Birth Resuscitation

Response to Hypoxia

- Hypoxia in adult
  - CO responsive to tissue O_2 levels
  - Hypoxemia results in
    - Increased CO
    - Increased gas exchange lungs
- Hypoxia in fetus
  - Hypoxemia results in
    - Decreased CO (decrease in HR)
    - Slowing placental perfusion
    - Increase BP
    - Requires intact CNS-adrenergic response

Fetal Adaptation to Hypoxia

- Unlike the lungs, placenta will not deliver more O_2 in response to fetal hypoxemia
- Increase umbilical venous resistance
  - Increases fetal placental surface area
  - Improves maternofetal gas exchange

Fetal Adaptation to Hypoxia

- Fetus/neonate - half of O_2 use is facultative
  - Growth, anabolic processes, thermoregulation
  - Not essential for survival
- Lamb
  - Fetal lamb – 30-40% of O_2 used for growth
  - Neonatal lamb – 30% of O_2 used for growth
- Induced maternal hypoxemia
  - Decrease O_2 delivery by 50%
  - No anaerobic metabolism, no fetal acidosis

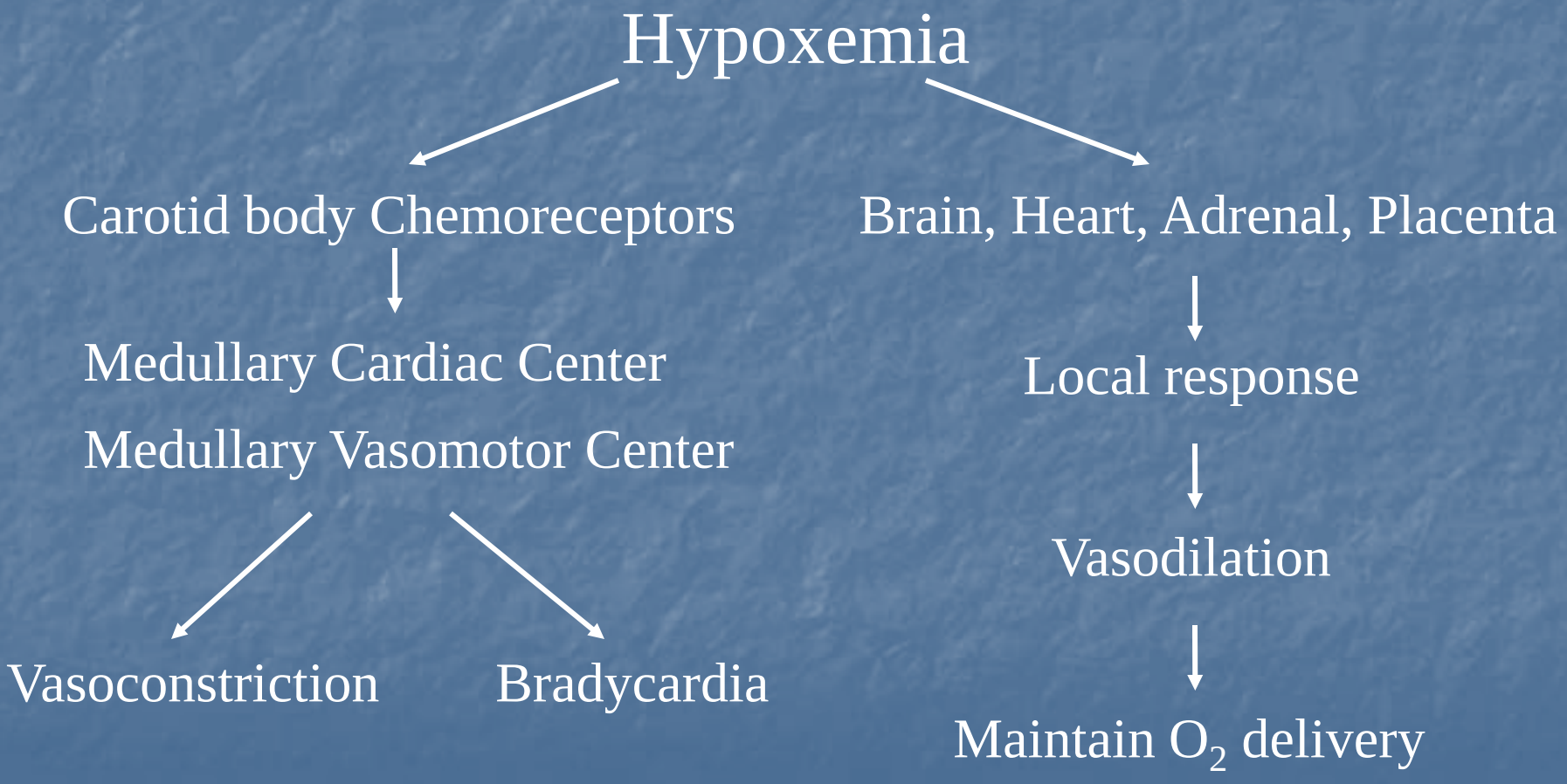
Fetal Adaptation to Hypoxia

- Tolerance to acute hypoxia
  - Turn off growth – how?
  - Decrease unnecessary activity (fetal breathing)
- Tolerance to chronic hypoxia
  - Hypoxia – tolerant cells
  - Postulated to have fewer ion channels
    - Require less energy to prevent Ca leak
- Organs with high metabolic activity
  - Brain, heart
  - Smaller % O_2 used for growth
  - Need to maintain O_2 delivery in face of hypoxemia

Fetal Adaptation to Hypoxia

