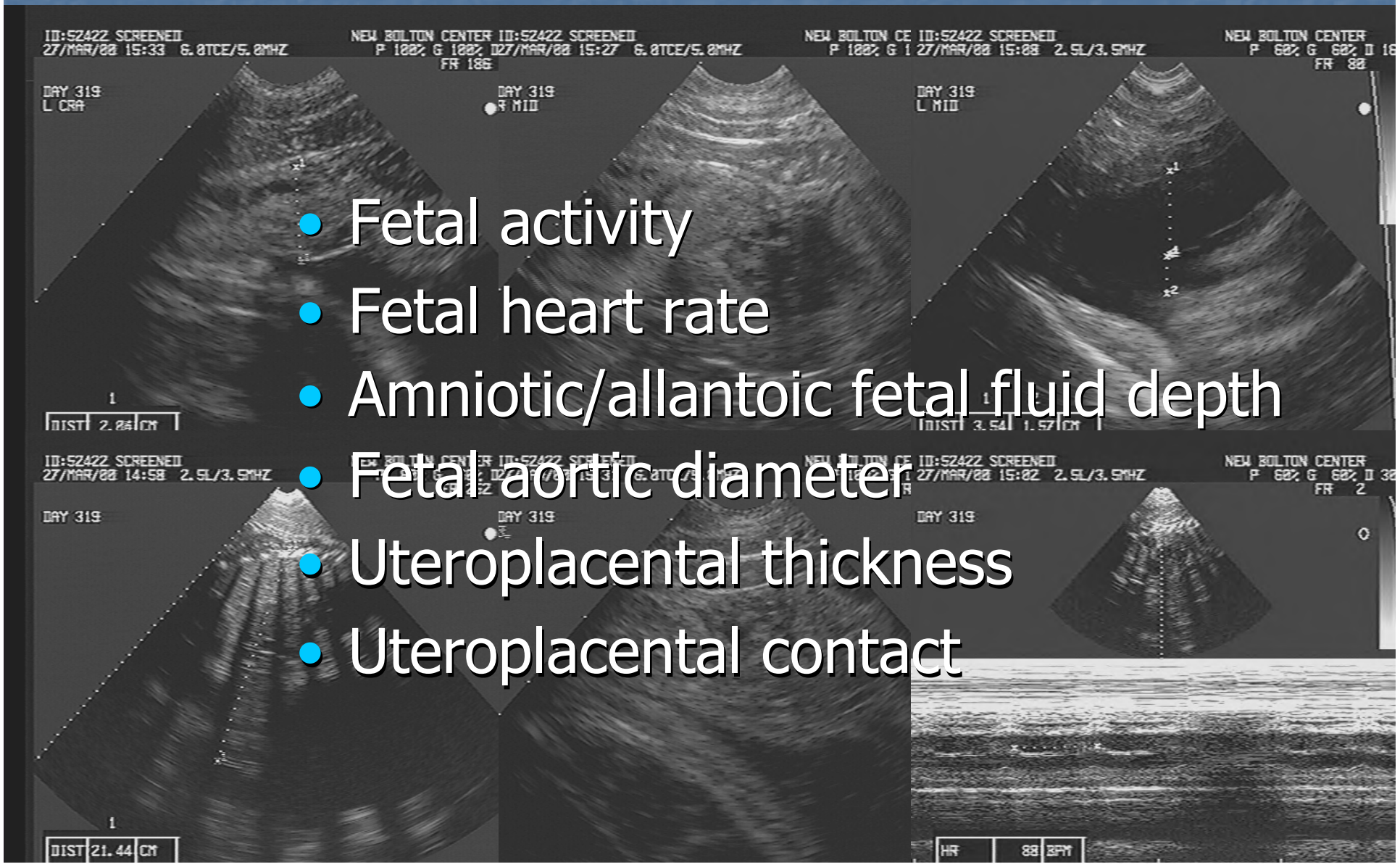
FHR = 48-52 MHR = 60





FHR = 136 - 158 - 130 MHR = 43-45

Equine Biophysical Profile



- Fetal activity
- Fetal heart rate
- Amniotic/allantoic fetal fluid depth
- Fetal aortic diameter
- Uteroplacental thickness
- Uteroplacental contact

Fetal Monitoring

- Fetal assessments using fetal monitoring techniques
  - Tempered by whole clinical picture
  - Not taken as a defining assessment
- Treat the fetus and mare
  - Not the test results
- When non-reassuring findings are consistent
  - Fetal resuscitation interventions are indicated
- Fetal death rate > neonatal death rate?
  - Abnormal uterine environment > success than intensive care

