

High Risk Pregnancy

A photograph showing a man with a beard and glasses, wearing a green shirt, and a woman with dark hair, wearing a light green scrub top. They are both looking down at a small, dark object held in the woman's hands. In the background, a horse is visible, partially obscured by a white cloth. The setting appears to be a clinical or hospital environment with a light-colored wall.

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1990 - 2005

2053 Neonates – 84% survivors







High Risk Pregnancy

- History of previous problems
- Development of new problems



High Risk Pregnancy Recurrent Problems

- Premature placental separation
- Recurrent dystocia
- Premature termination of pregnancy
 - Abortion or premature birth
- Prolonged Pregnancies
- Past uterine artery hemorrhage
- Past neonatal isoerythrolysis foals

High Risk Pregnancy

Current Problems

- Premature udder development
- Placentitis
- Twins
- Premature placental separation
- Overdue
 - Fescue toxicity

High Risk Pregnancy

Current problems

- Muscular skeletal problem
- Endotoxemia
- Recent hypotension/hypoxemia
- Recent abdominal surgical incisions
- Neurologic disease
- Hydrops allantois/amnion
- Pituitary hyperplasia
- Tumors





Perinatology

A photograph of a dark-colored horse in a hospital stall. The horse is standing behind a metal gate. To the right of the horse, there is a complex setup of medical equipment. Several blue electronic monitors are mounted on a metal stand. A white medical monitor on a stand is also visible. A person in a white lab coat is partially visible in the lower left corner, attending to the horse. The stall has a brown tiled wall and a window with a metal grate.

- What is the threat to the fetus/neonate?
- 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 O₂ 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
 - Hypoxic ischemic insult



Threats to Fetal Well-being

Unique Aspects Placental Blood Flow

- Fetal Foal
 - Placental blood flow - 50% of the dam's flow
- Other Fetal Species
 - Placental blood flow - 75% of the dam's flow
- Fetal Foal under anesthesia
 - Dramatic decrease in fetal placental blood flow
 - 38% of maternal flow
 - No significant change in maternal placental blood flow

Fetal Resuscitation

Maintenance of Placental Perfusion

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

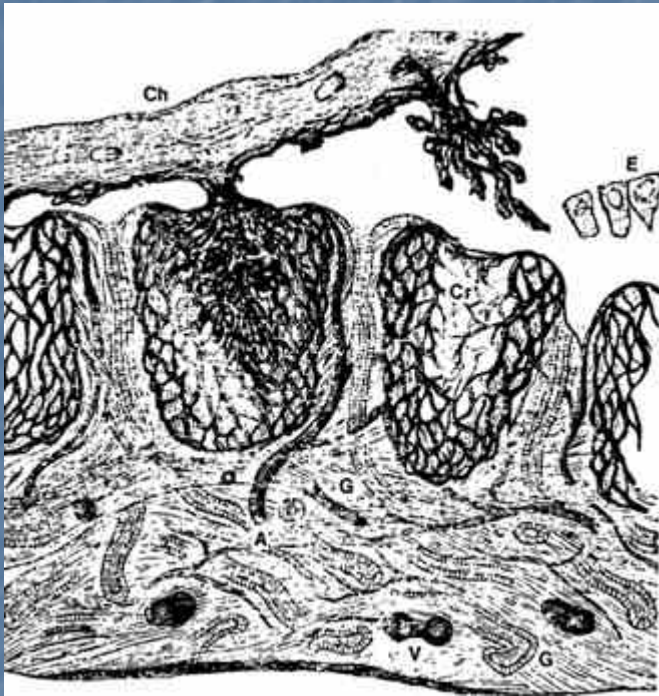


Threats to Fetal Well-being

Lack of O₂ Delivery

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

Historical Investigations of the Equine Placenta

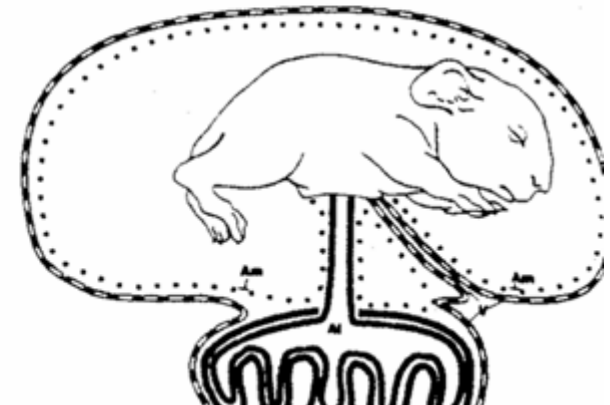
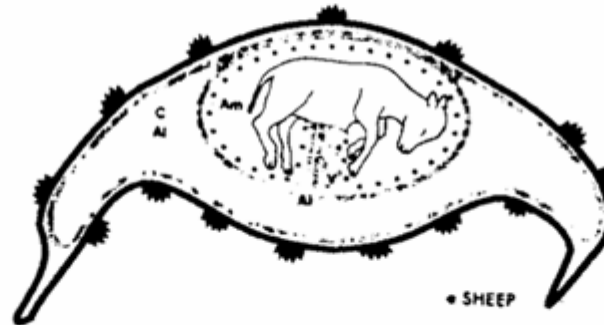
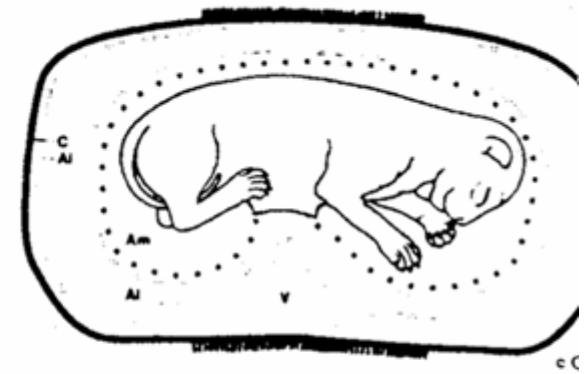
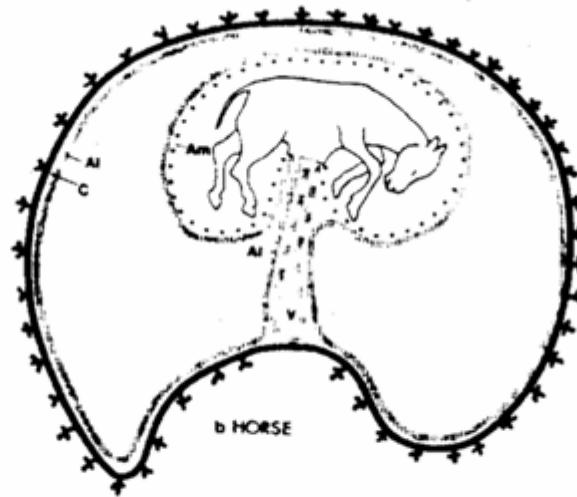
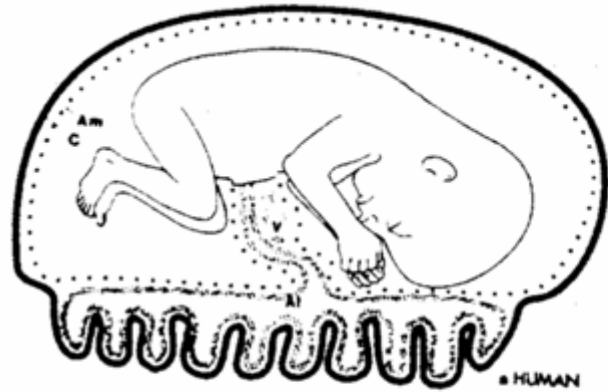