- Decrease CO (decrease HR)
  - Decrease cardiac O_2 need
  - But increase BP – cardiac work not change
- Redistribution of combined ventricular CO
  - To heart & brain increase from 7% to 25%
  - Increase to adrenal circulation

Fetal Response to Hypoxia



Fetal Response to Hypoxia Blood Flow Redistribution

- % distribution biventricular cardiac output
  - ↓ Gut, spleen, carcass
  - ↑ Brain, heart, adrenals, placenta
- Fetal lamb –↓Po_2 = 12-14 mmHg
  - 4 – 7 X ↑myocardial flow
  - No ↓ O2 delivery
  - Myocardial performance sustained
    - ↑ sympathoadrenal activity
    - ↑ coronary blood flow

Fetal Response Hypoxemia FHR

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

Fetal Cardiac Response to Hypoxic Stress

- Effect of Hypoxia/Acidosis on Cardiac fn

  As long as adrenergic support (CNS)

  Po_2 < 15 , pH normal → normal ventricular fn

  Po_2 normal , pH < 6.8 → normal ventricular fn

  Po_2 < 25 , pH < 6.8 → ↓↓↓ ventricular fn

- Basis of hypoxic ischemic disease

Fetal/neonatal myocardium

- Resistant to hypoxic damage
  - Due to high rate of anaerobic glycolysis
  - Greater glycogen stores
  - Resistance of fetal cells to damage from lactic acidosis
- Resistance to combined hypoxia/hypercapnia
- Resistance to ischemia dysfunction
  - After short periods quickly recovers
  - Will work as efficiently as before insult
- Resistant to ischemic myocardium cell death
- More likely to survive cardiopulmonary arrest