Delivery is only indicated

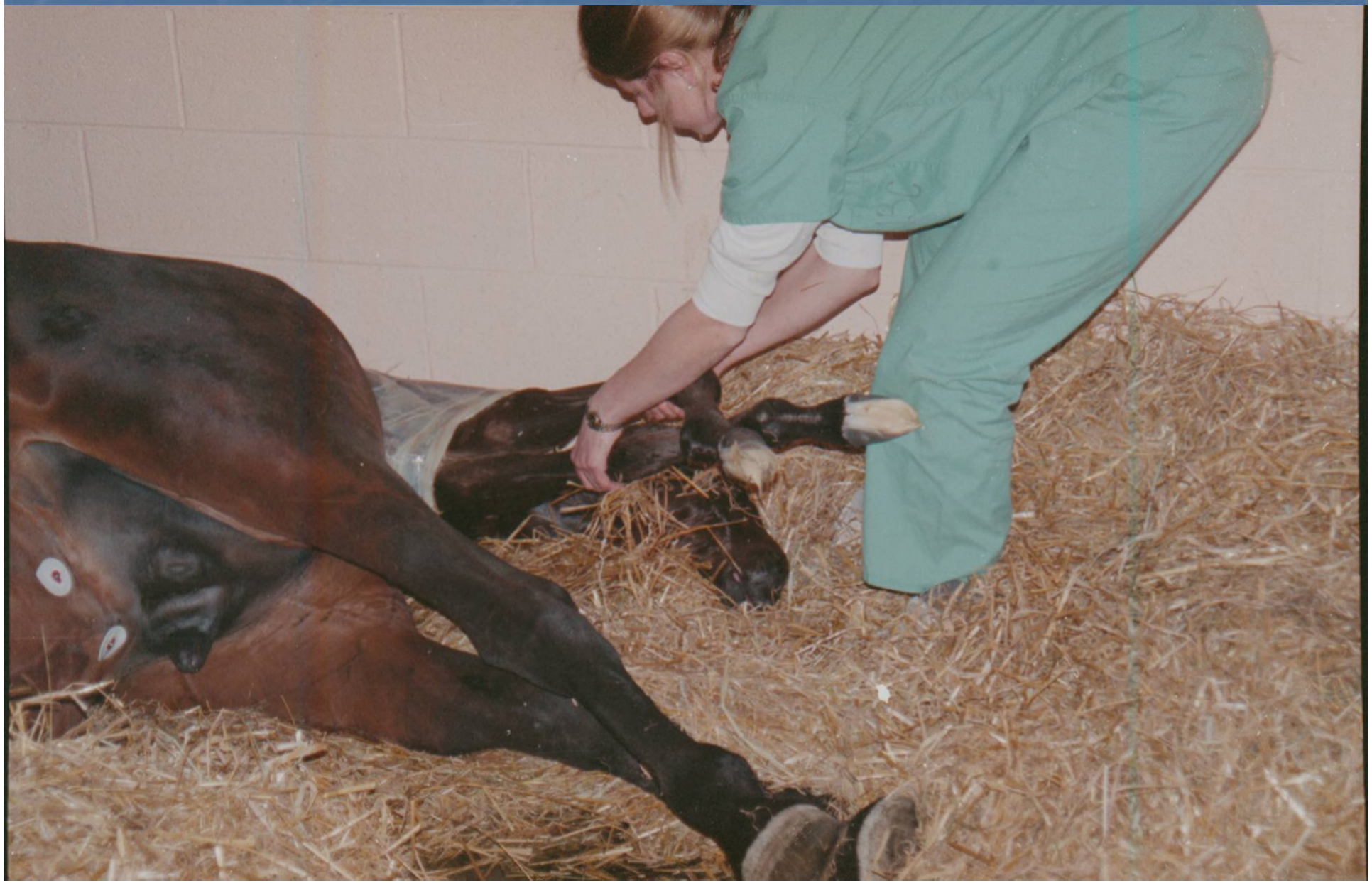
If extrauterine survival

is more likely than

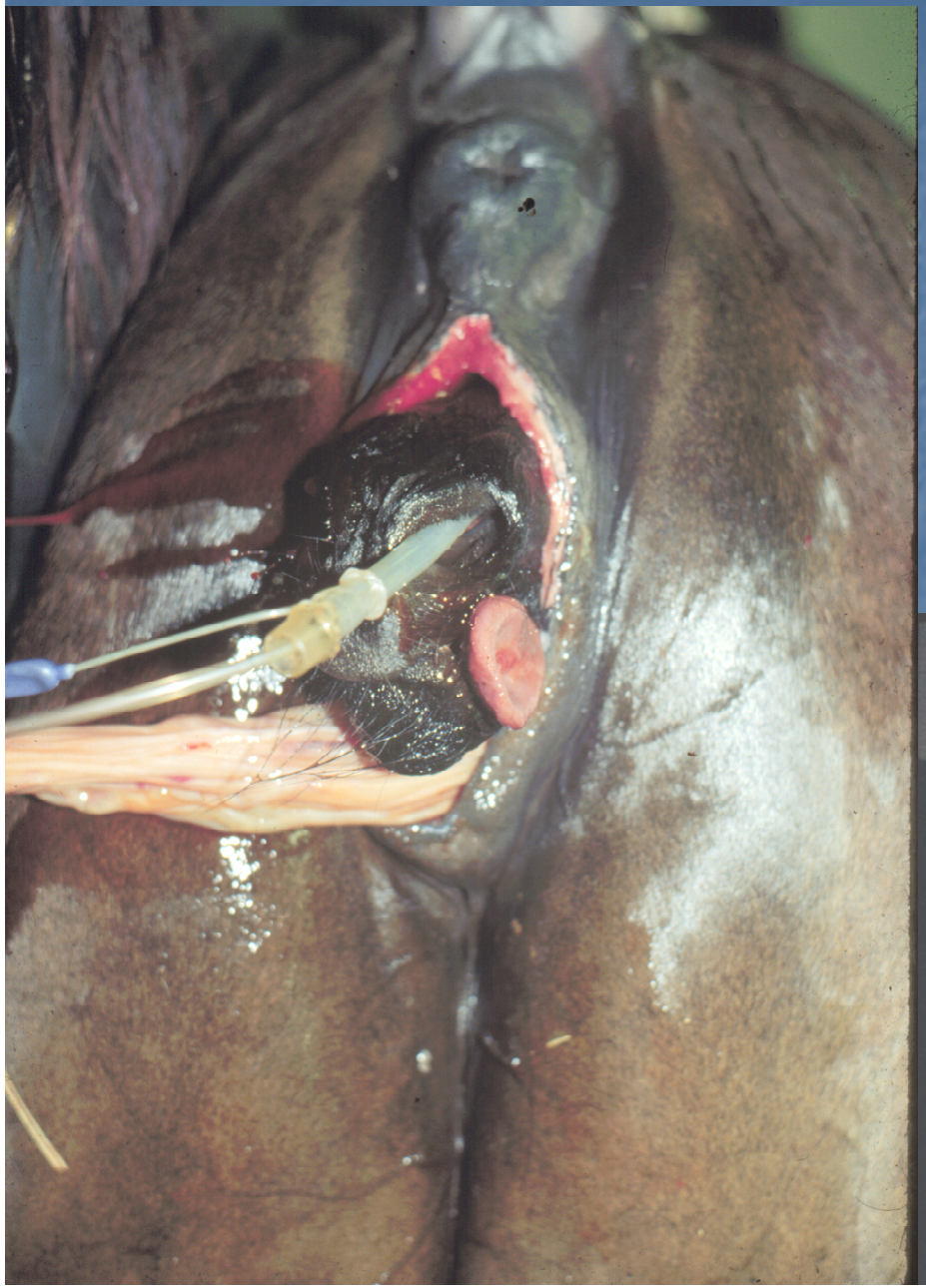
continued intrauterine

survival

No way back



EXIT Procedures



Ex-utero

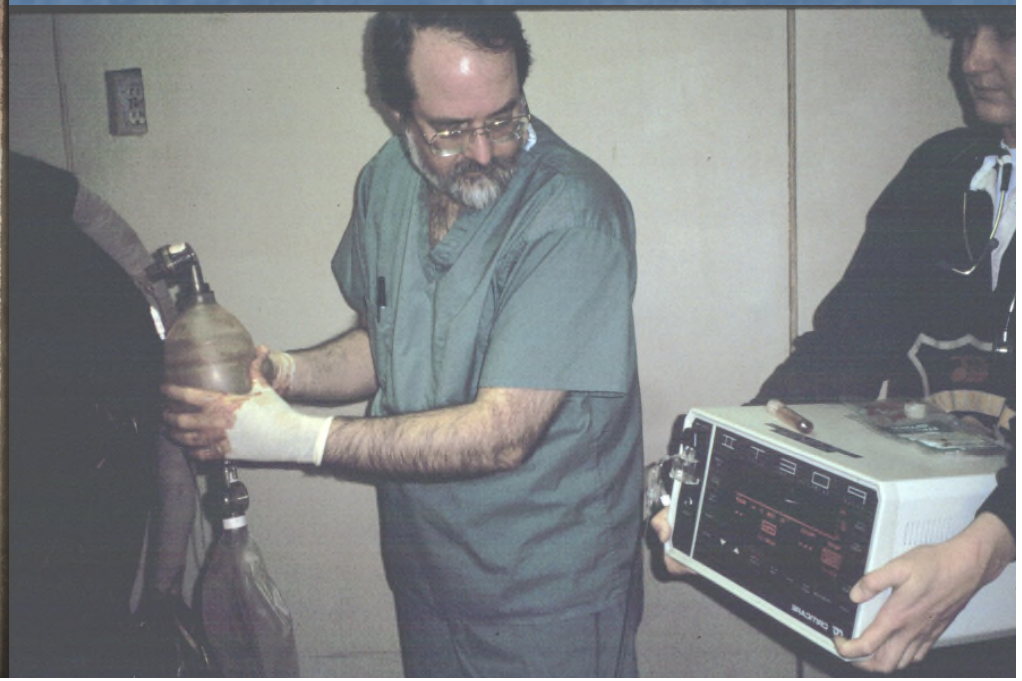
- Resuscitation during parturition

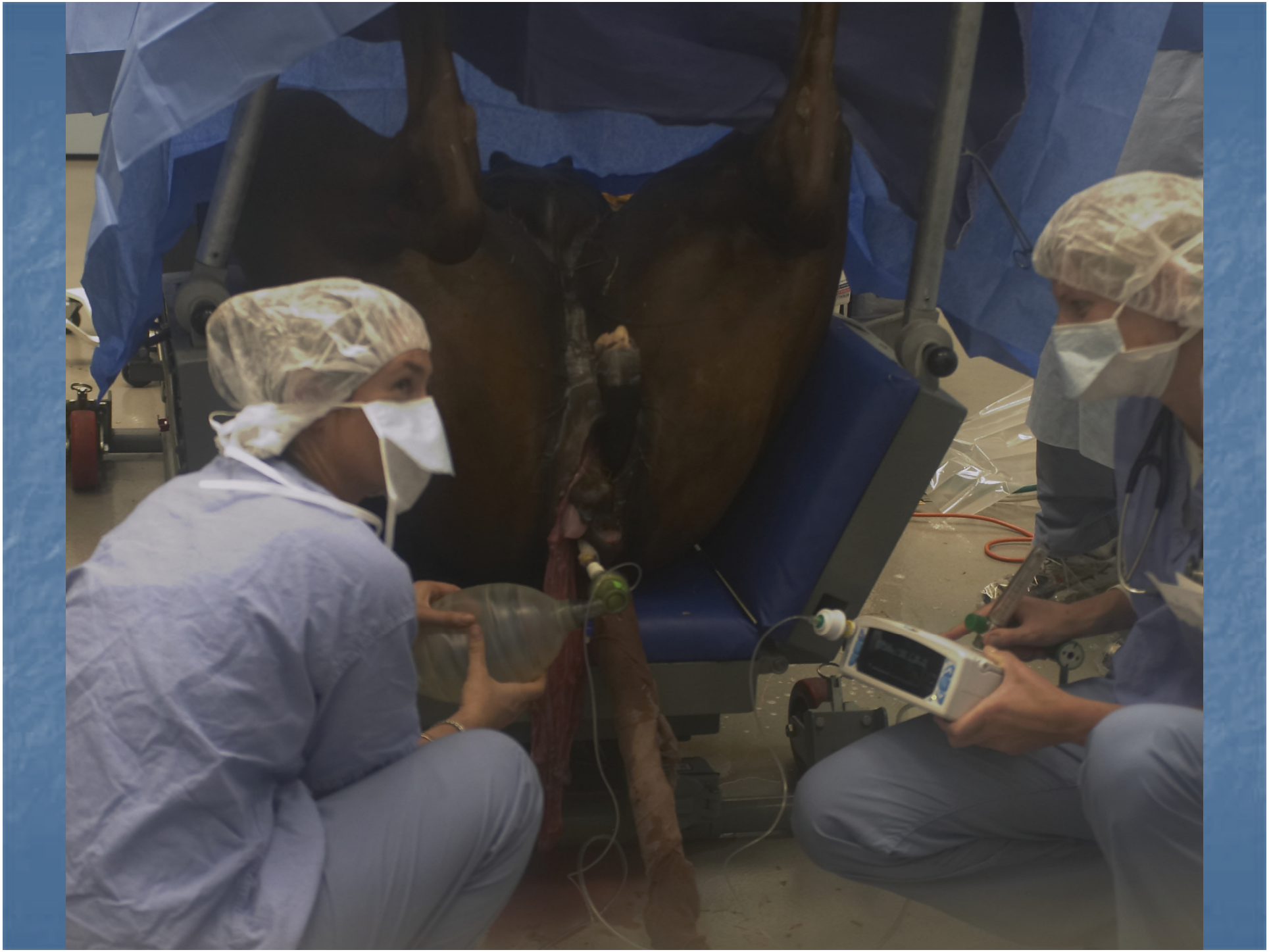
Intrapartum

- Oxygen therapy for the mare
- Facial oxygen
- Intubate if nose is available
- Resuscitation during parturition
- Use capnograph
