CPR:
A Focus on Prevention and Post Arrest Care

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Leading Specialty Healthcare for Pets

CPR

- Going to focus on anesthetic arrest
- Prevention
  - Anesthetic protocol
  - Monitoring
  - Preparedness
- CPCR (cardiopulmonary cerebral resuscitation)
- Post-arrest care

Prognosis

- Less than 10% survival in dogs/cats in hospital cardiopulmonary arrest (CPA)
- 20% survival in humans
- Veterinary literature
  - Anesthetic arrest improves prognosis (up to 50%)
  - Having IV catheter in place improves prognosis

Pre-Operative Planning

- Physical exam, weight, TPR
  - Cardiovascular system
    - Heart murmur
    - Lung
    - Stertor/stridor
    - Clear lungs
    - Arrhythmias
    - Good pulses
  - **Anesthesia personnel should perform also!**
- Chemistry/urinalysis – Liver and kidney function, albumin
- CBC – PCV/Hct > 25% recommended

Pre-Existing Medications

- NSAIDs
- Anticonvulsants
- Cardiac medications (ACEi, inotropes, diuretics)
- May predispose to GI bleeding, renal impairment, electrolyte abnormalities, hypotension, added depressant effects

Pain Control

- Administered before pain develops
  - Less drug therapy needed to control pain

Multi-Modal Anesthesia

- Lowers amount of each medication/inhalants needed

Opioids

- Act centrally to limit input of nociceptive information to the central nervous system (CNS)
- Pros
  - Rapid onset of action
  - Safe
  - Reversible
  - Potent analgesics
- Cons
  - Respiratory depressant
  - Bradycardia
  - Gastroparesis/ileus

Opioids

- Butorphanol – mixed mu agonist-kappa antagonist
  - Ceiling affect, good sedation, some pain control
- Buprenorphine – partial Mu
  - Ceiling affect, moderate pain control
- Methadone - pure Mu agonist, NMDA antagonist
  - Good pain control, duration 3-4 hours
- Fentanyl – pure Mu
  - Good pain control, short duration (<30 minutes)

Opioid Complications

- Bradycardia – anticholinergic
- Hypoventilation – controlled ventilation
- Reversal
  - **Naloxone – 0.004mg/kg IV**
    - Competitively displaces agonist
    - May only last 30 minutes, risk for re-narcotization
  - Butorphanol
    - Some reversal effects and still kappa effects
  - Buprenorphine – harder to reverse

Benzodiazepines

- Minor tranquilizer, no analgesic effects
  - May help prevent dysphoria with other agents
- Anti-anxiety, euphoria, anti-convulsant, muscle relaxation
- Decrease amount of additional medication
- Mild respiratory depression
- Reversible:
  - **Flumazenil – 0.01 mg/kg IV**

Alpha-2 Adrenergic Agonists

- Dexmedetomidine
- Sedation, analgesia, muscle relaxation
- Vasoconstriction, bradycardia, respiratory depression
- Use with anticholinergic?
  - Not routinely recommended
  - If hypotensive and bradycardic than recommend to maintain adequate cardiac output
- Reversible – atipamezole - IM

NMDA Receptor Antagonist

- Ketamine
- Analgesia (prevention of wind-up), amnestic
- Minimal cardiovascular depression and ventilatory depression
  - Can increase heart rate, blood pressure, CO
- Tremors, increased intracranial pressure
- Not reversible

Question 1

Which sedative cannot be reversed?

- 1. Fentanyl
- 2. Hydromorphine
- 3. Acepromazine
- 4. Midazolam

Induction Medications

- Propofol
  - Short acting
  - Cardiopulmonary depression
    - Hypotension, apnea
- Alfaxalone
  - Short acting
  - Cardiopulmonary depression similar to propofol (maybe slightly less)
- Etomidate – minimal cardiovascular effects
  - Adrenal dysfunction at >1-2mg/kg IV

Protocols

- Tailored to patient with multi-modal goal
- Pre-medication
  - Opioid and benzodiazepam
- Induction
- Maintenance
  - Inhalants
  - CRI (Opioids , Ketamine, Lidocaine, Dexdomitor, Propofol/alfaxalone)