Birth

Birth Transition

Transition begins weeks before birth

Transition days before birth

Transition continues through neonatal period

Vital Transitions

Initiation of Breathing

Triggering the First Breath

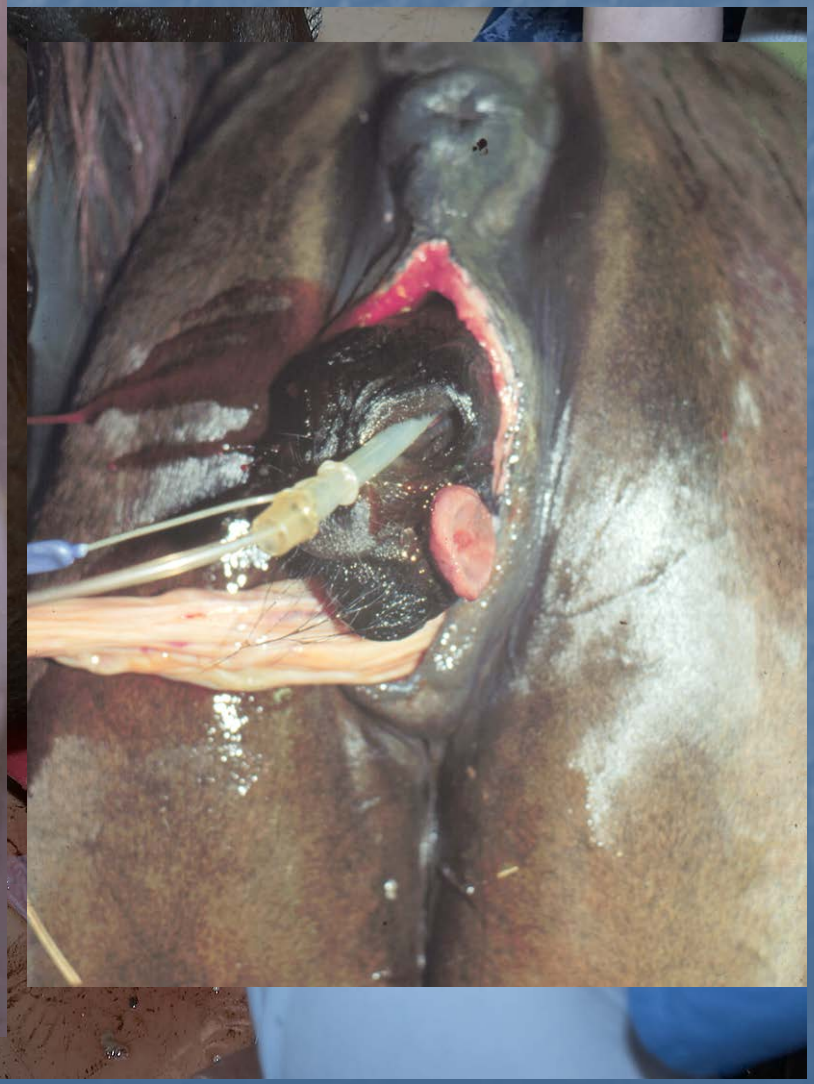
Preparation for Resuscitation

- 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

EXIT

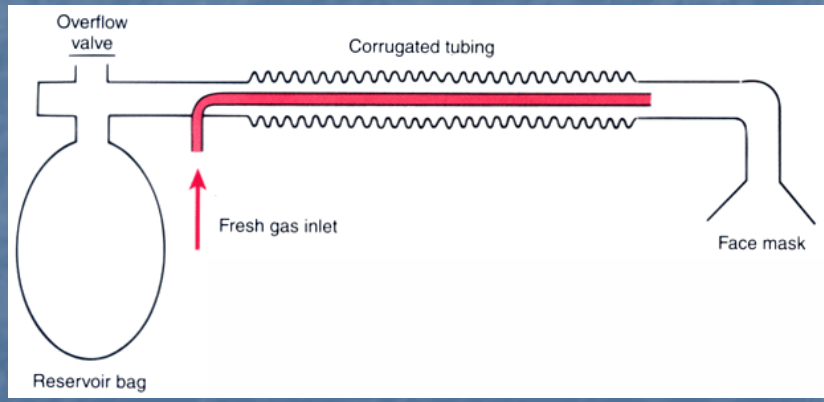
Ex-utero
Intrapartum
Treatment

Development of EXIT



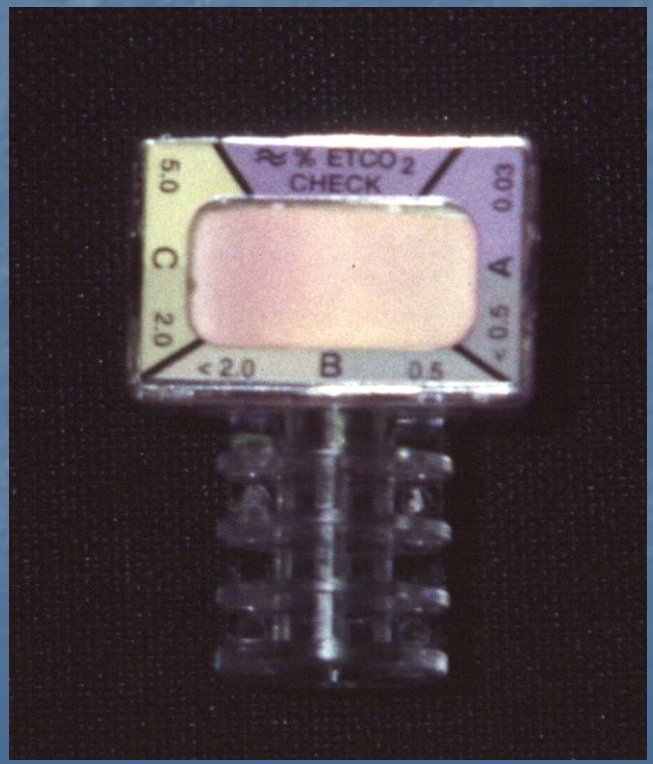
EXIT Technique

- During a dystocia
  - If nares are external
  - If nose in the pelvic canal, palpable
- Intubation attempted
  - Placement checked