- Can make the difference
- Expect initial poor lung perfusion

Treatment

- Facial oxygen
- Intubate if nose is available
- Resuscitation during parturition
- Use capnograph
- Can make the difference
- Expect initial poor lung perfusion

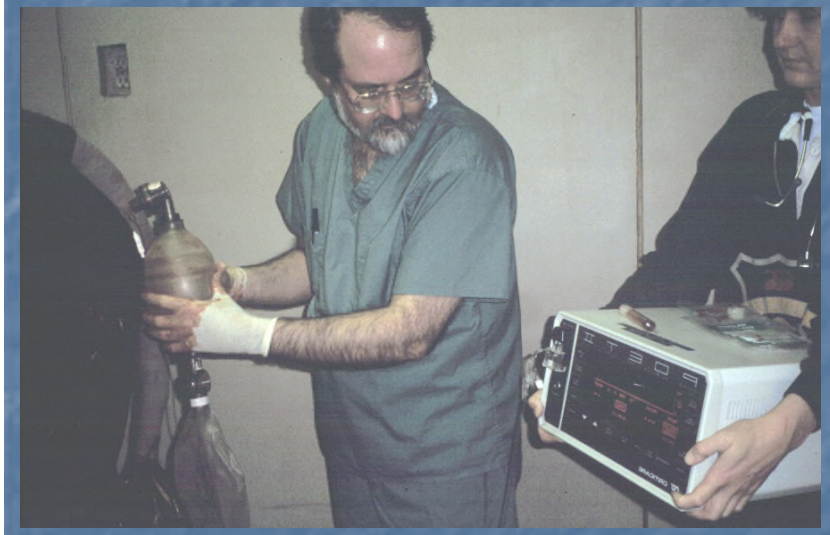




Support Foal During Stage II

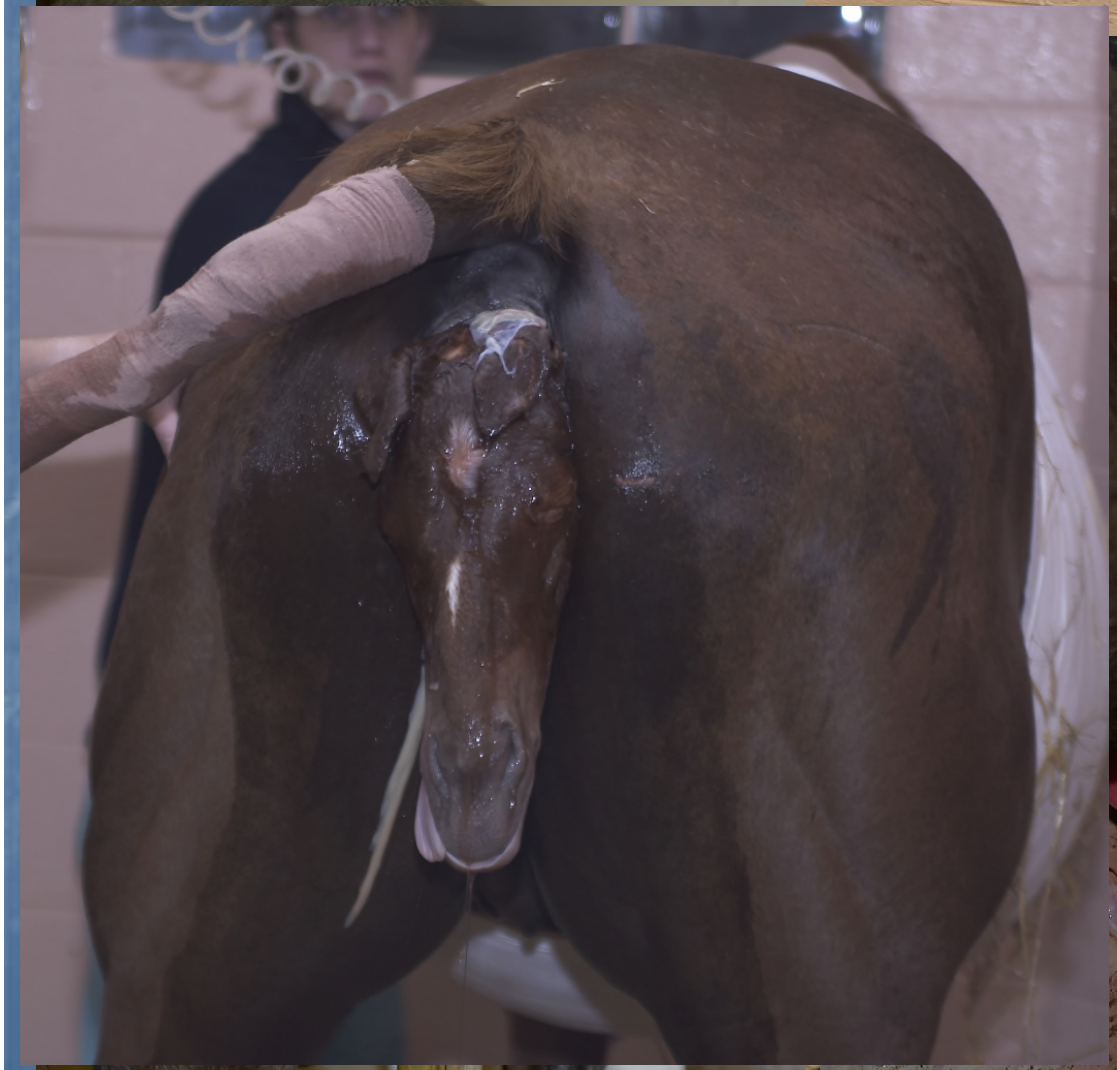
- Beginning resuscitation
- Relieving the haste to correct the dystocia
- Allowing for more time to safely correct the dystocia

