Cardiopulmonary Resuscitation

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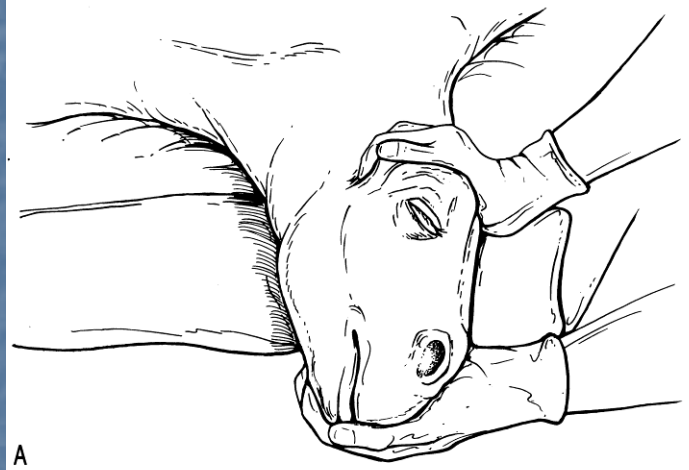
Cardiopulmonary Arrest

Goals

- Primary - ROSC
  - Start the heart
  - Return cardiac perfusion
- Secondary – preserve CNS
  - Maintain perfusion to brain

Cardiopulmonary Arrest

- Ventilation
  - Pulmonary arrest
  - Bradycardia
- Cardiac compression
  - Primary cardiac failure



Cardiopulmonary Resuscitation Ventilation

- Mouth-to-nose ventilation
- Endotracheal tube
  - Self-inflating bag-valve device
- Negative aspects
  - Decreases cardiac return
  - Decrease coronary/cerebral perfusion
- Positive aspects
  - Asphyxial arrest


Self-inflating
bag valve
device

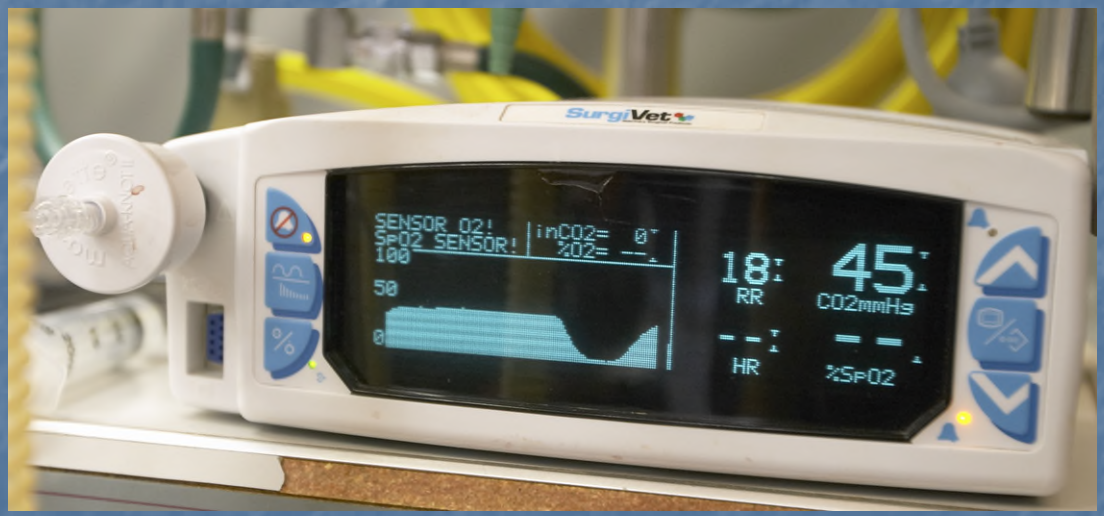
Cardiopulmonary Resuscitation Ventilation

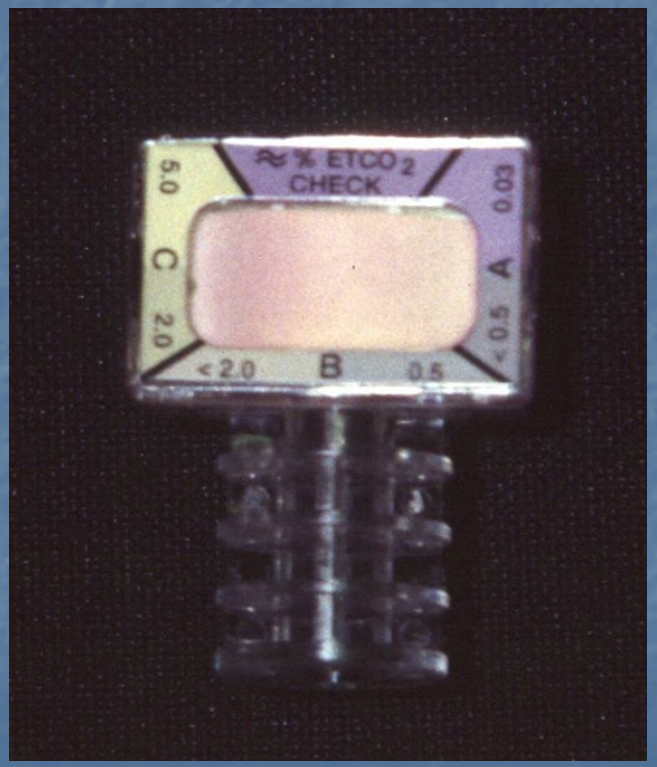
- Rapid infrequent breaths
  - Inspiration < 1 second
  - Rate < 8-10/min
- Goal is not a deep breath
- Observation of chest excursion
- Use of oxygen

Cardiopulmonary Resuscitation
Chest Compressions

- Nonperfusing cardiac rhythm
- Rate 100
  - Complete chest recoil between compressions
- Maximum interruption of 10 sec
- Monitoring
  - Pupil size
  - End-tidal CO_2 ($ETCO_2$)