  - Bain Circuit
- Ventilation initiated
  - Self inflating bag

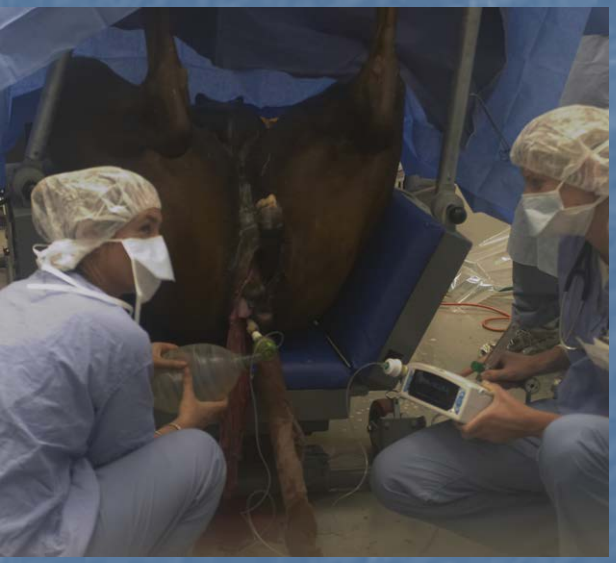


EXIT Technique

- Carbon dioxide detector
  - Capnograph
  - Easy Cap II

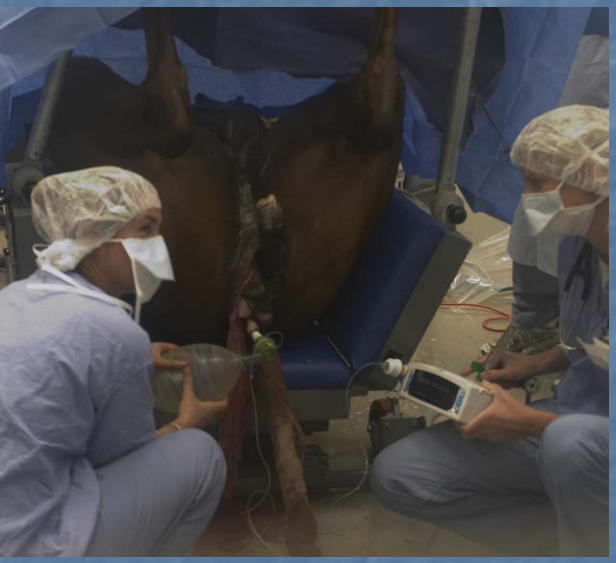


EXIT
Capnography



- Monitor cardiac output
  - $ETCO_2$ correlates with cardiac output
- Ventilation
  - Transition - placental to pulmonary circulation
- Initially $ETCO_2$ low (4 – 8 torr)
  - Residual tissue levels
- $ETCO_2$ levels predict viability
  - Dead fetus
    - Initially 4 – 8 torr
    - Quickly becomes 0
  - Compromised foal
    - 8 to 20 torr
  - Responding to resuscitation
    - 40 to 60 torr