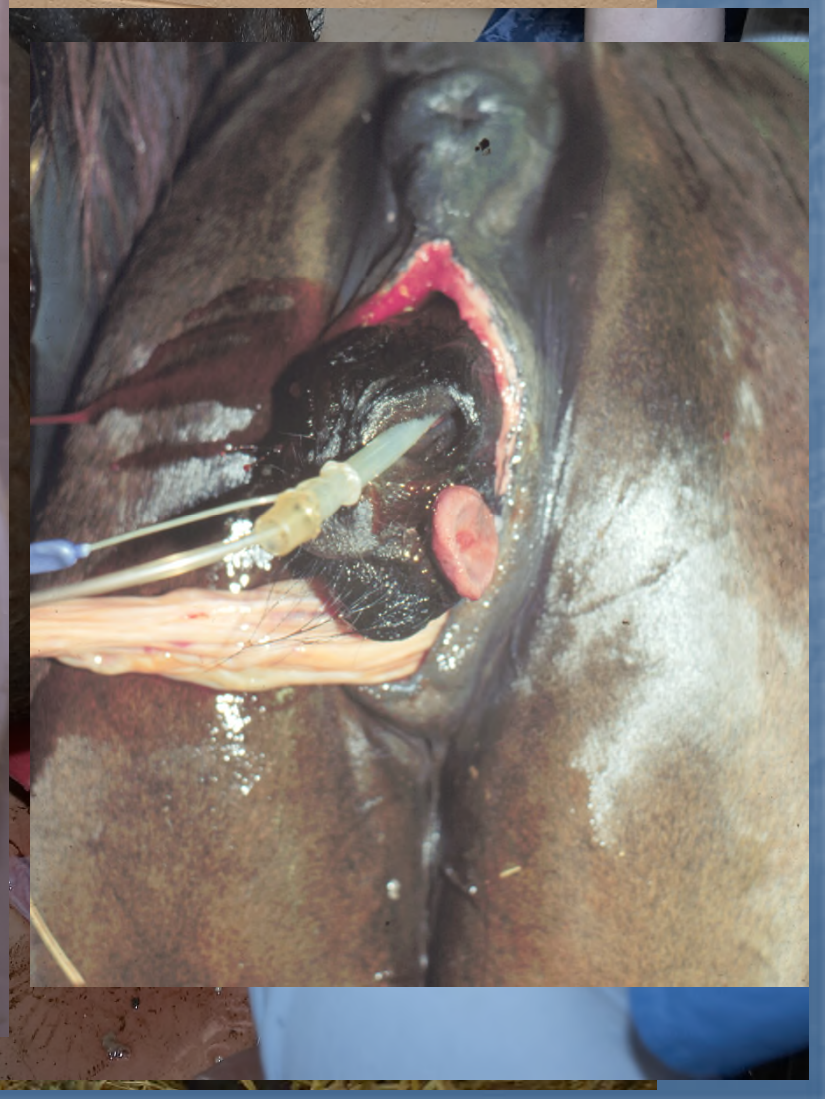
EXIT

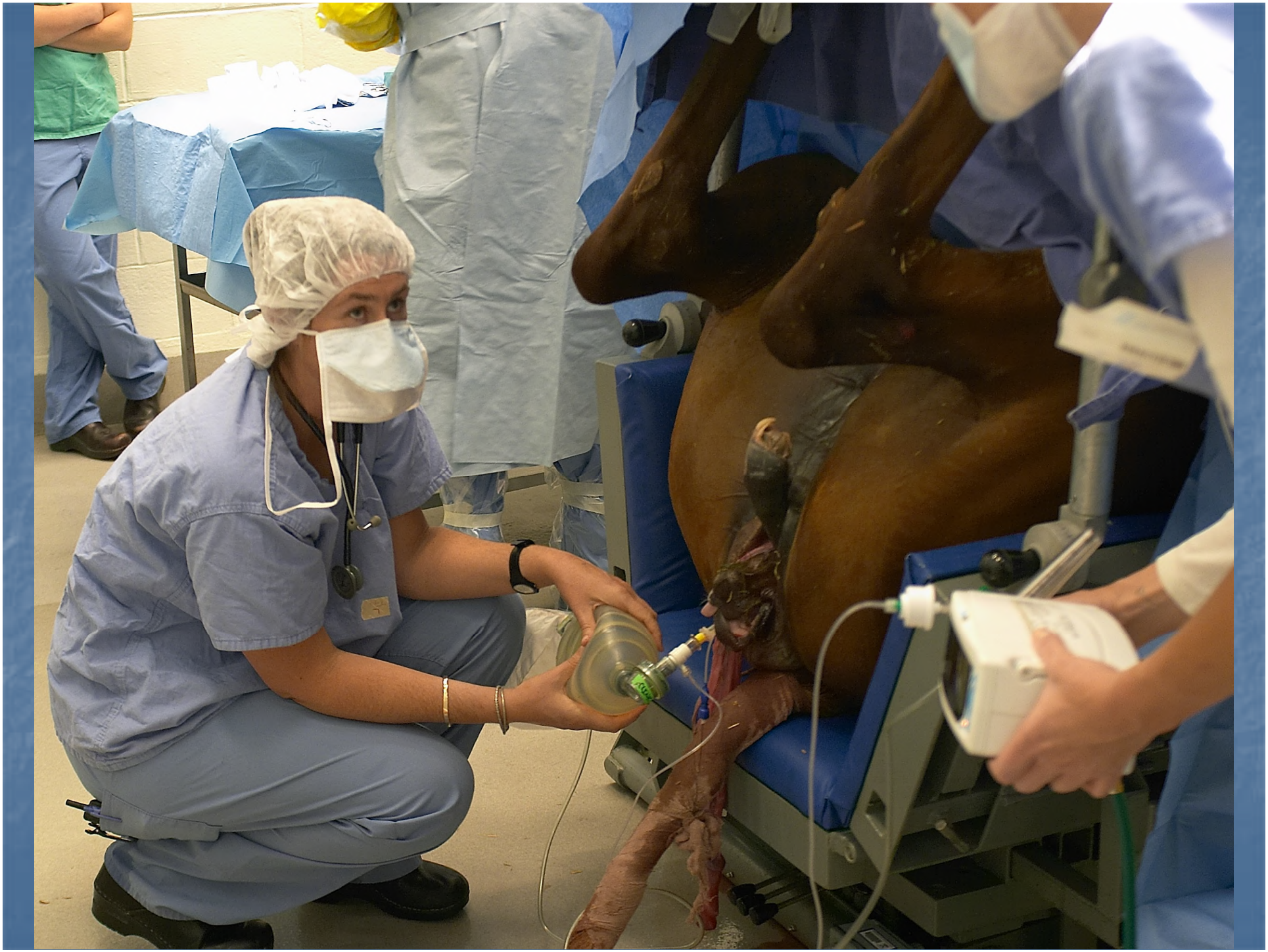


Ex-utero
Intrapartum
Treatment

Development of EXIT







EXIT

- Luxury of time to correct the dystocia
- Assess fetal viability
- Rescue foals during dystocia
- Increase successful referral radius