Example Protocol for High-Risk Patient

- Midazolam and Fentanyl +/- Etomidate
- CRI of Fentanyl, ketamine +/- inhaled anesthetic

Acepromazine

- Sedative/tranquilizer
- Hypotension

Brachycephalics

- Gastroprotectants – ideally started couple days prior to surgery
- Prokinetics
- Minimize respiratory depressants
- Brief airway exam
- Pre and post-oxygenation
- Insure adequate ventilation during recovery

Anxiety

- Trazodone
- May help reduce anxiety associated with recovery and being in hospital
- May increase sedation
- Serotonin syndrome, reacts with other medications

Minimize Anesthesia Time

- Protocols to minimize amount of time under anesthesia
  - Shave patient
  - Have monitoring and other equipment ready

Pre and Post Oxygenation

- Important to decrease amount of atelectasis
  - Absorption atelectasis, trapped gas
- Helps treat hypoxemia secondary to hypoventilation

Preparedness

- Emergency drug sheet for patients prior to any sedation/anesthetics
- Monitoring either on patient at induction or readily available
- Crash cart easily accessible

Crash Cart

- pre-stocked arrest stations and cognitive aids improves compliance with CPR protocols
- Include commonly used arrest medications:
  - Reversal
  - Epinephrine
  - Atropine
  - Dextrose/Bicarb/Calcium/Vasopressin

Question 2

What is your favorite Monitoring device?