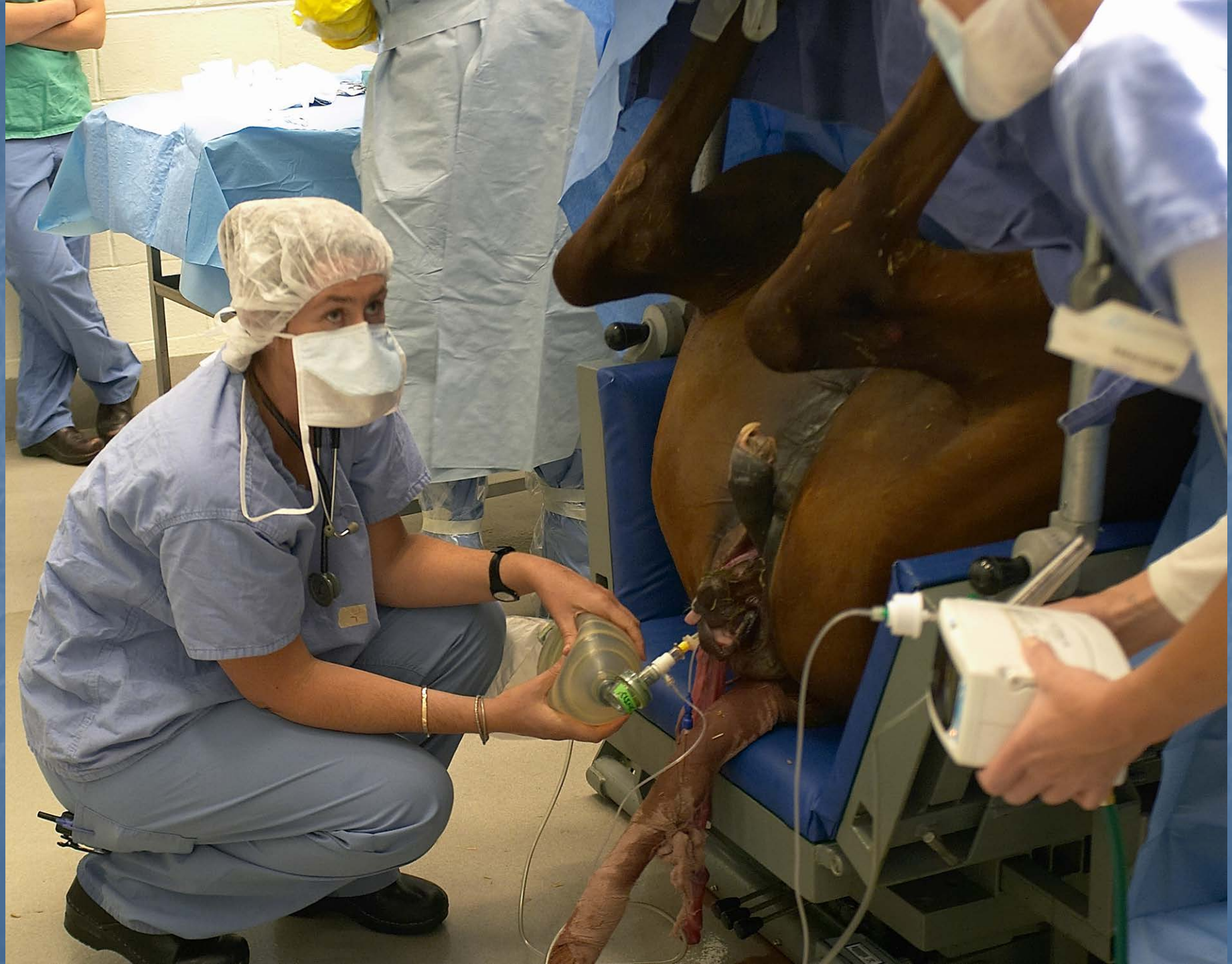
EXIT
Capnography



- Pitfalls
  - Hyperventilate
    - Drops to zero
    - Can be fatal
    - Slow down
  - Esophageal intubation
  - Capnograph malfunction
- When $ETCO_2$ lies
  - Respiratory pump
  - $ETCO_2$ stays low
  - If slow ventilation
    - $ETCO_2$ lower
  - If increase ventilation
    - $ETCO_2$ remains low