Birth Transition

Vital Transitions

Initiation of Breathing

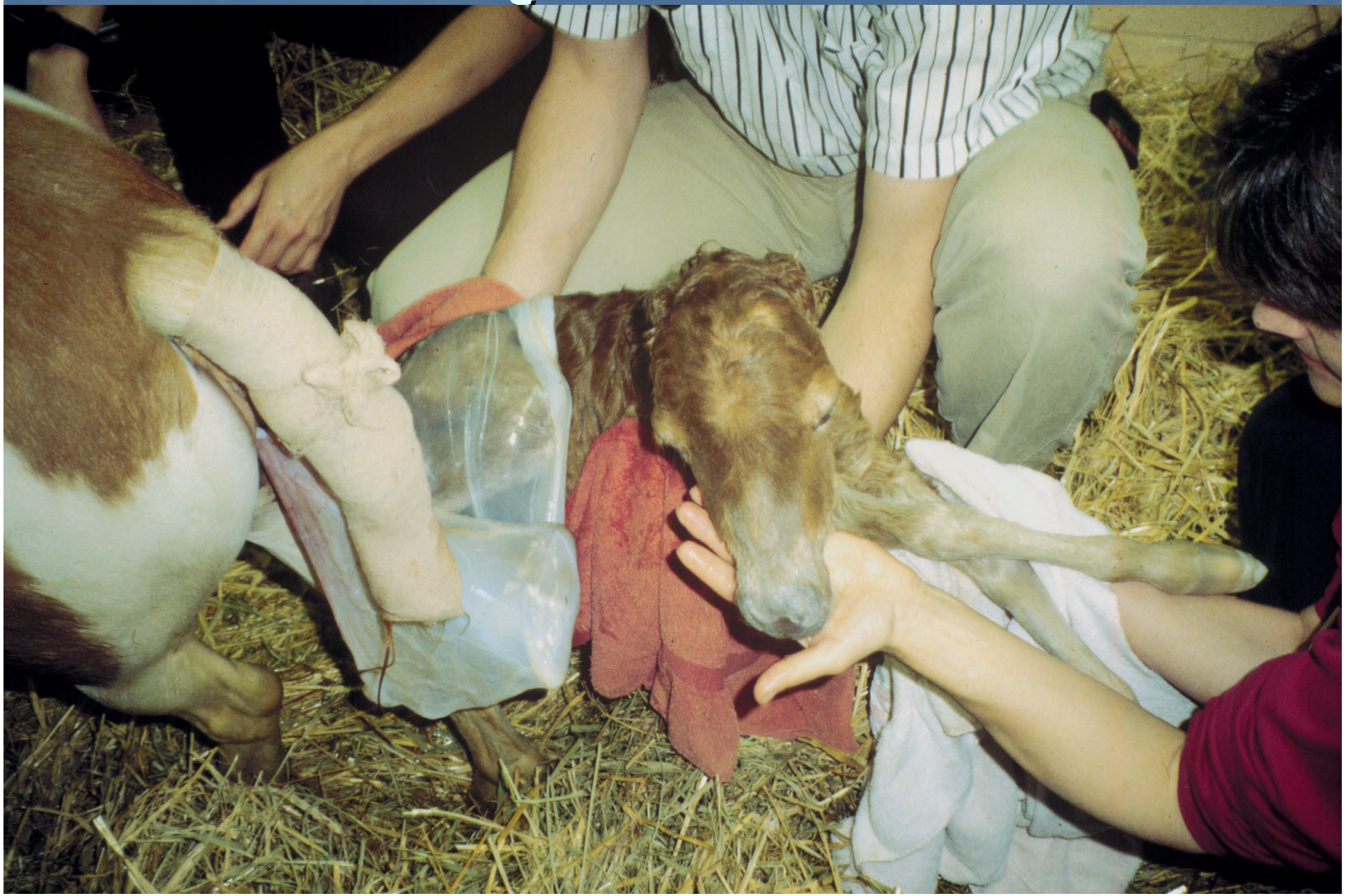
Preparation for Resuscitation

- Anticipation
- High risk situations
  - Obvious from history
  - Intrapartum course
- Unexpected
  - 50% of neonates requiring birth resuscitation
- Must always be prepared
  - Well thought out plan
  - Readily available equipment

Elements of Resuscitation

- *Initial Quick Overview*
- *Apgar score*
- *Clearing the Airway*
- *Tactile Stimulation*
- *Thermal management*
- *Free Flow Oxygen*
- *Ventilation*
- *CPR*
- *Post resuscitation care*

Initial Quick Assesment



Equine APGAR Score

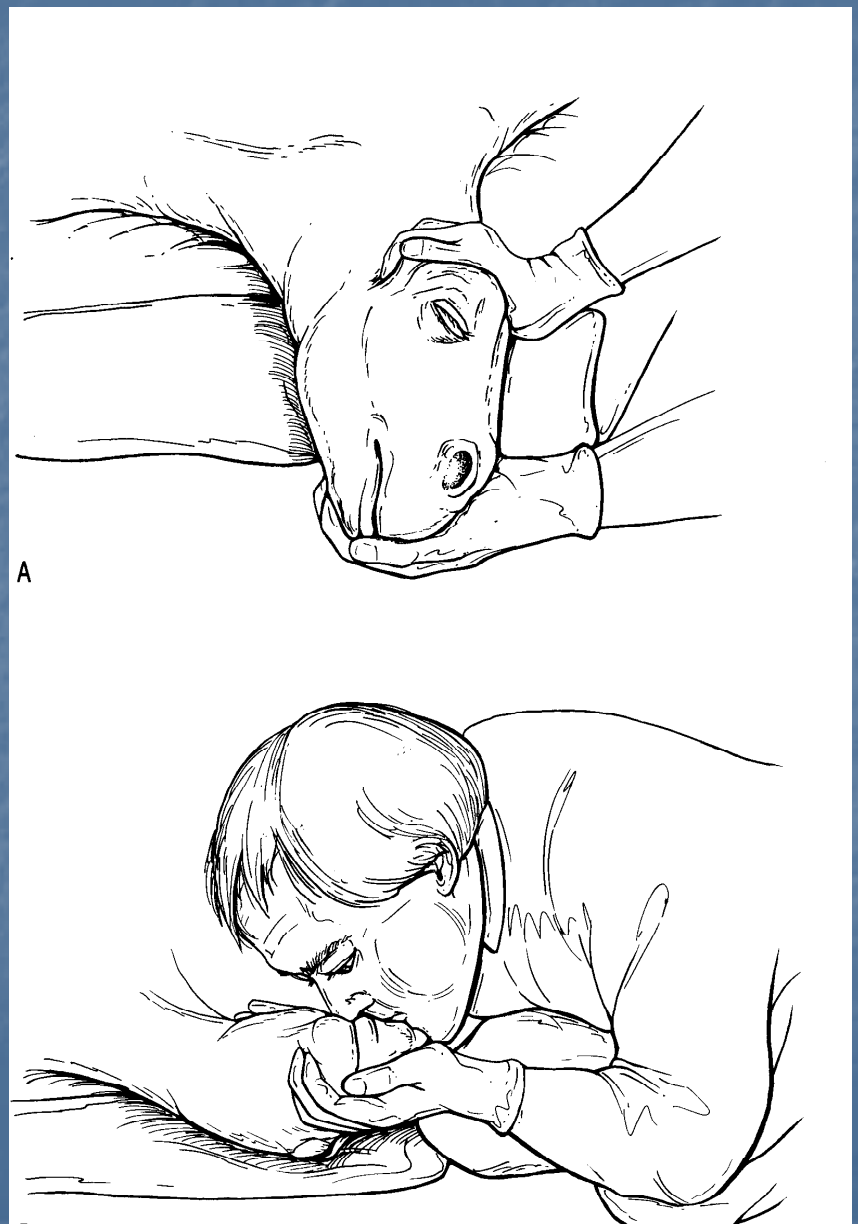
Score	*0*	*1*	*2*
Heart Rate	Absent	< 60 irregular	> 60 regular
Respiratory Rate	Absent	irregular	regular
Muscle Tone	Limp Lateral	Some Flexion	Active Sternal
Reflex ***Nasal Stimulation*** ***Ear Tickle***	No Response	Grimace Weak Ear Flick	Sneeze/Cough Ear Flick/Head Shake