- 1. ECG
- 2. Blood pressure
- 3. SpO2
- 4. Capnograph
- 5. Physical Exam

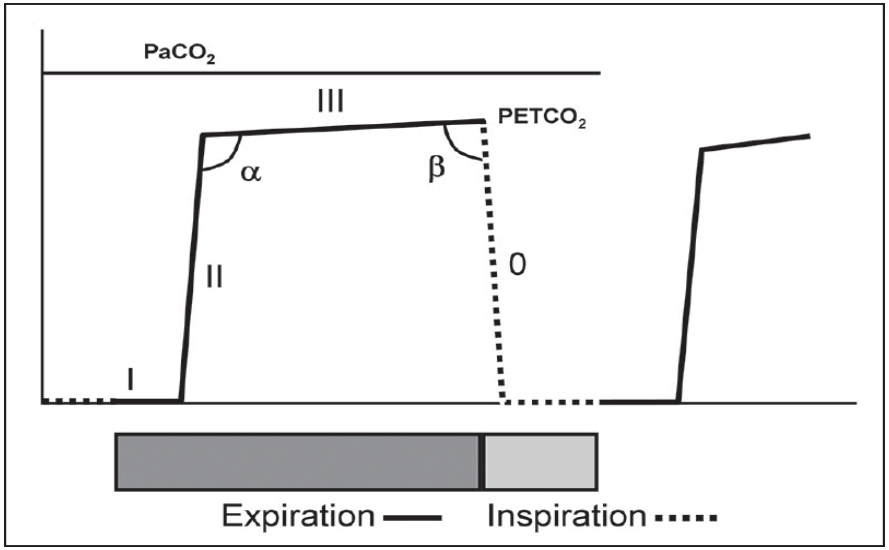
Monitoring

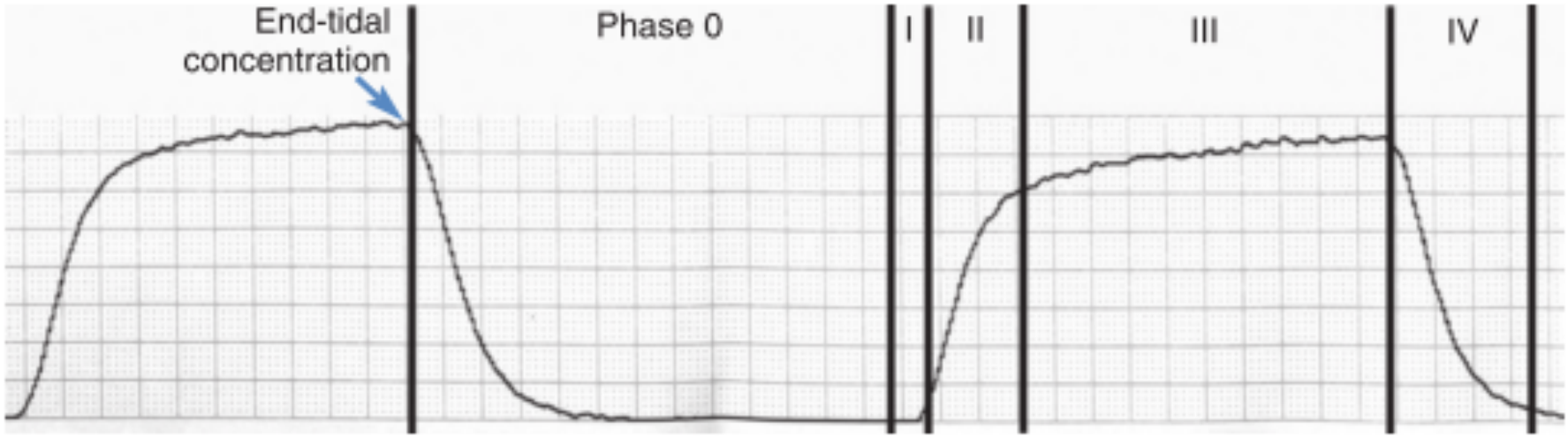
- ECG
- Blood pressure
- Capnography
- SpO2
- Temperature
- Physical exam

Capnography

- Assessment of carbon dioxide concentration
  - Considered $PACO_2$, surrogate for $PaCO_2$ which is typically about < 5 mm Hg more
- Best assessment of ventilation
- Can alert to abnormalities related to patient or the equipment
- Can be used during anesthesia, CPR, and recovery

Capnogram





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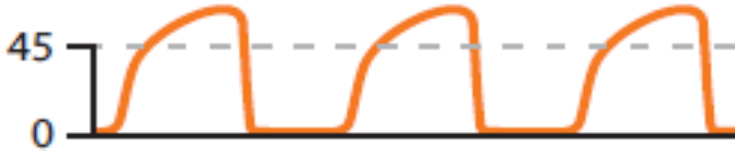
Interpretation

Bronchospasm (shark-fin appearance)

Asthma, COPD



Hypoventilation

Hyperventilation

Decreased $EtCO_2$ — Apnea, Sedation

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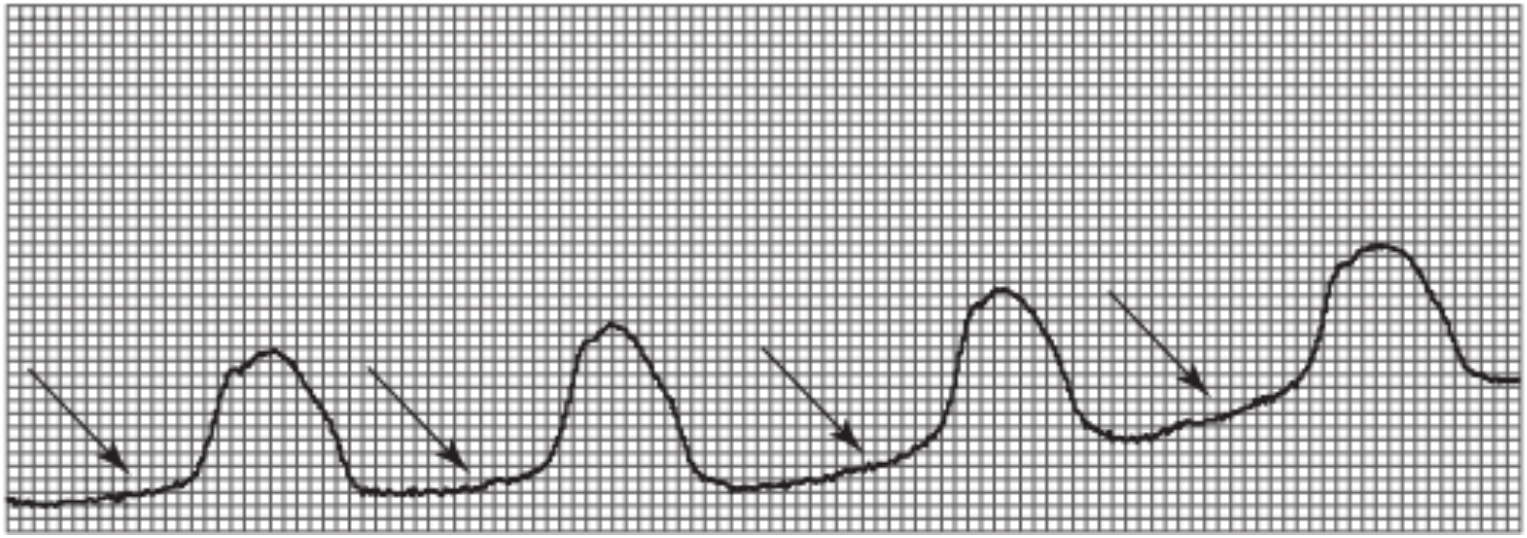
Question 3