From: Turner (1876)
Lectures on the Comparative
Anatomy of the Placenta

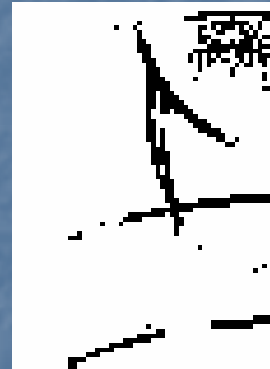
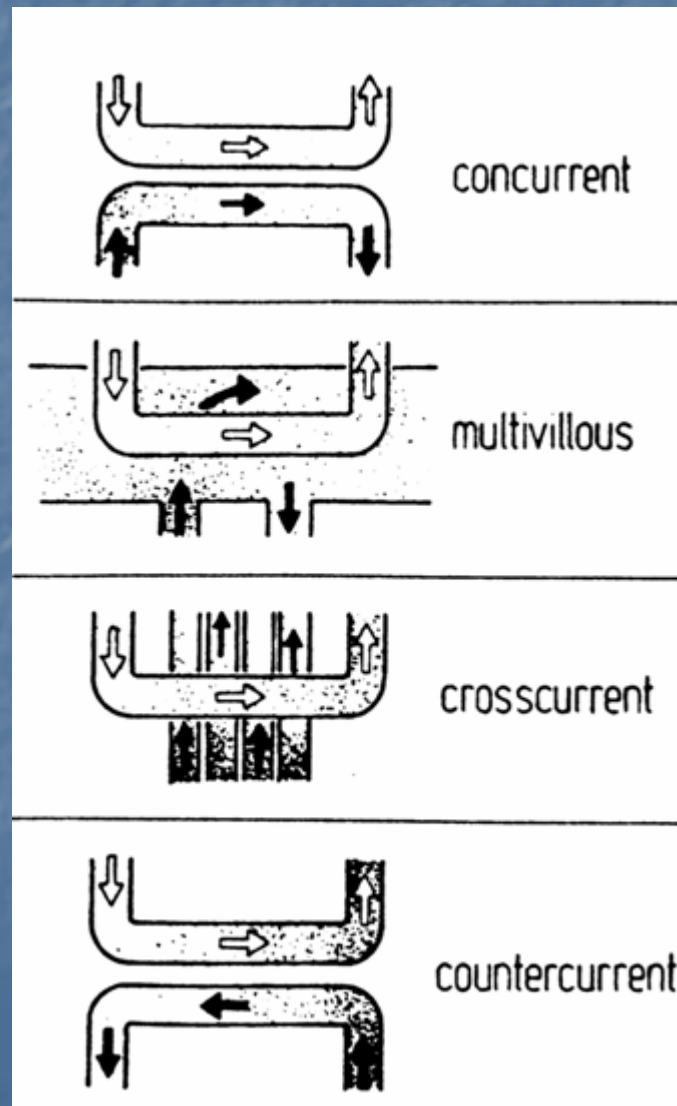