Apgar Score
1, 5, 10 minutes

Clear Airway

Tactile stimulation

Mouth-to-Nose Ventilation



If the neonate does not breath spontaneously


Self-inflating
bag valve
device

Free-flow O_2

- Intranasal O_2
- Flow-by O2

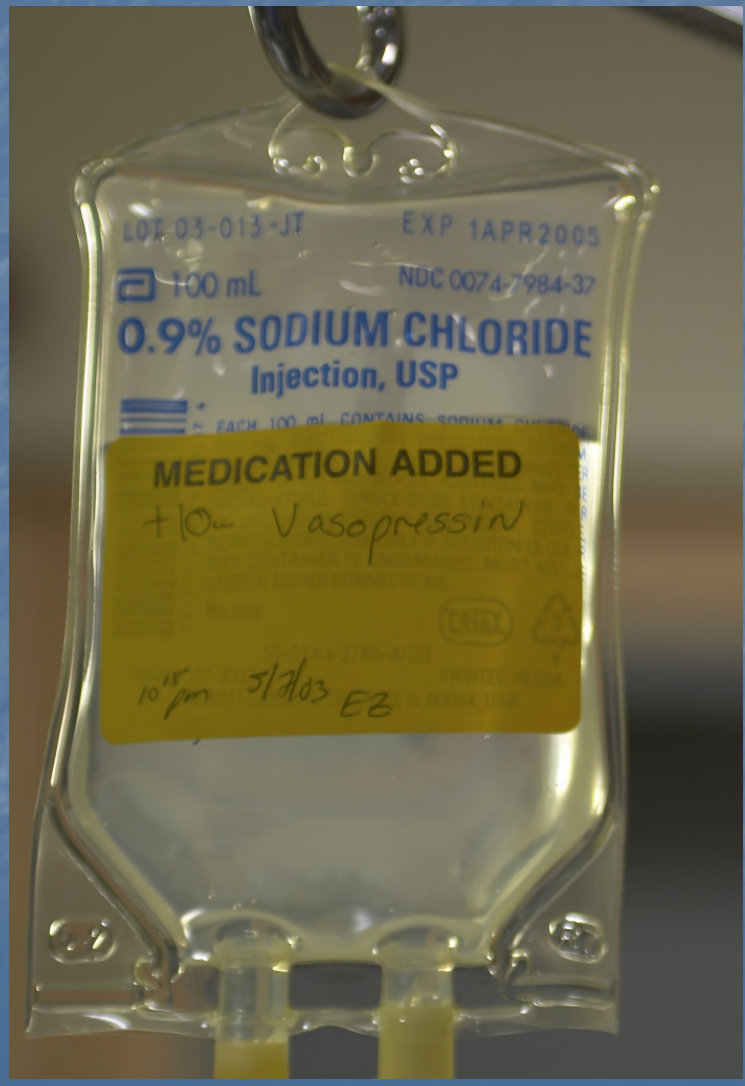

If early in asphyxia
30 sec ventilation will >HR
If late - myocardium failing
Need chest compression

Cardiovascular Support

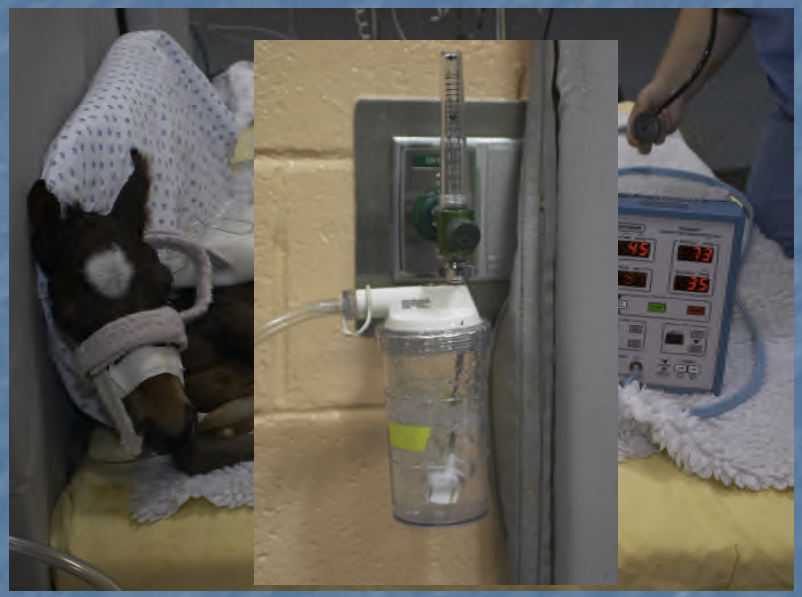
- Nonperfusing rhythm?
  - Most frequently bradycardia
- Chest compression
  - If not perfusing (bradycardia)
  - If HR not perfusing in 30 sec - use drugs

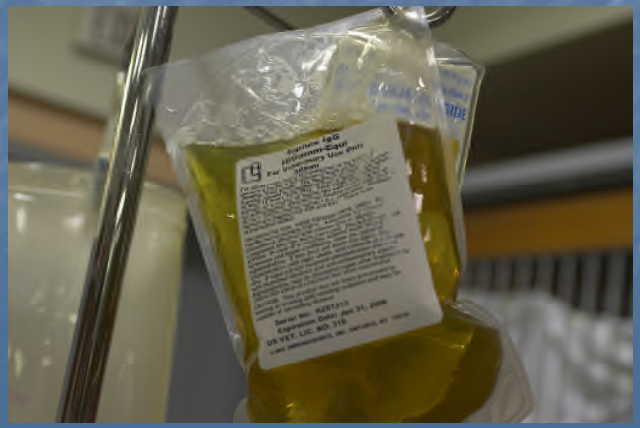
Advanced Life Saving – CPR

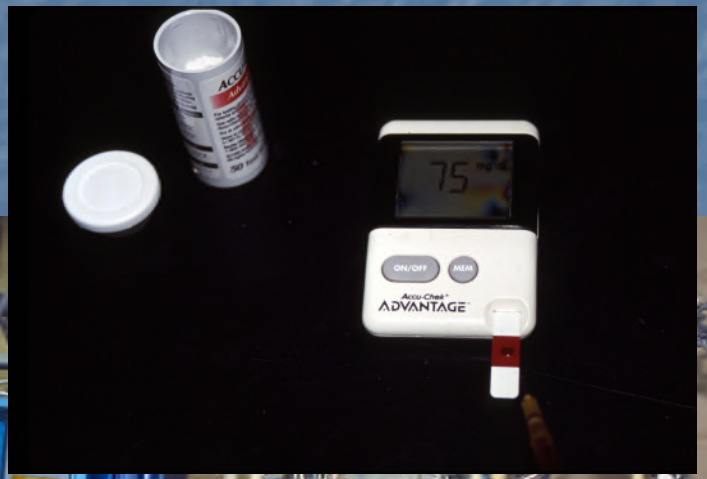
- Epinephrine
  - Low dose 0.02 mg/kg
  - High dose 0.1 mg/kg
  - Repeat every 3 min
- Vasopressin
  - 0.2 – 0.4 U/kg
  - Once