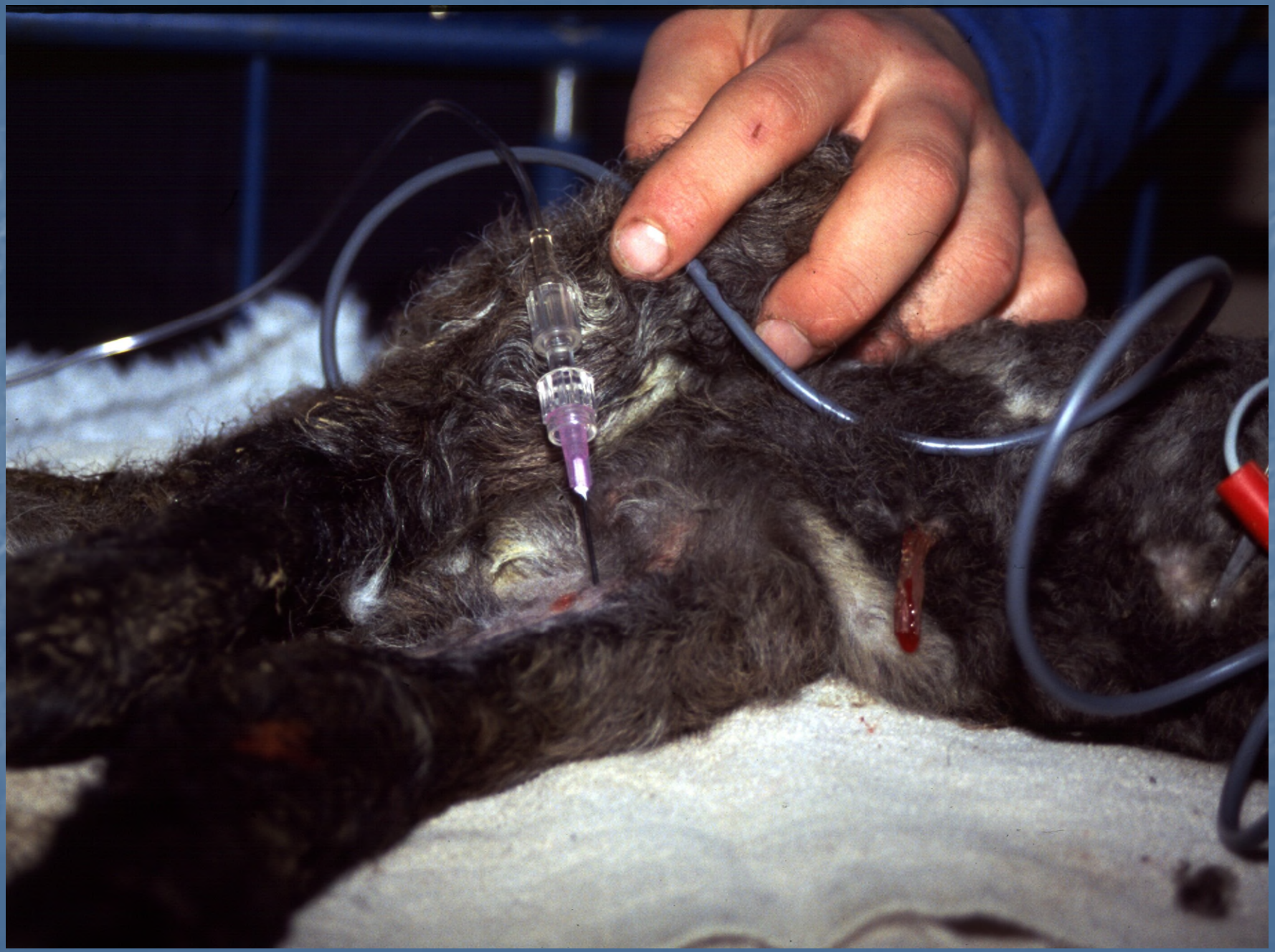
Capnography





Cardiopulmonary Resuscitation Vascular Access

- Jugular vein catheter
- Intratracheal route
- Intraosseous route
  - Special intraosseous device
  - Medial tibial plateau
  - Any drug can be administered