From: Ruini (1598) Anatomia del Cavallo

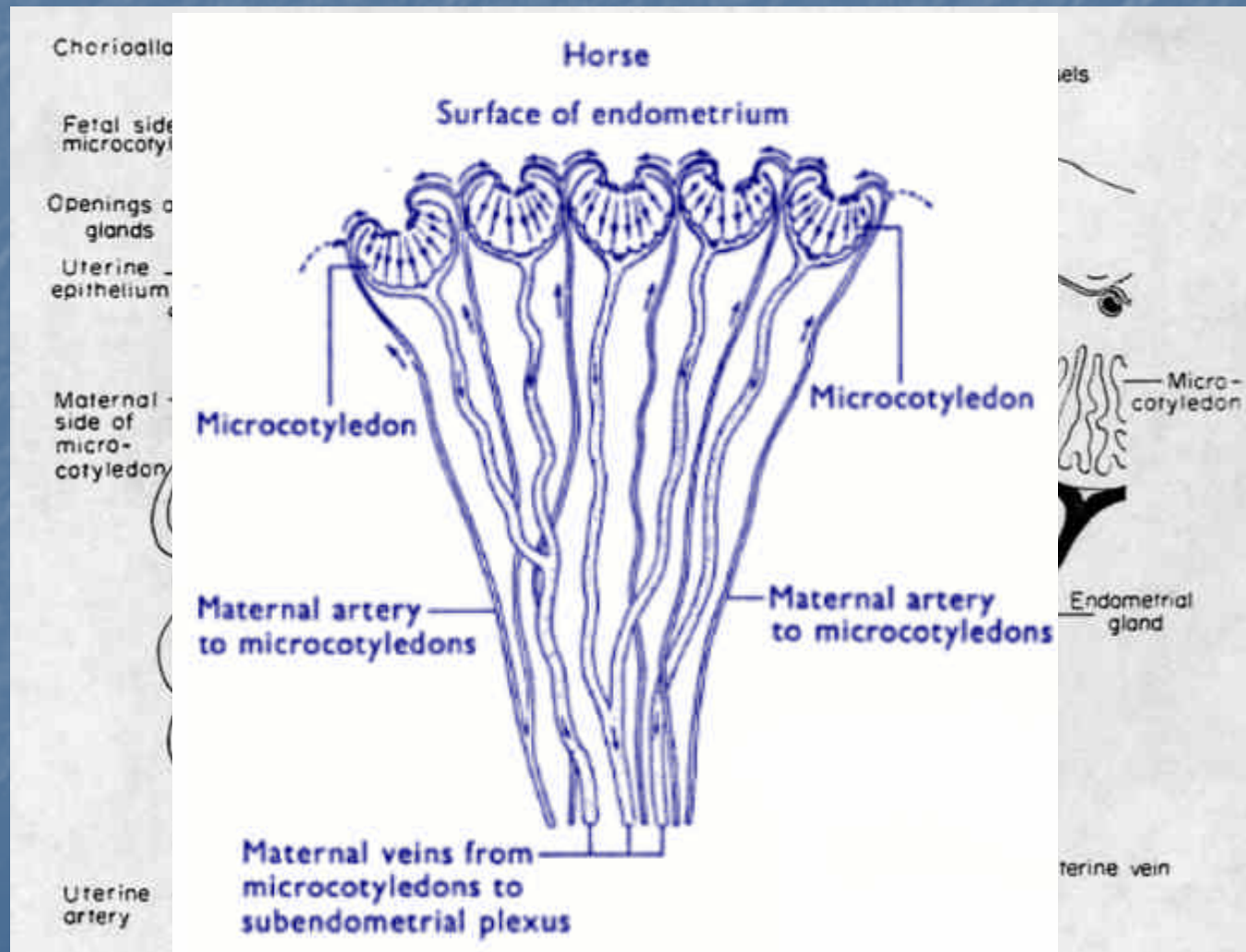
Placentation



Placental Circulation



Equine Placentation



Effect of Maternal Oxygen Therapy

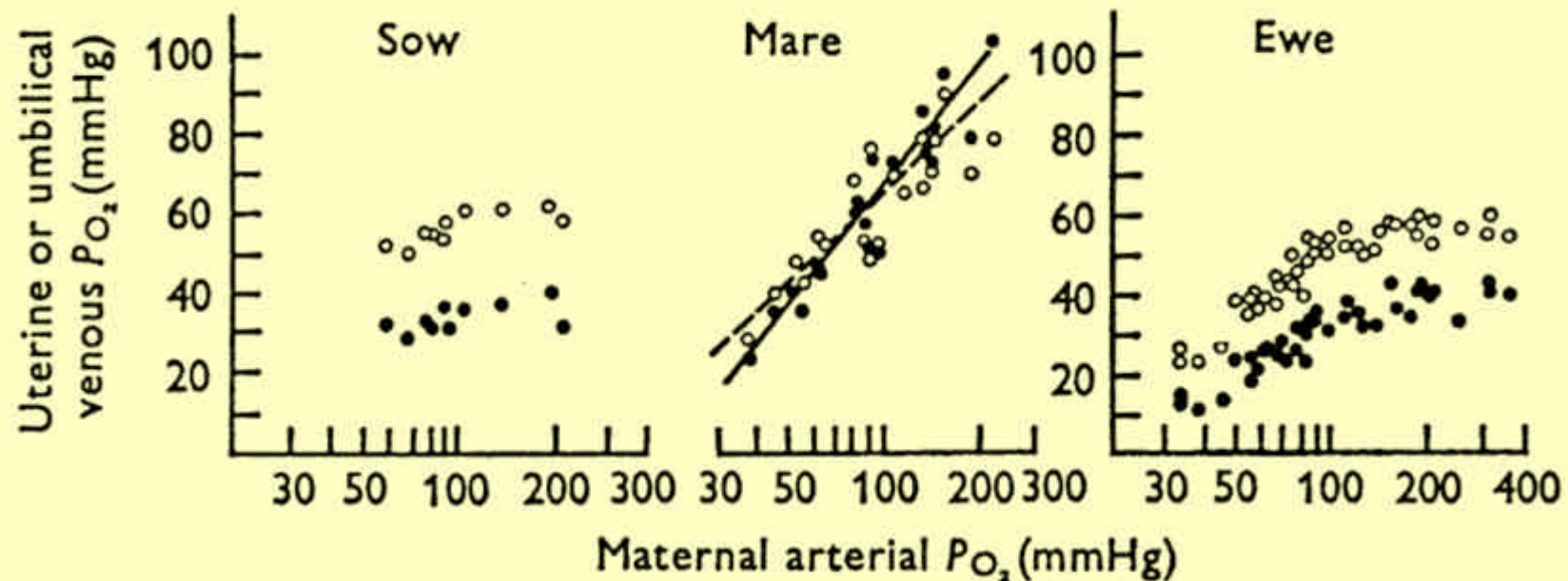
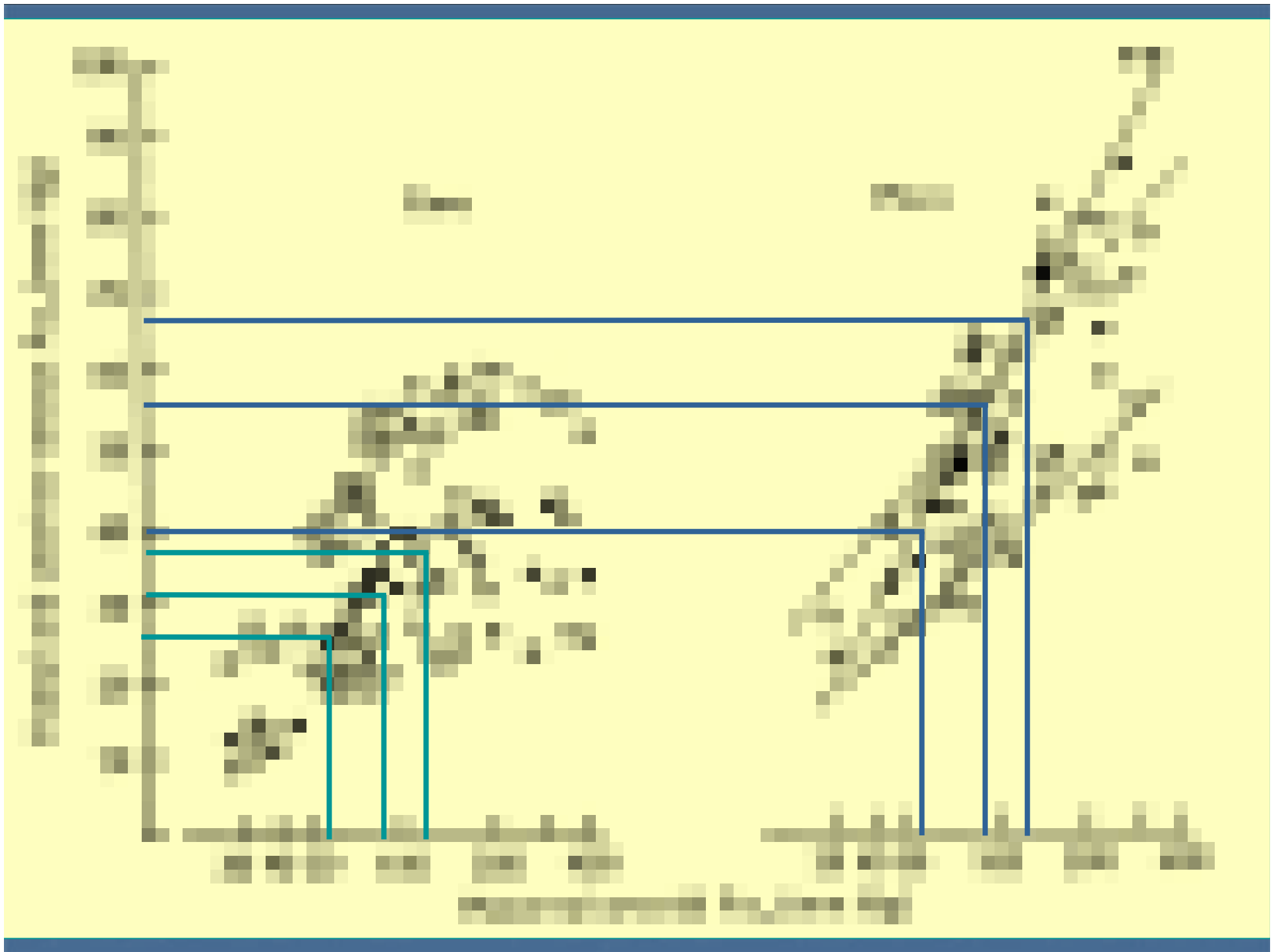