Post resuscitation care







EVALUATION OF THE HIGH RISK PREGNANT MARE/BIRTH RESUSCITATION

Jon Palmer, VMD, DACVIM
New Bolton Center, School of Veterinary Medicine
University of Pennsylvania, Kennett Square, PA

Gravid dams are considered to have a high-risk pregnancy when they have had a history of problems during past pregnancies or have developed a new problem during the current pregnancy. Problems in past gestations include placentitis, premature placental separation, recurrent dystocia, premature termination of pregnancy due to abortion or premature birth, prolonged pregnancies resulting in abnormal foals and uterine artery hemorrhage. Current problems include precocious udder development, development of placentitis, discovery of twin pregnancy, detection of premature placental separation on ultrasound examination, over-term gestation relative to past gestations, musculoskeletal problems such as fractures, laminitis and lameness, development of endotoxemia such as with colic or colitis, development of hypotension or hypoxemia, recent abdominal surgical incision, development of a body wall hernia, neurologic disease marked by ataxia, weakness or seizures, development of hydrops allantois or hydrops amnion, symptomatic pituitary hyperplasia, granulomatous intestinal disease, lymphosarcoma, melanomas in the pelvic canal, recent hemorrhage and innumerable other problems. The list of problems is endless. Although it is useful to recognize the presence of these predisposing problems, if the aim is to actively intervene to decrease the risk to the fetal foal, this is not enough. Rather the mare's problem should be viewed in terms of how it threatens fetal or neonatal well-being. After understanding the threat, then a plan to minimize or eliminate the threat can be made and carried out.

The mother has total control of the fetal environment. The fetus is dependent on and must receive everything from the mother. There is no means for the fetus to directly communicate its changing needs to the dam and if insufficient resources are delivered from the dam, the fetal foal can only survive if it can effectively redistribute the limited substrates present. The fetus has a number of sophisticated compensatory responses to the challenges brought about by disturbances in maternal homeostasis. Despite this it should be realized that as the fetus approaches term, its metabolic demands are close to exceeding the ability of the placenta to deliver substrates, so any disruption in placental function can have serious consequences.

THREATS TO FETAL WELL-BEING

Threats to fetal well-being include lack of placental perfusion, lack of oxygen delivery despite adequate perfusion, nutritional threats, placentitis/placental dysfunction, loss of fetal/maternal coordination of maturation, interaction with other fetuses (multiple pregnancy), iatrogenic factors such as drugs or other substances given to the mother or early termination of pregnancy (e.g. induction).

Lack of placental perfusion: Maternal cardiac output during pregnancy increases 30 to 50%. This may be one reason a significant percentage of late term mares have resting heart rates in the 50's and 60's without apparent discomfort. Only 50% of this increased cardiac output is directed to the gravid uterus. During the last trimester of pregnancy there is a dramatic increase in blood flow to the placenta in parallel with fetal growth. The late term fetus has a very high oxygen demand requiring a high rate of placental perfusion to deliver the needed oxygen. Any compromise of placental perfusion places the fetus at risk. The fetus can compensate for poor placental perfusion on the short term through redistribution of fetal blood flow but the margin of safety in late pregnancy is small. Whenever maternal perfusion is compromised, placental circulation and oxygen delivery may be compromised resulting in a significant threat to the fetus. Maternal hypovolemia must be treated aggressively to prevent fetal distress.

Lack of oxygen delivery to the fetus: Lack of oxygen delivery to the fetus may be a result of decreased placental perfusion, maternal anemia or maternal hypoxemia. The flow pattern of maternal and fetal blood as determined by the alignment of placental vessels determines the efficacy of gas transport. In the horse, alignment of fetal and maternal vessels results in a countercurrent flow pattern. The vessels are parallel to each other and the flows are opposite. The venous side of the fetal capillary bed is aligned with the arterial side of the maternal capillary bed so that the gradient of oxygen and other nutrients is the highest possible resulting in efficient diffusion. The countercurrent circulatory pattern results in a much higher fetal Pao_2 than other placental blood flow patterns found in most species. But despite this transport advantages there are negative consequences of countercurrent flow pattern. Maternal Pao_2 has a dominant effect on fetal Po_2. Changes in maternal P_{ao2} significantly change fetal Po_2. Maternal hypoxemia may have a profound effect on the fetal foal by predisposing the foal to hypoxic ischemic asphyxial disease. Species which do not have

countercurrent flow have developed aids to oxygen transport such as fetal hemoglobin so sufficient oxygen transport occurs despite equilibration with the low Po_2 of maternal venous blood. In the fetal foal, without such aids, maternal hypoxemia will result in insufficient oxygen transport causing hypoxic disease of the fetus and frequent neonatal disease.

Therapeutically, we can take advantage of the unique properties of the countercurrent circulatory system by increasing maternal Pao_2 resulting in more oxygen transport. This can easily be done by placing the mare on intranasal oxygen. The increase of mare's Pao_2 will significantly aid oxygen transport in perfused areas of the placenta. Serious consideration should also be given to blood transfusion therapy in anemic dams to prevent fetal hypoxemia although it may predispose to neonatal isoerythrolysis in future foals.

Nutritional Threats to Fetal Well-being: The mare's nutritional state may directly affect the fetal foal's well-being. Chronic maternal malnutrition caused by lack of intake, malabsorption, tumor cachexia or other conditions will cause significant fetal malnutrition resulting in intrauterine growth restriction (IUGR). Acute fasting in the late term mare such as during colic can cause serious problems if it is complete and lasts 30-48 hrs. The fetus can compensate for the acute lack of calories through induction of glucogenic enzymes resulting in mobilization of its own resources. The major problem caused by acute fasting is the mare's metabolic response to starvation. With decreased oral nutrition and falling blood glucose levels, the mare begins to mobilize her fat stores and plasma free fatty acids levels increase. There is an associated increase in prostaglandin production resulting in a rise of plasma and perhaps placental prostaglandin levels. This increase appears to be responsible for a cascade of events resulting in early delivery of a weak foal which is not ready for birth about a week after the fasting episode. This rise in prostaglandins and the early delivery can be prevented by treating the mare with intravenous glucose at a rate that will suppress fat mobilization (the glucose delivered does not need to meet the mare's nutrition needs). Allowing the mare to eat will also prevent the process.

It is important to support the mare's nutritional needs at the end of gestation and encourage her to stay on a high plane of nutrition. Acute fasting should be avoided. If the mare has to be fasted or becomes completely anorexic, intravenous glucose supplementation (0.5-1 mg/kg/min) should be given.