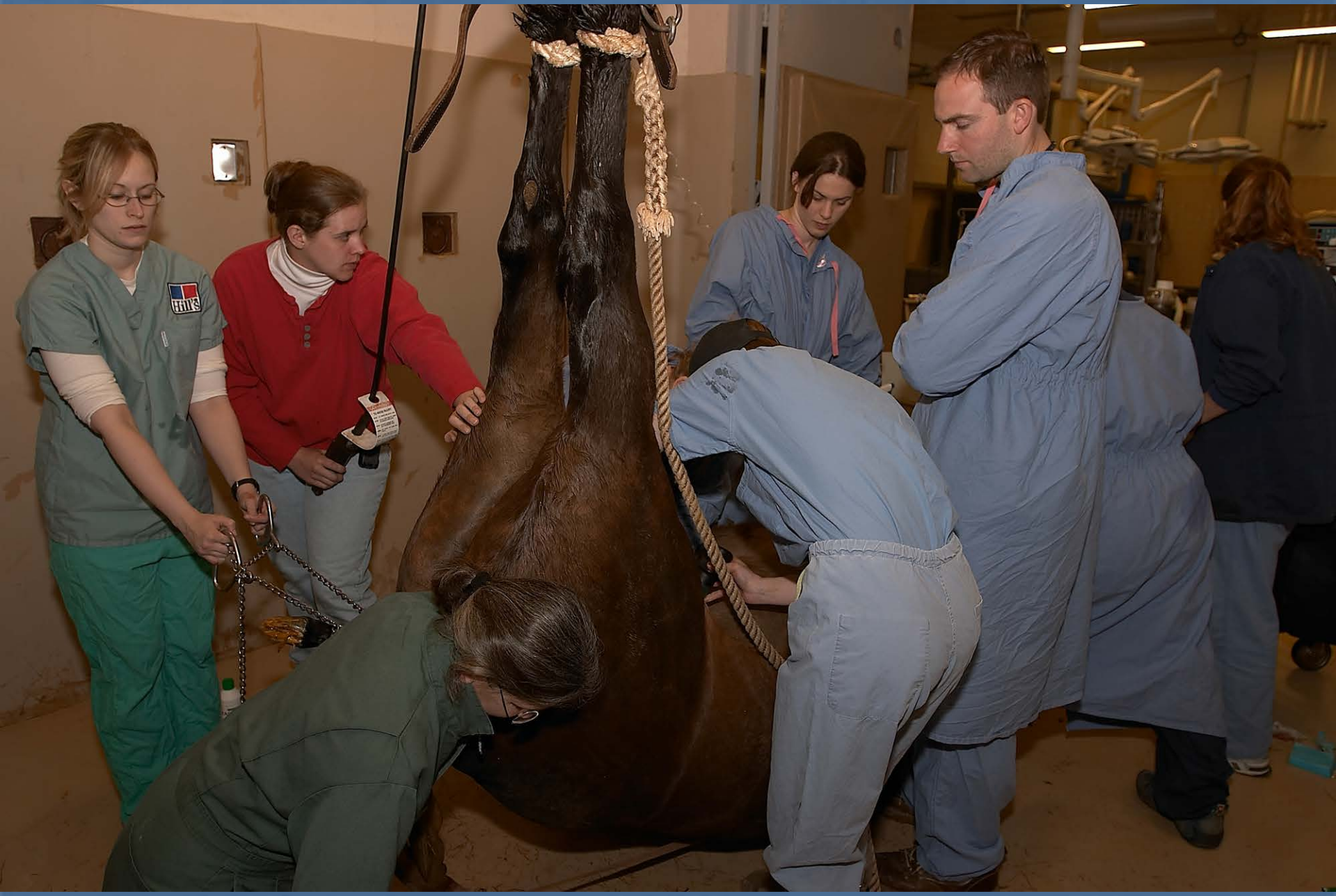
EXIT Technique

- Drug therapy
  - Catheter through endotracheal tube
  - Epinephrine
  - Vasopressin
  - Naloxone
- Pulse/ECG
- Arterial blood gas
  - Lingual artery