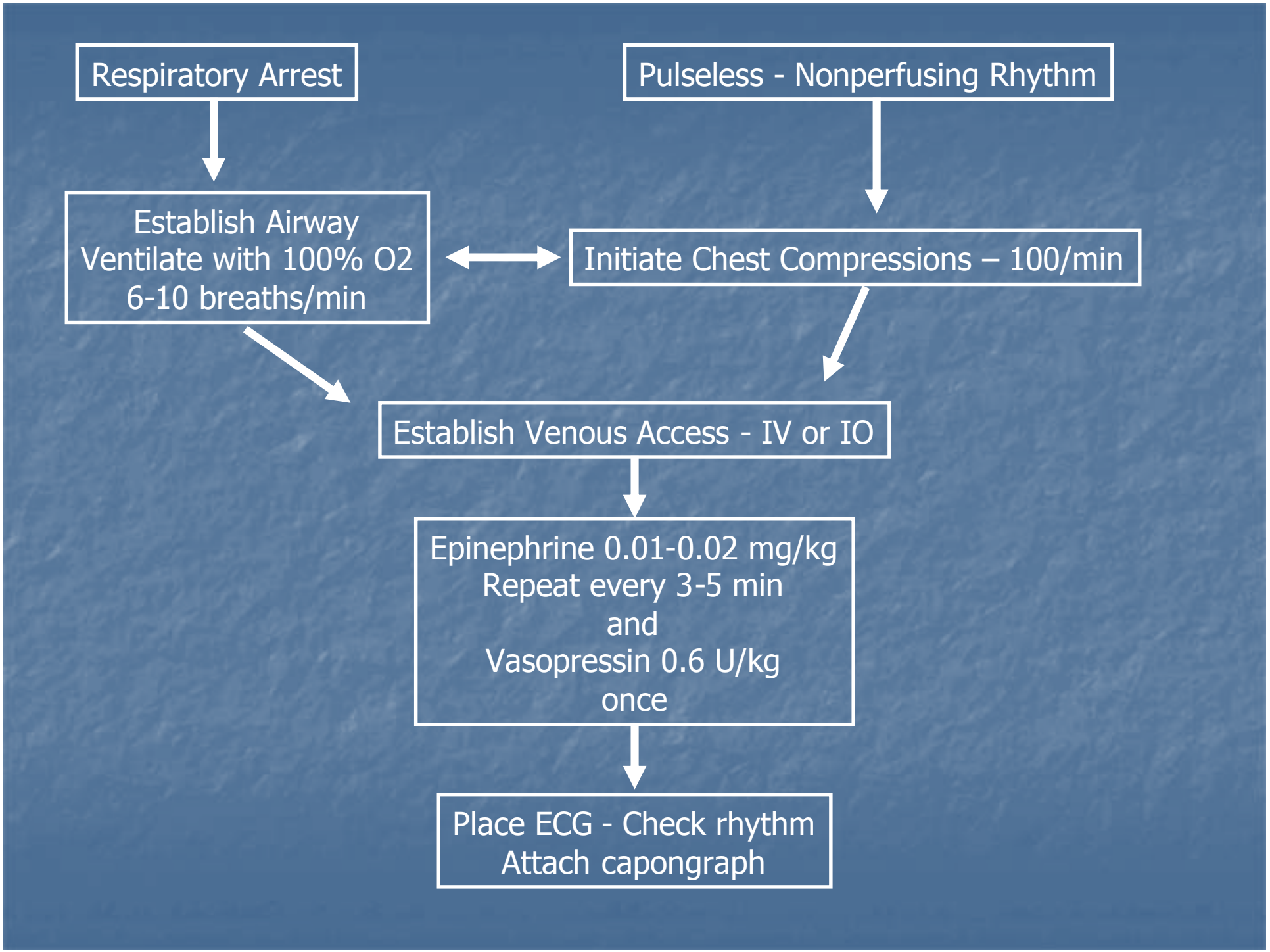


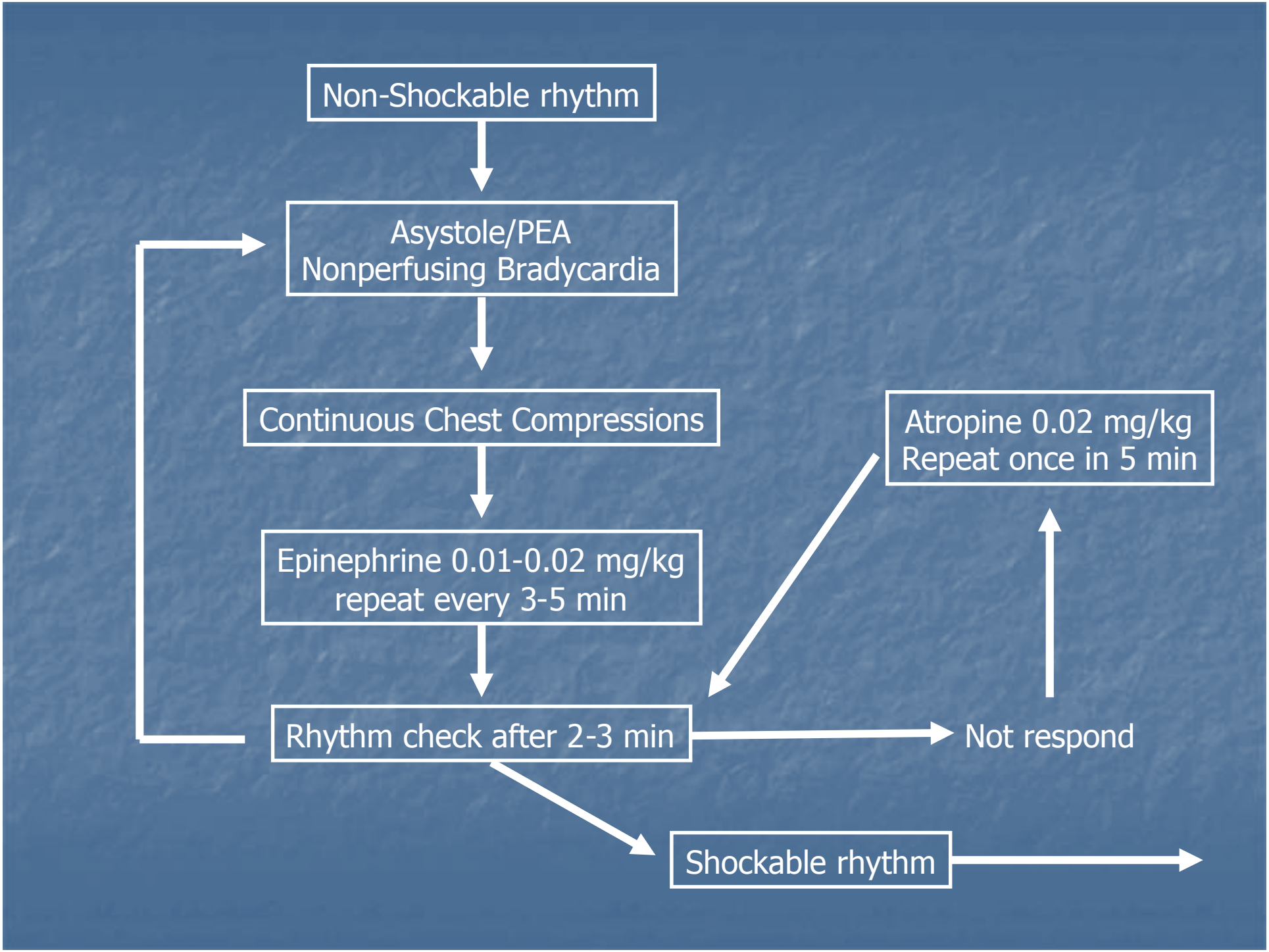
Cardiopulmonary Resuscitation Drug Therapy