Fig. 4. The relationship between P_{O_2} in maternal arterial blood (log scale) and that in the uterine vein (\bigcirc) and umbilical vein (\bullet) in seven ewes and seven mares (data from Comline & Silver 1970b), and in five sows.



Effect of Placental Circulation Pattern on fetal PO_2

	Counter current circulation	Other circulation
Normal Conditions	PO_2 48-54 torr	PO_2 30-34 torr
Maternal hypoxia	↓↓↓ fetal PO_2	↓ fetal PO_2
↑ Maternal Pao_2	↑↑ fetal PO_2	↑ fetal PO_2

Maternal Oxygen Therapy



Fetal Resuscitation

Lack of O₂ Delivery

- Maternal Anemia - blood transfusions
- Fetal hypoxemia - supplement with INO₂
 - Take advantage of the countercurrent system
 - Even if normal Pao₂ in mare, foal may benefit
 - Could be important with placental edema

Threats to Fetal Well-being

Nutritional Threats

- Chronic malnutrition of the dam
 - Lack of intake
 - Malabsorption
 - Tumor cachexia
- Acute fasting of the dam
 - Forced fasting
 - Capricious appetite - late gestation

Placental Functions

Glucose Transport

- Predominant source of energy for fetus
- Fetal:maternal glucose ratio
 - Man & Rabbits 70-80%
 - Horse 50-60%
 - Pigs 40-50%
 - Ruminants 20-30%



Placental Functions

Glucose Utilization

- The placenta
 - Actively metabolic tissue
 - High glucose utilization by placenta in horse
 - Glucose for placenta also comes from fetus
- Maternal distress – less glucose
 - More glucose delivered from fetus
 - Can lead to negative net glucose transport to fetus

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

Fetal Resuscitation

Nutritional Threats

- Support the mare's nutritional needs
 - Enteral supplementation
 - Parenteral supplementation
 - Encourage a high plain of nutrition
- Avoid acute fasting
 - Avoid elective procedures requiring fasting
 - Encourage anorexic late term mares to eat
 - Supplement with intravenous glucose therapy
- Consider flunixin meglumine therapy

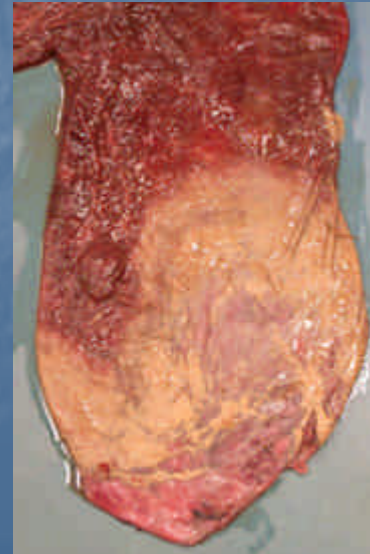
Threats to Fetal Well-being Placentitis/Placental Dysfunction

- Premature placental separation
- Infection
- Inflammation
- Degeneration
- Edema
- Hydrops



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
- NSAID
 - Flunixin meglumine
- Progestin therapy
 - Altrenogest (ReguMate)
- Oxygen therapy
- Vitamin E
- Pentoxifylline