EXIT

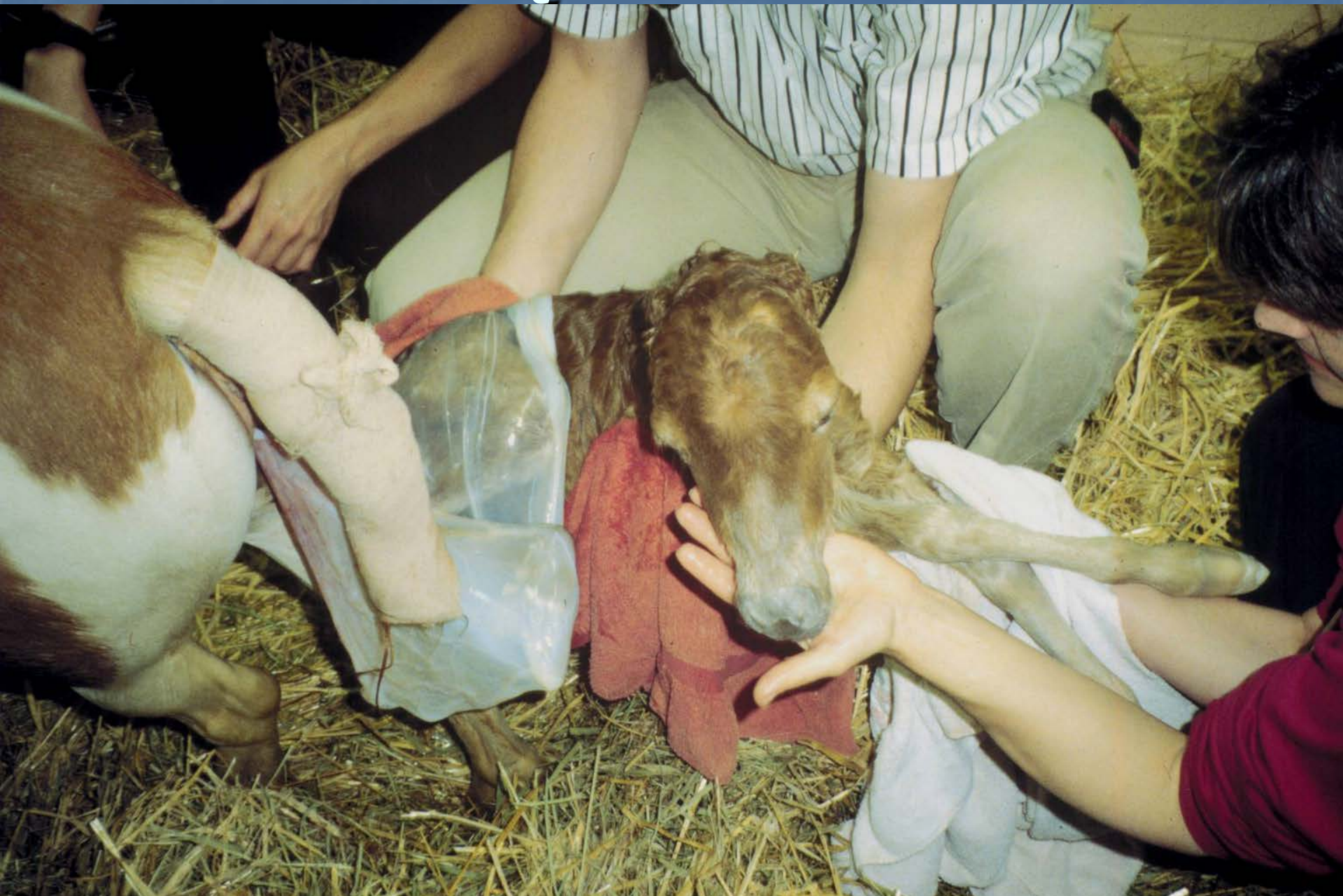
- General anesthesia in mare
- Redirection of placental blood flow to the lungs
- Decrease in placental blood flow
  - Associated with rise in fetal PO_2
  - O_2 Rx
    - Controversial
    - Role in redirection of placental blood flow
      - Favor of its use during EXIT
  - Decrease transfer of anesthetic agents/other drugs
  - Reduction in neonatal depression after delivery



EXIT
Summary

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

Initial Quick Overview



Apgar Score
1, 5, 10 minutes

APGAR Score

Current Researches in Anesthesia and Analgesia—July-August, 1953

A Proposal for a New Method of Evaluation of the Newborn Infant.*

Virginia Apgar, M.D., New York, N. Y.

Department of Anesthesiology, Columbia University, College of Physicians and Surgeons and the Anesthesia Service, The Presbyterian Hospital

RESUSCITATION OF INFANTS at birth has been the subject of many articles. Seldom have there been such imaginative ideas, such enthusiasms, and dislikes, and such unscientific observations and study about one clinical picture. There are outstanding exceptions to these statements, but the poor quality and lack of precise data of the majority of papers concerned with infant resuscitation are interesting.

There are several excellent review articles[1] [2] but the main emphasis in the past has been on treatment of the asphyxiated or apneic newborn infant. The purpose of this paper is the reestablishment of simple, clear classification or "grading" of newborn infants which can be used as a basis for discussion and comparison of the results of obstetric practices, types of maternal pain relief and the effects of resuscitation.