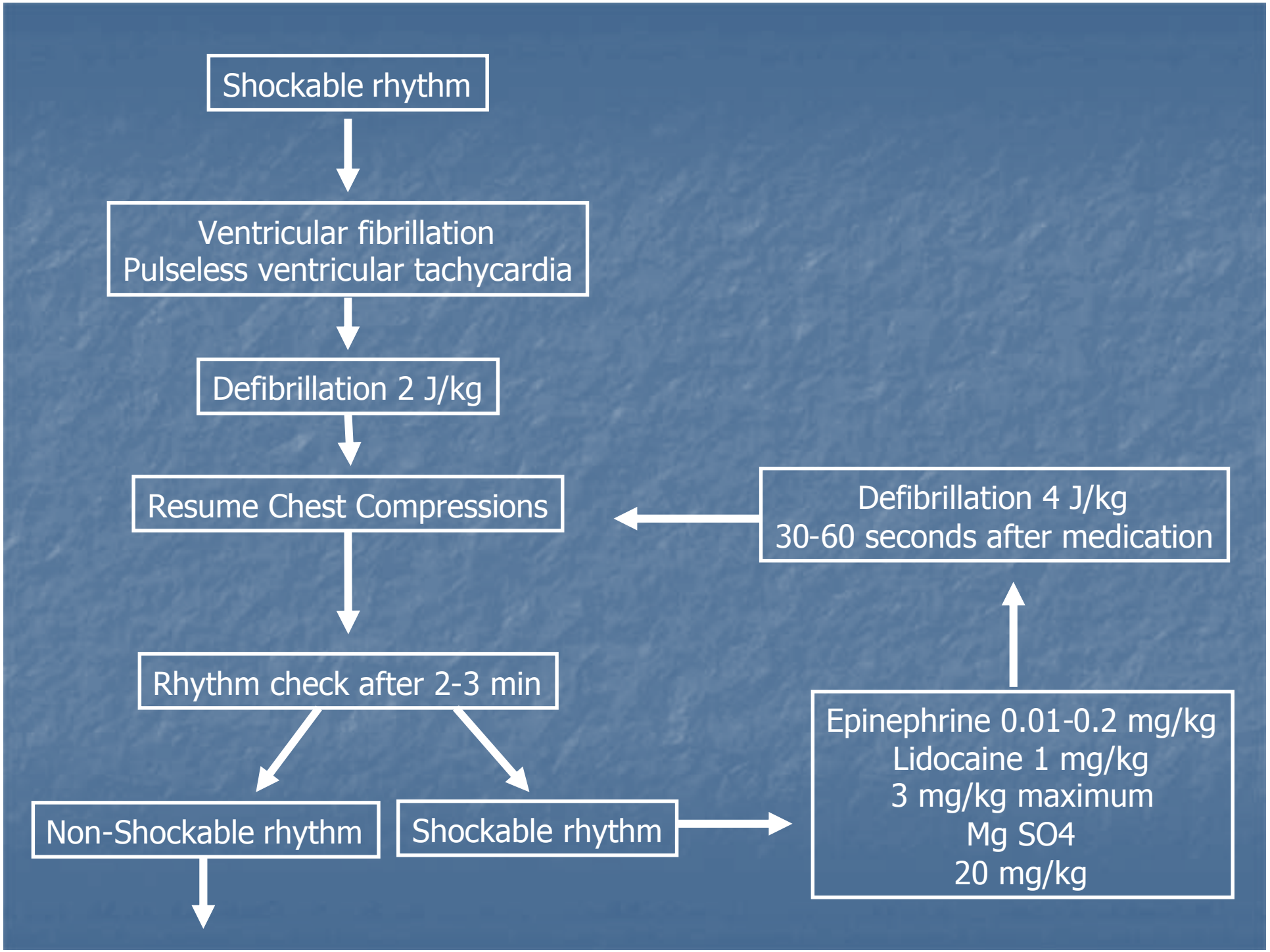
- Epinephrine
- Vasopressin
- Lidocaine
- Atropine
- $MgSO_4$

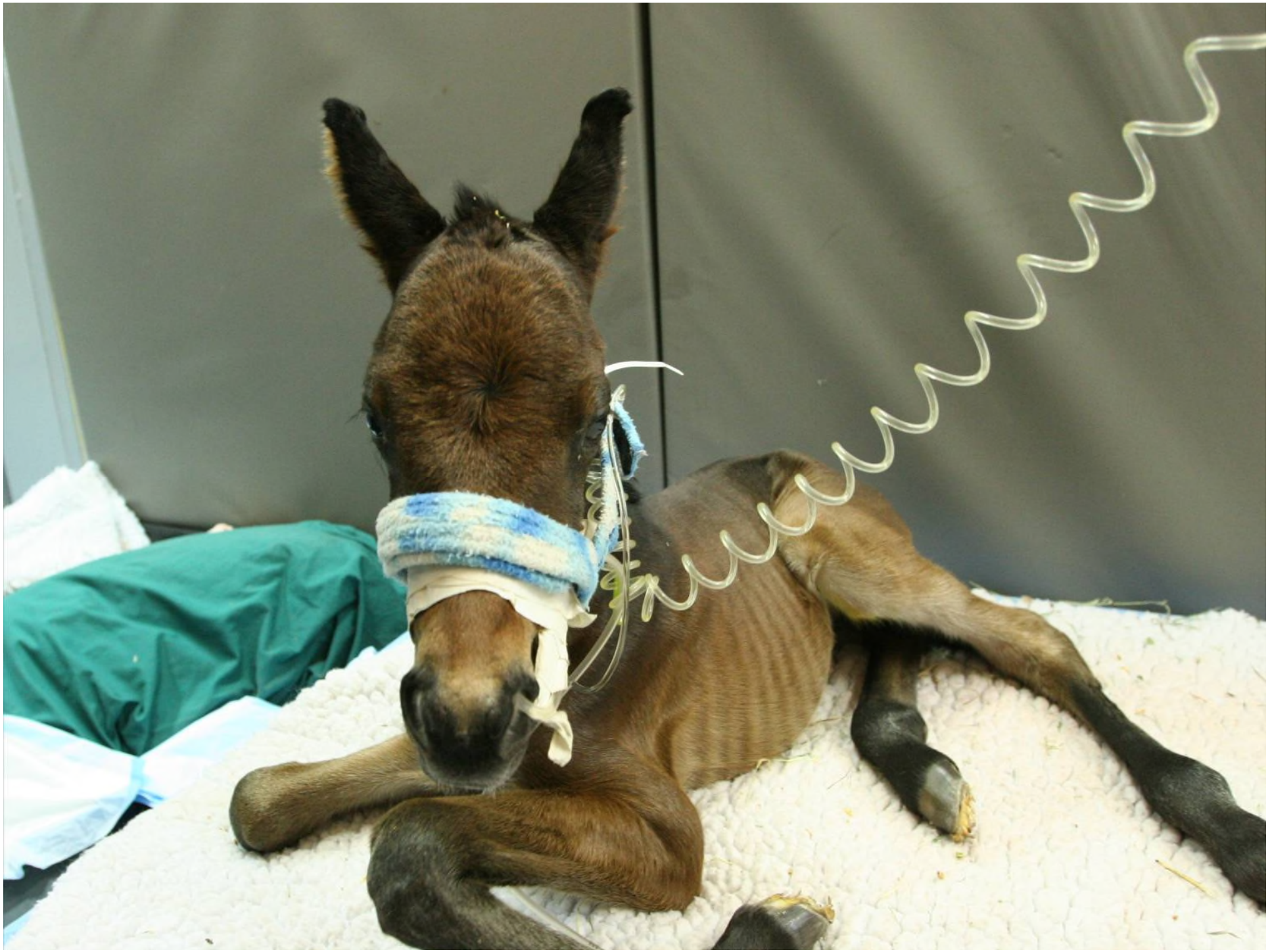
Cardiopulmonary Resuscitation Drugs to Avoid