Placentitis – Therapy

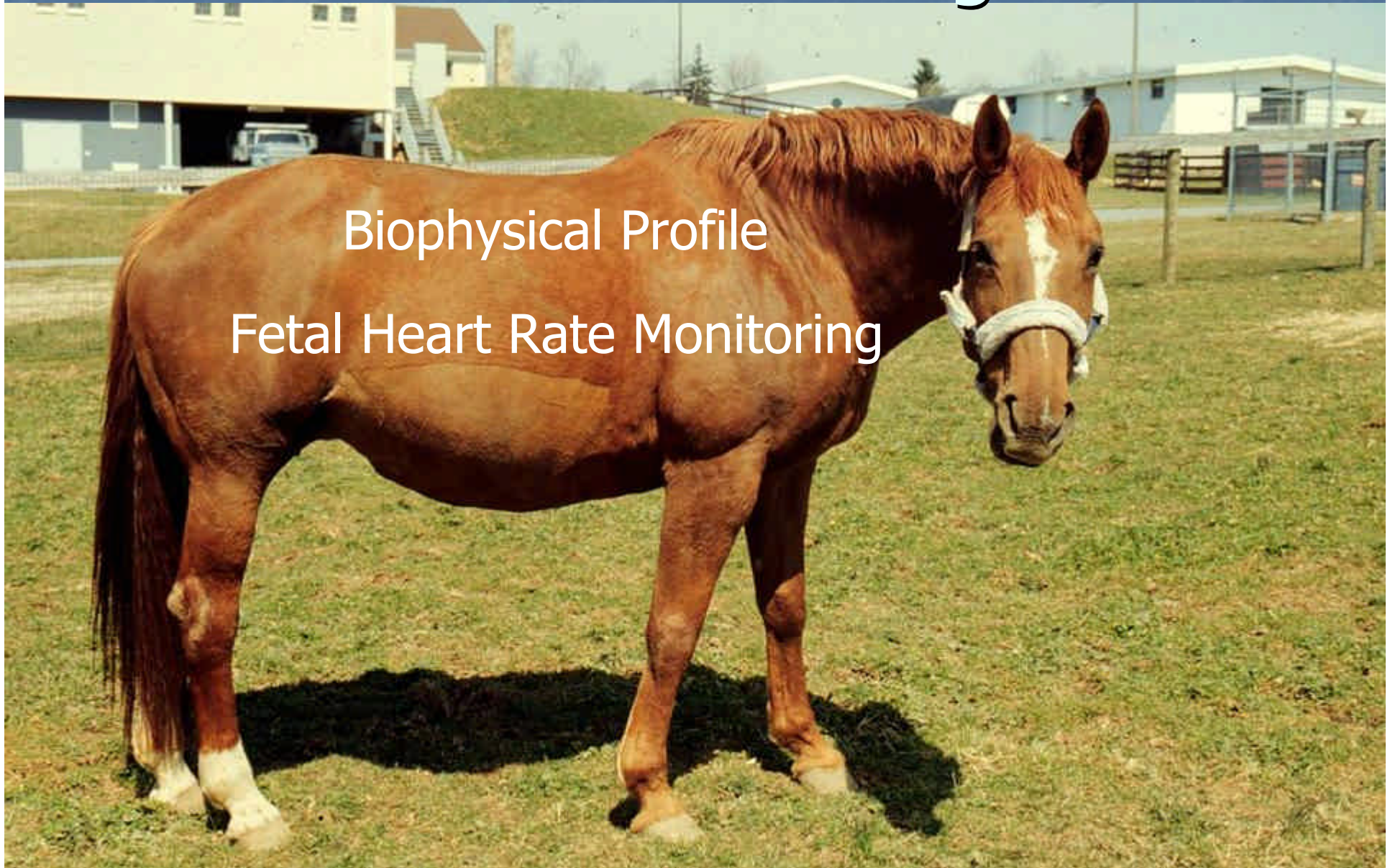
What Protects the Neonate?

- Antimicrobials
 - $p = 0.002$, OR 0.04
- NSAID
 - $p = 0.004$, OR 0.03
- Progestin therapy
 - $p = 0.003$, OR 0.03