The principle of giving a "score" to a patient as a sum total of several objective findings is not new and has been used recently in judging the treatment of drug addiction.[3] The endpoints which have

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

Clear Airway

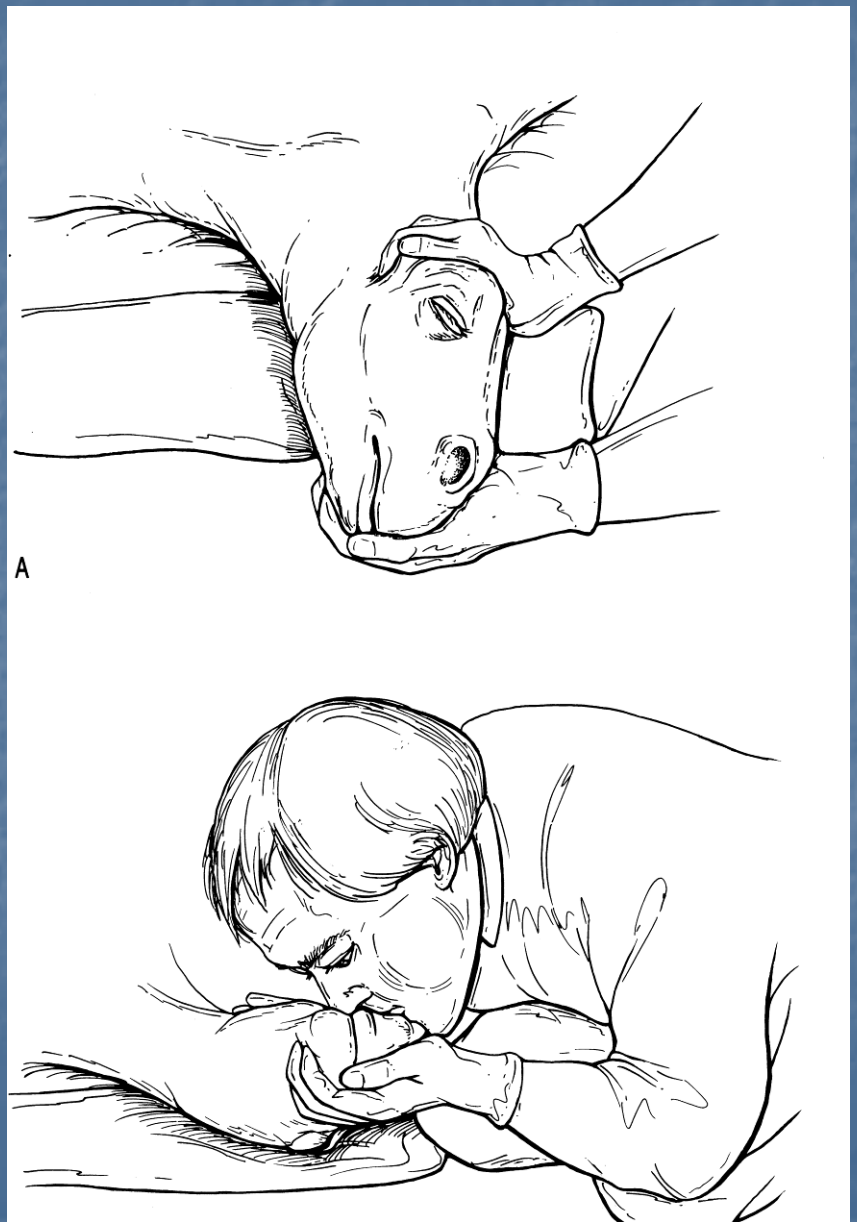
Meconium Aspiration

Tactile stimulation

Free-flow O_2

- Intranasal O_2
- Flow-by O2

Mouth-to-Nose Ventilation



If the neonate does not breath spontaneously


Self-inflating
bag valve
device

Drug-depressed Neonate

- Alpha2-adrenoceptor agonists reversal
  - Atipamezule
  - Yohimbine
  - Not tolazoline
- Diazepam reversal - flumazenil
- Opiate reversal – naloxone
- Volatile anesthetic reversal
  - Ventilation


If early in asphyxia
30 sec 100% O2 will >HR
If late - myocardium failing
Need chest compression

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