- Fluids
- Sodium Bicarbonate
- Glucose
- Ca^{++}


Respiratory Arrest
Pulseless - Nonperfusing Rhythm
Establish Airway
Ventilate with 100% O2
6-10 breaths/min
Initiate Chest Compressions – 100/min
Establish Venous Access - IV or IO
Epinephrine 0.01-0.02 mg/kg
Repeat every 3-5 min
and
Vasopressin 0.6 U/kg
once
Place ECG - Check rhythm
Attach capongraph


Non-Shockable rhythm
Asystole/PEA
Nonperfusing Bradycardia
Continuous Chest Compressions
Epinephrine 0.01-0.02 mg/kg
repeat every 3-5 min
Rhythm check after 2-3 min
Atropine 0.02 mg/kg
Repeat once in 5 min
Not respond
Shockable rhythm


Shockable rhythm
Ventricular fibrillation
Pulseless ventricular tachycardia
Defibrillation 2 J/kg
Resume Chest Compressions
Rhythm check after 2-3 min
Non-Shockable rhythm
Shockable rhythm
Epinephrine 0.01-0.2 mg/kg
Lidocaine 1 mg/kg
3 mg/kg maximum
Mg SO4
20 mg/kg
Defibrillation 4 J/kg
30-60 seconds after medication

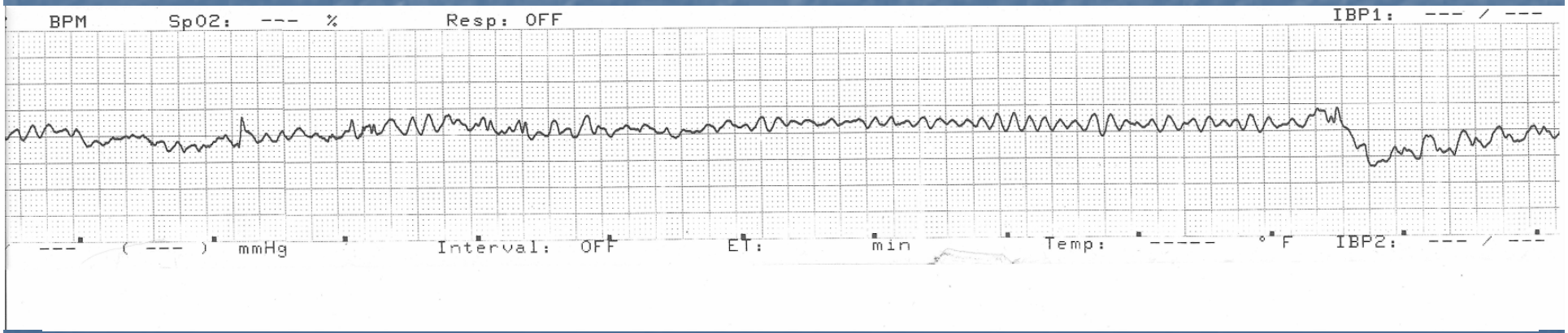


Case

- Presented 4 hours old
- 334 day gestation
- Problems:
  - IUGR – 77 lb
  - FIRS/placentitis
  - Neonatal Encephalopathy
  - Neonatal Nephropathy
  - Upper Airway Obstruction

Cardiopulmonary Arrest

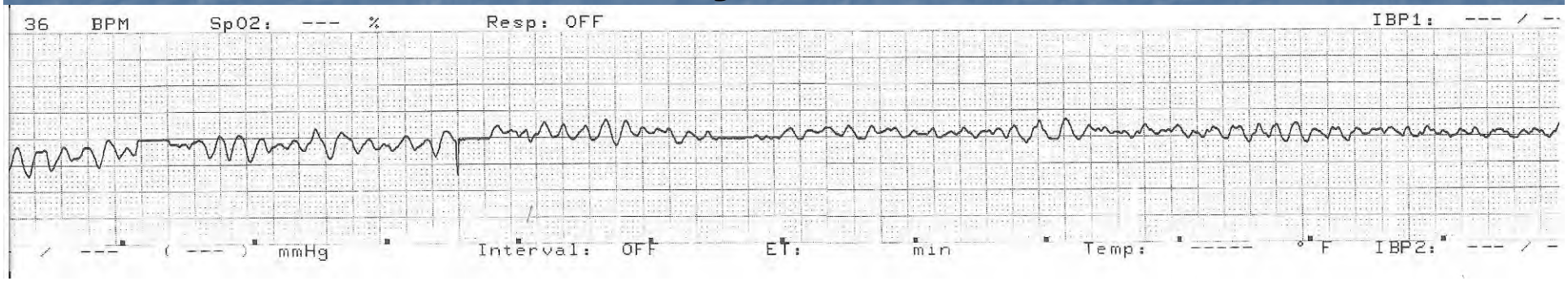
- During endotracheal tube change
  - Apneic/Bradycardia
- 9:20a Cardiopulmonary arrest recognized
  - Moved off mattress
  - Began cardiac compression
  - Intubated with some difficulty



Cardiopulmonary Arrest

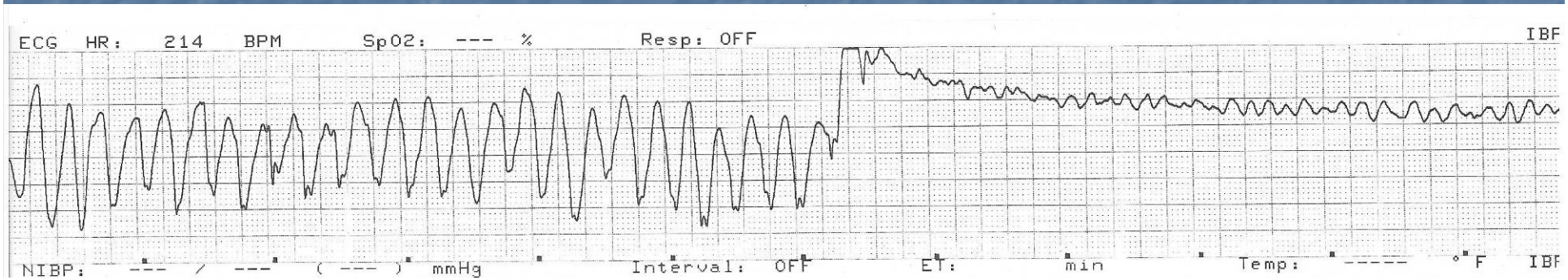
- Clipping chest for defibrillation
- 9:28a - $ETCO_2$ = 5
- 9:30a - auscult heart