Fetal Monitoring

Biophysical Profile

Fetal Heart Rate Monitoring



Early Udder Development Precocious Lactation

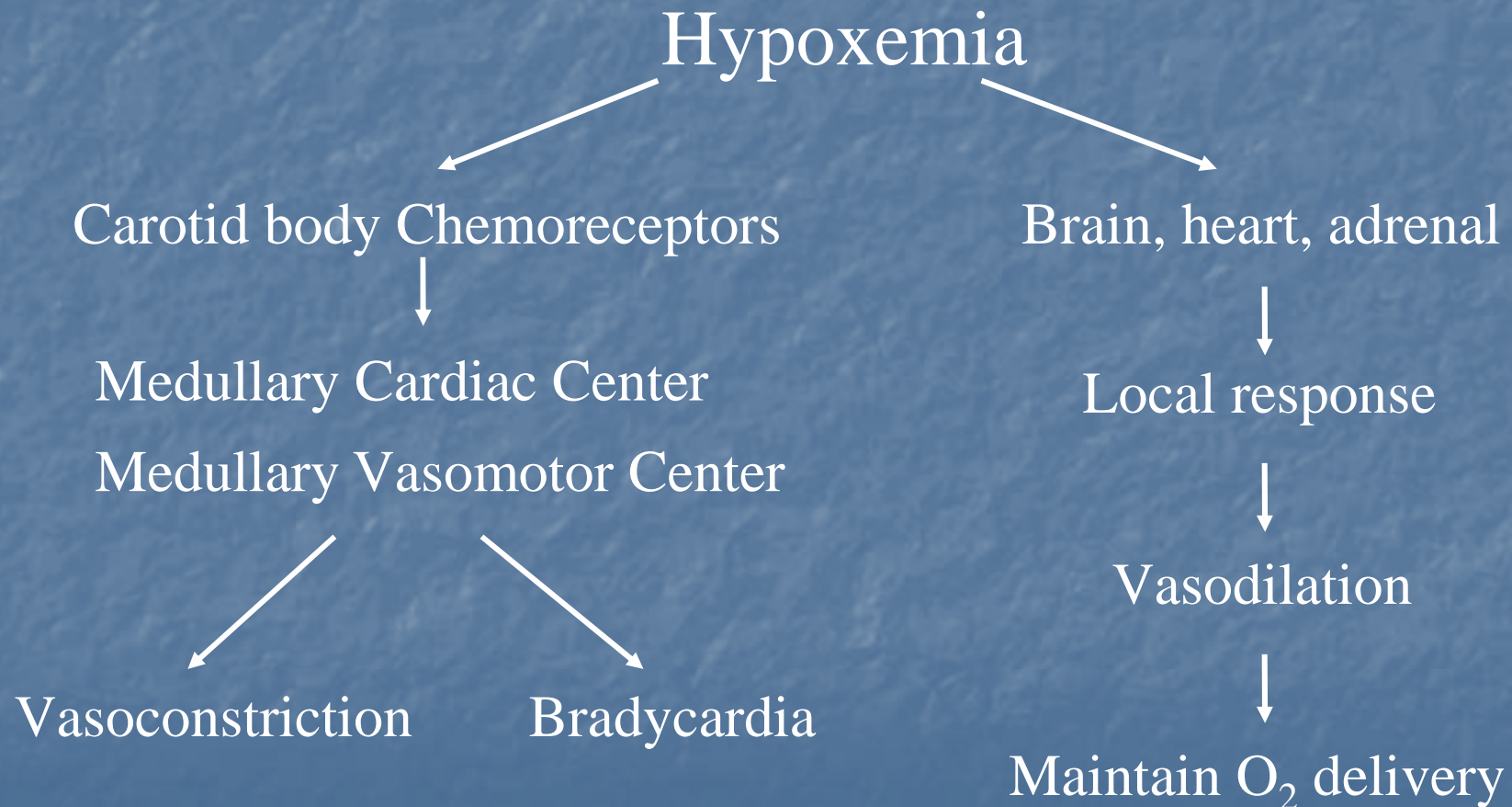
Most reliable sign of fetal distress



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 Response to Hypoxia



Fetal Adaptation to Hypoxia/Stress

- Other changes
 - Decreased activity
 - Fetal activity stops
 - Fetal breathing stops
 - Fetal swallowing stops
 - Decreased growth
 - 50% O₂ consumption

Fetal Physiologic Response to Hypoxia Ischemia

- Insult becomes severe
 - Peripheral tissues prolonged hypoxia
 - 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

Fetal Response Hypoxemia

FHR

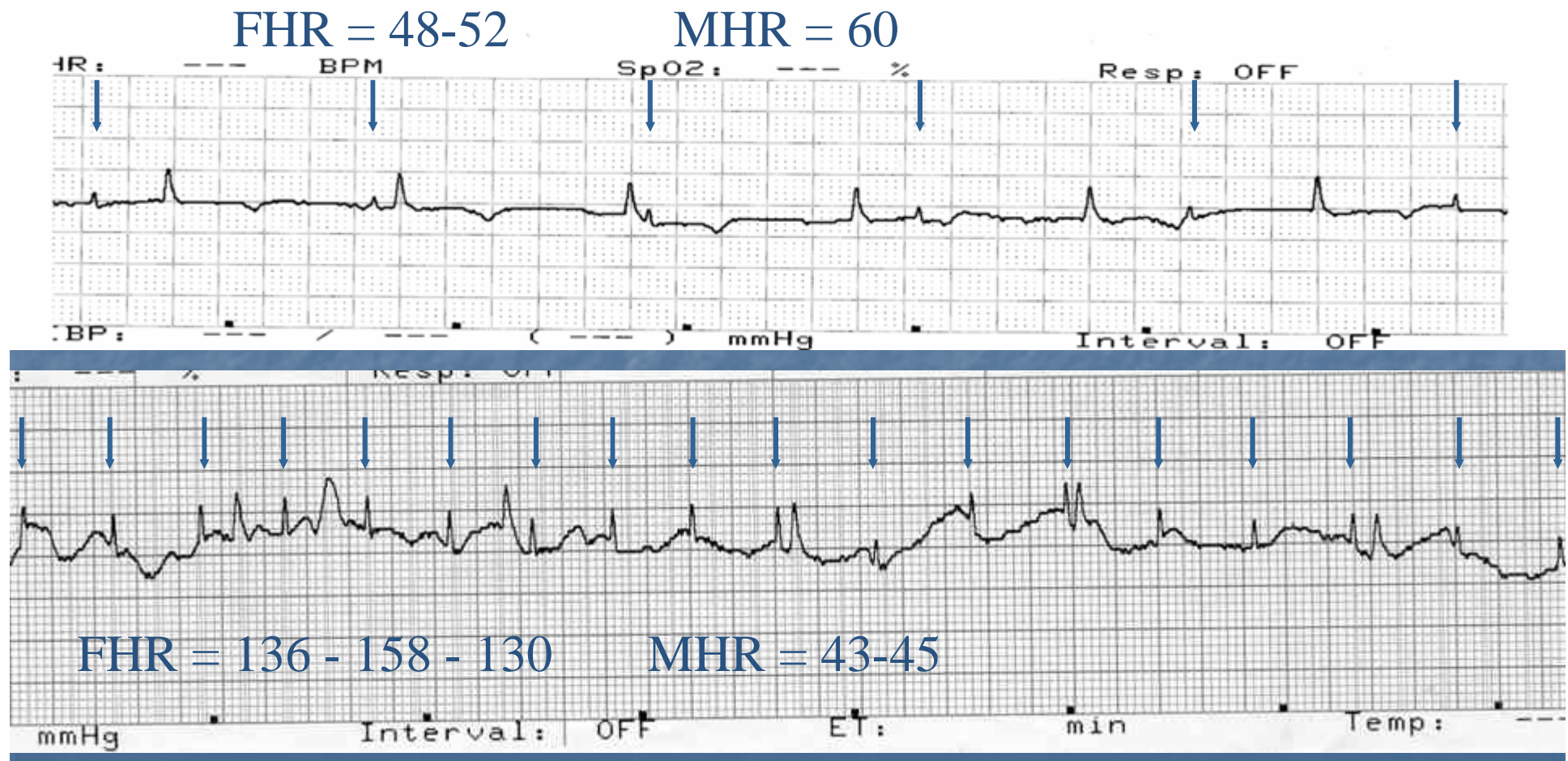
- Hypoxia stressed fetus with compensation
 - Will have slow FHR
 - Will have fewer FHR accelerations
- Decompensation
 - Will lose central adrenergic response
 - Develop persistent tachycardia
 - Terminal bradycardia