Placentitis/Placental Dysfunction: Placental disease is the most common cause of fetal morbidity in late term mares. The most common placental problems include placental infection resulting in placentitis, premature placental separation, non-infectious inflammation, placental degeneration, idiopathic placental edema and hydrops allantois/amnion. Percentage of placenta affected by inflammation is not a predictor of the fetal insult. The presence of placentitis, no matter how extensive, is enough to predict a serious problem. Foals born with placentitis are at much greater risk of having neonatal disease than those who don't. Whether this abnormality is manifested by a very compromised neonate, an asymptomatic neonate with laboratory indications of SIRS or as a neonate with precocious maturation with SIRS depends on the nature of the placentitis and the duration before parturition. All cases of placentitis should be treated as bacterial placentitis until proven otherwise. The treatment regime which I use consists of an antimicrobial (usually trimethoprim potentiated sulfa drugs), an antiprostaglandin drug (flunixin meglumine) and hormone supplementation (altrenogest) for its anti-inflammatory properties. Some clinicians use other antimediator drugs, but I usually don't find this necessary.

Other Problems: Other problems include loss of fetal/maternal coordination of readiness for birth, iatrogenic mishaps, presence of twins and idiopathic occurrences. Many foals born with hypoxic ischemic asphyxial disease have no history of abnormalities occurring during gestation or parturition. Although it is attractive to blame problems during parturition, most these problems occur during the antepartum period.

FETAL MONITORING

Equine veterinarians, in parallel with physicians, have been searching for a reliable means of insuring fetal well-being for at least the past 40 years. Most techniques attempt to detect fetal physiologic responses which when normal are reassuring and when abnormal could justify active intervention. Fetal monitoring takes advantage of physiologic responses using gross body movements as predicative of fetal health. When activity that normally arises from a given brain center is observed (e.g. fetal breathing) then that regulatory center is assumed to have adequate oxygenation and normal metabolism. Thus observing movement provides insight into fetal CNS integrity. The brain is among the most oxygen dependent tissues and among the first to sense deficits and responds by orchestrating compensatory reactions. Failure to observe a response within a prescribed period of time suggests hypoxemia as a possible cause. But in clinical practice, fetal hypoxemia is

the least likely cause of the absence of a given acute fetal biophysical response. In most cases, the failure to observe the activity is a result of normal periodicity of the variable or the effect of the normal rest/activity cycles of the fetus.

Both transabdominal and transrectal ultrasound have an important place in evaluating the health and well-being of the late term fetal foal. A number of transabdominal ultrasound observations have been related to fetal health. Fetal heart rate and rhythm, fetal aortic diameter, fetal breathing movements, fetal activity, fetal tone, fetal fluid depths, uteroplacental thickness and integrity and fetal positioning all have been utilize. The skilled ultrasonographer can obtained useful morphometric variables, survey fetal morphology, note fetal positioning, survey for placental abnormalities in the ventral viewable areas and search for twins quite efficiently. Unfortunately, there is a significant learning curve for the neophyte in obtaining accurate and repeatable observations. Assessment of the uteroplacental unit is a very important part of transabdominal ultrasound assessment of fetal health. Although it is relatively easy to measure the uteroplacental unit since the borders are distinct, avoiding areas near the nonfetal horn is very important. The uteroplacental unit is quite thick (up to 4 cm) in the nonfetal horn and the areas adjacent to it and gradually decreased as the gravid areas of the uterus are approached. This can cause considerable confusion in uteroplacental unit thickness measurement. With all the opportunity for error in morphometric measurements and subjective evaluations necessary, transabdominal ultrasound might better be considered an art most efficient learned through apprenticeship and experience rather than a science that can easily be reproduced by rote.

Over the past decade I have spent considerable time observing fetal heart rate patterns in our high risk pregnancy population. Although our understanding of changing patterns is certainly rudimentary, observations can be helpful in gauging fetal health. When recording fetal heart rate (FHR) patterns both beat-to-beat variations and changes in baseline heart rate levels with periodic accelerations are important. Periodic accelerations are generally associated with fetal activity and suggest fetal health. Persistent tachycardia, on the other hand, suggests fetal distress. Fetal bradycardia may be present as a normal adaptive heart rate pattern suggesting either efficiency or early adaptation to hypoxia. Extreme fetal bradycardia may occur during terminal stages of fetal distress. So the usual pattern of fetal heart rate changes seen with fetal distress would be an initial bradycardia dropping below the baseline heart rate without periodic accelerations during early compensation followed by a persistent fetal tachycardia without periods of return to baseline levels because of fetal decompensation and finally terminal bradycardia.

During the last weeks of pregnancy fetal foals usually have a baseline heart rate between 75-60 with a low heart rate in the range of 75-40 bpm (80% will have a low fetal heart rate < 70, 55% low FHR < 60, 14% low FHR < 50) and the high FHR in the range of 83-250 bpm (86% will have a high fetal heart rate > 100, 50% high FHR > 120, 20% high FHR > 200). As indicated, transient low heart rates <60 bpm are very common and should not be considered ominous unless they are consistent with no accelerations. Also, FHR transiently may be > 200 bpm. Transient FHR > 120 bpm are not ominous unless they are consistently in that range and do not dropped to baseline levels. In either case, when FHR are <60 or greater than 120 throughout an observation period, repeat assessment within 24 hours or less is indicated. Beat-to-beat variability generally ranges from 0.5-4 mm with most in the range of 1 mm. The finding of no beat-to-beat variation in the absence of maternal drugs that many sedate the fetus is an ominous sign and repeat observations are indicated.

All fetal assessments using fetal monitoring techniques must be tempered by the whole clinical picture and not taken as a defining assessment. The goal is to treat the fetus and mare and not the test results. When non-reassuring findings are consistent, fetal resuscitation interventions are indicated. If the neonatal death rate is likely to exceed the fetal death rate the fetal foal should be left where it is. Even an abnormal uterine environment is often more successful at maintaining the fetal foals life than neonatal intensive care is. Delivery is only indicated if extrauterine survival is more likely than continued intrauterine survival.

EXIT TECHNIQUE

The explosive nature of parturition in the mare makes dystocia a life threatening event for both the mare and the foal. The duration of Stage II labor has an inverse relationship with foal survival rate. I have developed a technique designed to support foals during a dystocia, called EXIT (*ex utero* intrapartum treatment), allowing for survival of the foal during a prolongation of Stage II and thus rescuing the foal, relieving the haste to correct the dystocia and allowing for more time to safely correct the dystocia.

During a dystocia, if the nose presents in the pelvic canal and is palpable or if the nares are external, intubation should be attempted. Placement of the tube can be checked by passing the hand to the level of the cranial esophagus and insuring that the tube has not been inadvertently placed in the esophagus. Once ventilation is initiated using a self inflating bag-value device, a capnograph can be utilized to monitor cardiac output ensuring that the foal is alive.

The EXIT technique was a natural evolution of early birth resuscitation. During a dystocia, if the fetus is found viable through successful EXIT, then fetal manipulations can be performed with less haste since EXIT will support fetal life until delivery. Another advantage of EXIT in situations where general anesthesia is necessary to correct the dystocia is the redirection of placental blood flow to the lungs. Once EXIT initiates pulmonary ventilation, there is a decrease in placental blood flow. The advantage of reduced placental blood flow is decrease transfer of anesthetic agents and other drugs from maternal circulation to the fetus. Although difficult to quantitate, antidotal experience shows a remarkable reduction in neonatal depression after delivery by cesarean section when EXIT is performed throughout the surgery. EXIT is only possible if the nares are palpable in the birth canal and intubation is successful, which excludes a percentage of dystocia cases. EXIT procedures provide the luxury of time to correct the dystocia, a means to assess fetal viability and a means to rescue fetal foals during dystocia. During equine dystocia the use of EXIT should be considered.