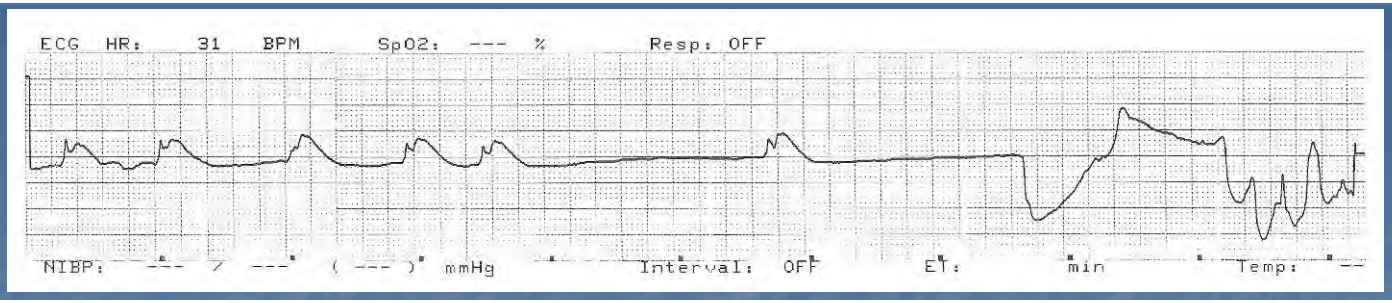
  No organized activity
- 9:31a - Epi
- 9:32a - Shock 100j

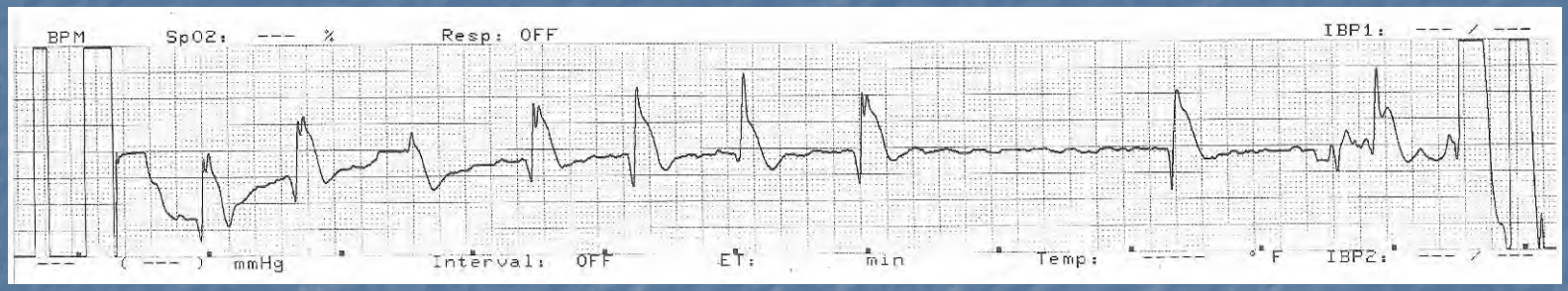


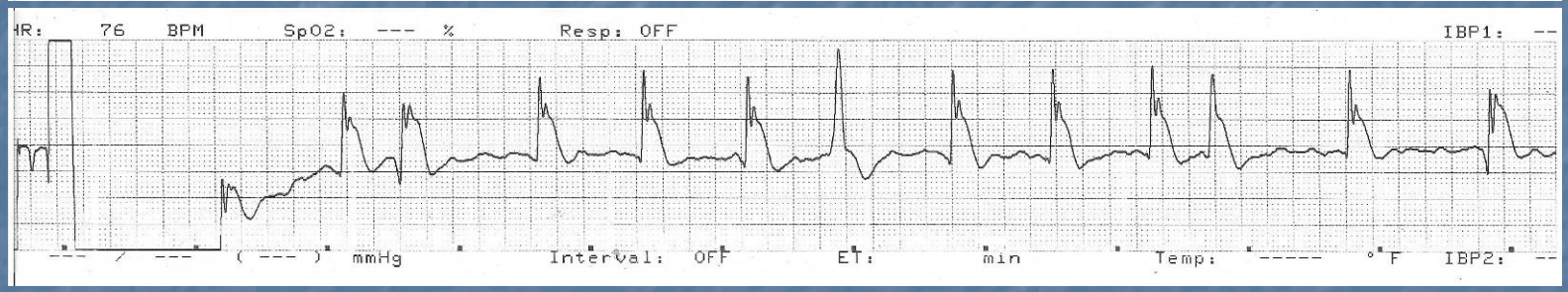
Cardiopulmonary Arrest

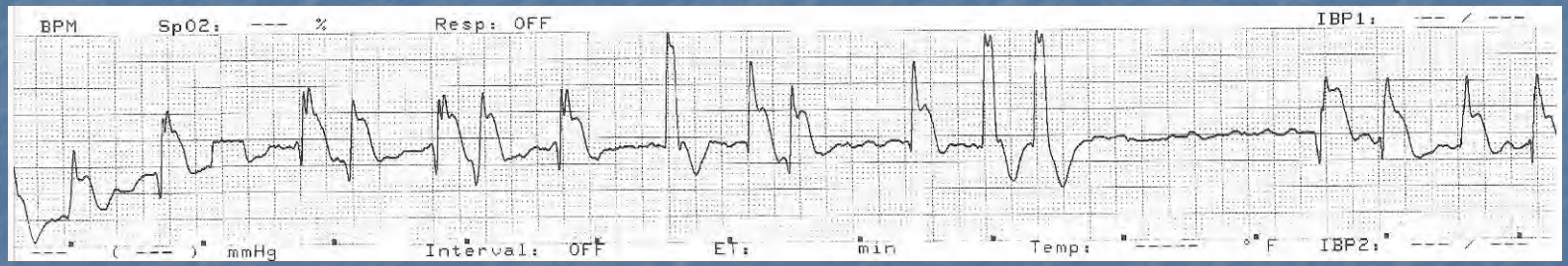
- 9:33a - shock 200j
- 9:34a - cont chest compressions;
  3 cc lidocaine iv
- 9:35a - shock 200j; chest compressions; nonperfusing bradycardia
- 9:37a - chest compressions
- 9:38a - 1 ml epi; back in v tach/v fib
- 9:40a - shock 200j - conversion