FHR Monitoring Technique



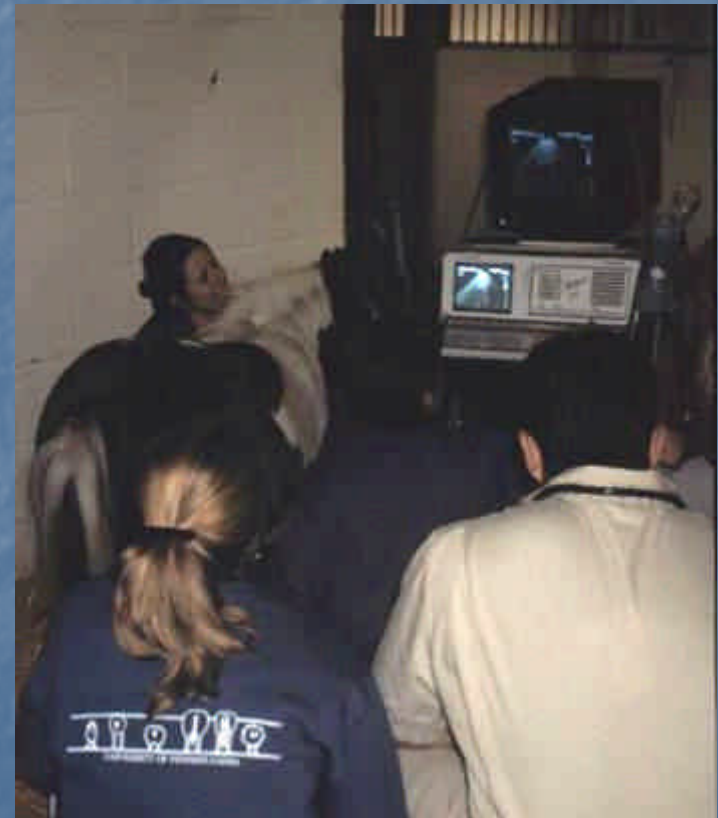
Fetal heart rate measurements

Fetal ECG



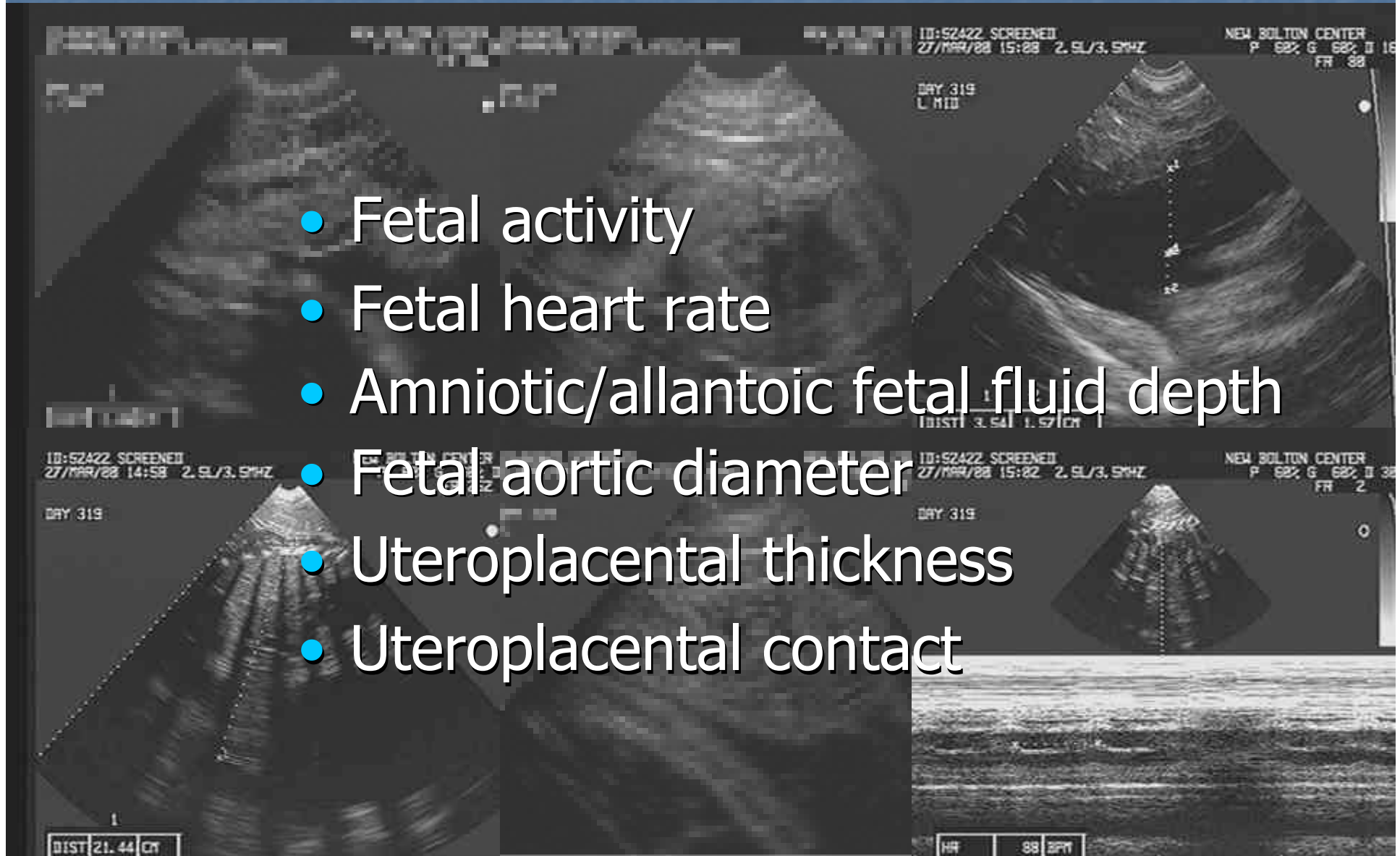
Fetal Foal Transabdominal Ultrasound

- Useful morphometric variables
 - Fetal aortic diameter
 - Fetal breathing movements
 - Fetal activity
 - Fetal tone
 - Fetal fluid depths
- Survey for placental abnormalities
 - Uteroplacental thickness
 - Uteroplacental integrity
- Note fetal positioning



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
- **Treatment** Can make the difference
- Can make the difference
- Resuscitation during parturition
- Use capnograph
- Expect initial poor lung perfusion
- No going back





Neonatal Problems





The High Risk Pregnancy Mare

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Gravid dams are considered at high-risk of a poor outcome to their gestation when they have a history of problems during past pregnancies or have developed a new problem during the current pregnancy. Problems in past gestations most often resulting in classification as a high-risk pregnancy 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 often resulting in classification of high-risk pregnancy 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 possible problems leading to risk of gestational 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 in response 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 devastating results.