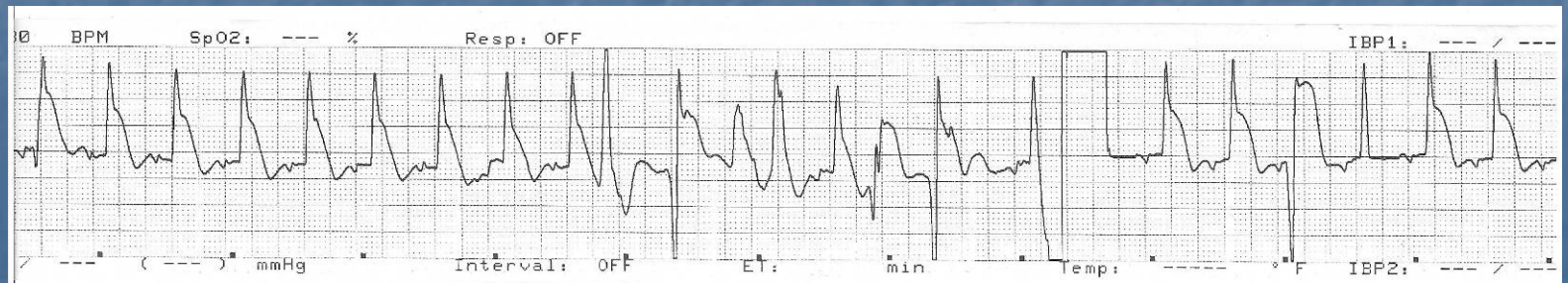



ECG HR: 31 BPM SpO2: --- % Resp: OFF
NIBP: --- / --- (---) mmHg Interval: OFF ET: min Temp: --


BPM SpO2: --- % Resp: OFF IBP1: --- / ---
--- (---) mmHg Interval: OFF ET: min Temp: ----- °F IBP2: --- / ---


HR: 76 BPM SpO2: --- % Resp: OFF IBP1: --
--- / --- (---) mmHg Interval: OFF ET: min Temp: ----- °F IBP2: --


BPM SpO2: --- % Resp: OFF IBP1: --- / ---
--- (---) mmHg Interval: OFF ET: min Temp: ----- °F IBP2: --- / ---


BPM SpO2: --- % Resp: OFF IBP1: --- / ---
--- (---) mmHg Interval: OFF ET: min Temp: ----- °F IBP2: --- / ---

Cardiopulmonary Arrest

- 9:42a - ROSC
- 9:44a - $ETCO_2$ 87; $ETCO_2$ 91; $ETCO_2$ 99
- 9:45a - $ETCO_2$ 66-68
- 9:46a - Temp 99.9
- 9:48a - Back on all fluids
- 9:49a - Ambu resp rate 30, HR 92, $ETCO_2$ 56

Cardiopulmonary Arrest

- 9:53a - spontaneous respiration - $ETCO_2$ 40
- 9:55a - assisted ventilation again
- 9:56a - assisted ventilation $ETCO_2$ 62
- 9:58a - Temp 99.7; $ETCO_2$ 52
- 10:02a - HR 92; RR 28
- 10:03a - spontaneous respiration trial
  $ETCO_2$ 62, HR 91, RR 24
- Failed spontaneous ventilation trial
- 10:35a – stable
  - Needs CV and respiratory support

Cardiopulmonary Resuscitation

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Take Home Message

All equine practitioners should be versed in cardiopulmonary resuscitation (CPR) of the foal. The challenge is not the physical skills but having a well thought out plan. The following is a brief description of the essentials of CPR which have been reviewed in greater detail elsewhere.[1]

Introduction

Cardiopulmonary arrest in foals is usually secondary to other serious conditions such as septic shock or respiratory failure. Often, the underlying condition is progressive eventually leading to respiratory arrest, then bradycardia which deteriorates to asystole. Early recognition and treatment of the predisposing condition will often prevent the arrest. When arrest does occur prompt intervention is vital.

The most important step in resuscitation is preparation. During the crisis there is no time to formulate a plan. Well thought out algorithms must be preplanned and ready to initiate once the nature of the crisis is recognized. Resuscitation equipment and drugs must be organized and easily transported stall side. Printed algorithms with drug doses in terms of amounts needed for the typical foal should be readily available. The steps necessary for successful CPR include establishing an airway, ventilation, chest compressions, administering drugs, determining the cardiac arrhythmia and treating accordingly.

Ventilation

When cardiopulmonary arrest is caused by primary cardiac failure, initiation of cardiac compressions is the priority. Ventilation is much less important. On the other hand, when cardiopulmonary arrest is caused by systemic disease initiating pulmonary arrest leading to cardiac failure ventilation is more important. If there is at least a minimally perfusing bradycardia when the crisis is recognized, ventilation should be established before any other action is taken. Delaying cardiac contractions in the first case or delaying ventilation in the second case will significantly decrease the chances of successful resuscitation.[2]

If an endotracheal tube is not immediately available to establish an airway mouth-to-nose ventilation can be very effective, facilitated by the fact that foals are obligate nasal breathers. But a 55 cm long cuffed endotracheal tube should be placed as soon as available. Nasotracheal intubation can be performed without assistance allowing others to begin cardiac compressions, establish intravenous access, prepare appropriate drug dosages or attach monitoring equipment.

Ventilation can be achieved with a self-inflating bag-valve device designed for human adult resuscitation. Ventilation can negatively affect outcome in CPR.[3] Increased thoracic pressure induced by positive pressure ventilation interferes significantly with cardiac return, decrease coronary and cerebral perfusion.[4] When primary cardiac arrest is present and respiratory function was normal until the arrest, negative aspects of ventilation outweigh the positive affects and ventilation should be pursued minimally. But when, as in most neonates, it is an asphyxial arrest (respiratory failure proceeds cardiac arrest), the positive aspects of ventilation become more important to survival. When giving breaths, the goal should be rapid infrequent breaths. Inspiration should be limited to no more than 1 second and rate no more than 8-10/min.[2] Careful observation of chest excursion is the best way to gauge tidal volume. The goal is not a deep breath.

Chest Compressions

Chest compressions should be initiated immediately, if a nonperfusing cardiac rhythm is present. Do not delay until cardiac contractions stop. The goal should be a rate 100 with complete chest recoil between compressions. It is important to minimize interruptions in compression with a maximum interruption of 10 sec.

Cardiac compression can be done by placing the foal on a firm surface with withers against a wall so that it does not move during forceful compressions. While kneeling between the foal's front and hind legs, place the palm of the hand with the fist closed over the heart. Place the other hand on top of the first to reinforce the compressing hand. The elbows should remain straight and the motion for compression should originate from the waist. Chest compression results in no more than 25-30% of normal cardiac output. If an airway is secured, coordination between ventilation and chest compression is not needed.

Monitoring pupil size is an indirect indication of adequate cerebral perfusion. When perfusion of the head becomes inadequate the pupils dilate widely. When chest compression results in adequate perfusion to the head, the pupils will assume a more neutral size. Chest compression technique should be adjusted as indicated by pupil size.

The most effective measurement of cardiac output during CPR is end-tidal CO_2 (P_{ETCO_2}). When there is no cardiac output to the lungs, the P_{ETCO_2} is 0. When chest compression results in effective cardiac output and lung perfusion occurs, the P_{ETCO_2} increases. As cardiac output increases with effective chest compressions, the P_{ETCO_2} will increase to a level of 12-18 torr. ABG and pulse oximetry are not useful monitors of effectiveness of CPR.[5]

Vascular Access

Establishing vascular access is essential if the neonate does not respond immediately to chest compressions and ventilation. The jugular vein should be catheterized using whatever available materials the resuscitator believes will result in the most rapid vascular access. Drugs can be given by the intratracheal route, but the absorption is very poor so this route should only be utilized if there are no other possibilities. A more useful circulatory access route when vascular

access is not immediately available is the intraosseous route. Unless the foal is very premature with incomplete ossification, a special intraosseous device is needed with the most successful access on the medial tibial plateau. Drugs which can be administered by the intraosseous route include fluids, glucose, Na bicarbonate, $CaCl_2$, blood, plasma, epinephrine, lidocaine, atropine, dopamine, dobutamine, vasopressin, antimicrobials, phenobarbital, diazepam, butorphanol, insulin and virtually any drug which can be administered intravenously.[6-9]

Drug Therapy

The most helpful drug in CPR is epinephrine. Epinephrine can improve coronary perfusion pressure during cardiac arrest. Both beneficial and toxic effects of epinephrine administration during CPR have been shown.[6,7] Low dose (0.01- 0.02 mg/kg) and high dose (0.1 mg/kg) regimens have been proposed. Anecdotal experience in foals suggests that myocardial necrosis is more extensive with high dose therapy. Although there may be some cases which could benefit from the high dose regimen, it should not be used routinely. Epinephrine therapy is indicated for cardiac arrest regardless of the underlying cause or rhythm. It is appropriate to administer 0.01-0.02 mg/kg dose every 3 minutes.

Vasopressin is a nonadrenergic endogenous stress hormone which is a very potent peripheral vasoconstrictor which is marked in cardiac arrest.[5] Vasopressin is as effective as epinephrine, no matter what the presenting nonperfusing cardiac rhythms, in aiding return to spontaneous circulation. When the nonperfusing rhythm is asystole, the use of vasopressin or vasopressin in combination with epinephrine may be more effective than epinephrine along in returning spontaneous circulation.[10,11] The dose of vasopressin for pulseless cardiac arrest is at total of 0.6 U/kg given either as a single dose or divided. Repeat doses are unnecessary.

In pulseless cardiac arrest associated with PEA (pulseless electrical activity) or asystole which does not respond to ventilation, cardiac compression and epinephrine/vasopressin, treatment with atropine is appropriate. The atropine dose should be 0.02 mg/kg repeated once in 5 minutes if necessary. Potential complications include tachycardia leading to exacerbation of hypoxic insult by increasing the oxygen demand of cardiac muscle. Paradoxically atropine at low doses can exacerbate bradycardia.[12,13]

Large volumes of fluids are important in treating septic shock which frequently leads to cardiac arrest. But during cardiac arrest, fluid administration is contraindicated. A nonperfusing rhythm produces a situation resembling congestive heart failure, with ineffective cardiac output. With effective chest compressions, the cardiac output is only 20 - 25% of normal. If fluids are given rapidly the venous pressure rises, impeding coronary perfusion and return of a normal cardiac rhythm, despite effective chest compressions and doses of epinephrine. If volume replacement is indicated because of severe dehydration, bolus administration is preferred rather than a continuous high flow rate. Once a perfusing rhythm returns, increased fluid rates may be needed to help maintain cardiac output. Severe hypoglycemia can lead to cardiac arrest; however, glucose containing fluids should be avoided during resuscitation unless a patient-side glucose determination indicates severe hypoglycemia. Hyperglycemia and hyperosmolality, secondary to rapid glucose infusion, during resuscitation is associated with poor neurologic outcome.[14-17]

The use of sodium bicarbonate during cardiac arrest remains controversial. There is little data supporting its use and many contraindications. Indications for bicarbonate administration include hyperkalemia (as secondary to ruptured bladder), preexisting metabolic acidosis leading to arrest, or phenobarbital overdose. More controversial indications include prolonged, nonresponsive cardiac arrest and after return of spontaneous circulation. Hypoxic lactic acidosis is a clear contraindication.

Summary

When a pulseless cardiac rhythm is identified in a foal, the first priority should be to begin cardiac compressions. That should be followed by establishing an airway and beginning ventilation. Next, vascular access should be established. Drug therapy with epinephrine/vasopressin should follow. Then monitoring equipment (ECG, capnography) should be attached. The cardiac rhythm should be established as shockable (VF/pulseless tachycardia) or non-shockable (PEA or asystole). At this point the scheme should branch with either defibrillation/epinephrine/lidocaine or continued CPR/epinephrine/possible atropine paths. The cardiac rhythm should be periodically monitored as the scheme followed may change from one branch to the other at any time.

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