Threats to fetal well-being which I will discuss 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 with the remainder directed to the skin, GI tract and kidneys to compensate for the increased demands of pregnancy. 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, as noted above, maternal anemia or maternal hypoxemia secondary to poor pulmonary gas exchange. Survival of the fetus depends on efficient oxygen transport which is determined by unique aspects of placentation in combination with placental oxygen transport mechanisms. Placental gas transport is completely flow dependent and not significantly affected by factors affecting diffusion. There is no significant loss of transport in the face of a diffusion barrier such as edema until flow patterns are affected. It is the flow

pattern of maternal and fetal blood as determined by the alignment of placental vessels which 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 P_{aO_2} than other placental blood flow patterns found in most species. Similarly, transport of other nutrients and removal of waste products occurs to a greater extent because of the juxtaposition of the vessels. But despite these transport advantages there are negative consequences of countercurrent flow pattern. Maternal P_{aO_2} has a dominant effect on fetal P_{O_2} . Changes in maternal P_{aO_2} significantly change fetal P_{O_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 P_{O_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 P_{aO_2} resulting in more oxygen transport. This can easily be done by placing the mare on intranasal oxygen. Even if the mare's P_{aO_2} on room air is normal, the increase (usually to 115-125 torr with an intranasal oxygen flow rate of 10-15 lpm) 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. However, giving blood transfusions to a brood mare may predispose her to produce antibodies against blood groups resulting in 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 (because of lack of opportunity), 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 for elective surgical procedures or when the mare has colic or because of a capricious appetite as is common in late gestation 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 responses 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. It is important that late term colic mares denied oral nutrition be supplemented with glucose in their fluids but care needs to be taken in delivery rate and blood glucose monitored since late term mare's have poor tolerance to intravenous glucose. When periodically anorexic mares are refractory to encouragement to eat, treatment with flunixin meglumine (0.25 mg/kg TID) 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. Infectious placentitis is often caused by ascending bacterial or fungal pathogens but can involve hematogenous spread of viral, bacterial, ehrlichial or fungal pathogens. Percentage of placenta affected by inflammation is not a predictor of the fetal insult. A foal born with widespread placental lesions may be better off than a foal with a focal placental lesion. The presence of placentitis, no matter how

extensive, is enough to predict a serious problem. Approximately 80% of foals born with placentitis are abnormal. 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. Normal timing of parturition is decided cooperatively between the fetus, the mare and the placenta. There are important maternal events, fetal events and placental events which must occur in preparation for parturition. There is a dynamic interaction between these three distinct forces. Loss of coordination will result in a premature foal or a dysmature foal or a postmature foal.

There are a large number of possible iatrogenic mishaps which may affect the outcome of pregnancy. The major one is poor timing of induction of delivery. This may occur when delivery is timed based on the calendar or convenience. It may also occur when delivery is timed based on emergency considerations for the mare such as a gastrointestinal accident requiring euthanasia. Maternal drug therapy can affect the fetus in a variety of ways. Tranquilizers and analgesics such as detomidine or butorphanol have immediate (seconds) and profound effects on the fetal cardiovascular system. Although drugs clearly indicated for the mare should be given, their effects on the foal and possible alternative should be considered.

The mare is somewhat unique in her inability to support multiple fetuses. The reason for this is not entirely clear. It is often stated that this is because of lack of ability to support more than one fetus. Other species solve this problem by splitting resources between smaller twins and indeed, in most cases the sum of the weight of full term foal twins usually equals that expected from a singleton from that mare. Equine twins compete with each other in a manner detrimental

probably because of inflammation associated with competing aggressive placentation. One twin suffering from fetal distress may initiate early parturition. The presence of twins increases the risk of dystocia.

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.

Taking advantage of these fetal physiologic responses, Manning, a pioneer in the area, developed a Fetal Biophysical Profile scoring system for the human fetus in 1980 to predict the presence or absence of fetal asphyxia. The profile used in human medicine consists of 4 ultrasound derived observations (presence of fetal breathing movements, gross body movements, general body tone, and amniotic fluid volume) and result of fetal heart rate responses to fetal movements (nonstress test). In the 25 years since it was developed, evaluation of over 150,000 tests have validated this scoring system. A normal score is a reliable indicator that the fetus is unlikely to die during the 7 days after the test.

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 utilized. The skilled ultrasonographer can obtain 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 efficiently learned through apprenticeship and experience rather than a science that can easily be reproduced by rote.

Attempts have been made to produce an equine biophysical profile. Unfortunately, the predictive value of a normal test is not as uniformly accurate as it is in human medicine and clinical experience seems to indicate that the predictive value of an abnormal test falls short of the ideal. The most recent version of the equine biophysical profile includes transabdominal ultrasound measurements of the fetal aortic diameter in relationship to the mare's weight, uteroplacental thickness, maximum amniotic and allantoic fetal fluid depth, uteroplacental contact, fetal activity level and heart rate. The lack of sensitivity and specificity of the profile may have to do with the selection of parameters measured. In development of the profile for human fetus is, Manning selected parameters that would reflect an acute hypoxia (fetal movement and tone, fetal heart rate and amniotic fluid volume). The equine biophysical profile has a mix of parameters resulting from acute or chronic hypoxia. The equine biophysical profile shows promise but still requires refinement.

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. Fetal heart rate was first heard and described in 17th-century France and first proposed as a measure fetal distress in the mid-19th century. The first electronic FHR recording was in 1906, the first continuous as fetal ECG in 1958 and in 1960 the first technique for recording equine fetal ECGs was published. Since then there have been numerous publications with occasional reference to relating fetal heart rate patterns to fetal foal 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.

The presence of significant beat-to-beat variation suggests intact sympathetic/parasympathetic tone and central control suggesting normal CNS responsiveness and normal local metabolic environment reflecting fetal health. The disappearance of beat-to-beat variation is an ominous sign. Maternal medications such as detomidine or butorphenol reduce fetal heart rate variability transiently.

Other changes that may be noted on FHR ECG tracings include two distinct fetal patterns associated with twins, changes in complex orientation associated with fetal movements, changes in complex high associated with changes in fetal fluid volumes and fetal arrhythmias. Extrasystoles are an occasional finding which generally have little consequence if they are rare.

The most common fetal arrhythmia is atrial fibrillation. This pattern is usually transient and seen in fetal foals that subsequently have some degree of indications of fetal compromise. Very rarely paroxysmal tachycardia or a wide complex tachycardia may be identified which suggests extreme fetal distress.

Any ECG recorder that can print a tracing can be used to measure FHR. The electrical signal from the fetal heart has a low amplitude necessitating placement of the electrodes as close to the fetus as possible. Electrodes are placed in the pattern that produces the best amplitude in fetal complexes. In general, one electrode is placed in the lumbosacral area of the mare and the other two in the mid flank region. If a single daily observation is made, it should be over a minimum of 10 minutes with periodic recordings. I routinely use a telemetry which allows periodic inspection of the heart rate throughout a 12-hour period while the mare is stabled at night.

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.