What is your diagnosis?

- 1. Bronchoconstriction
- 2. Re-breathing
- 3. Hypoventilation
- 4. Hypotension



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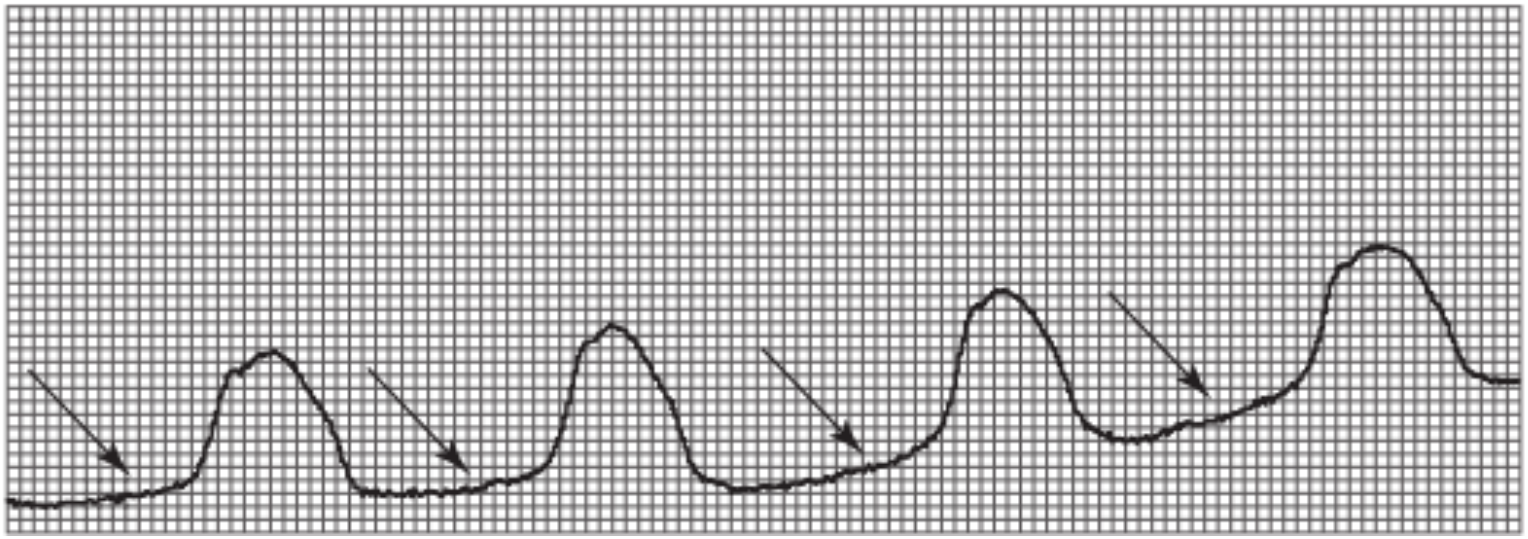
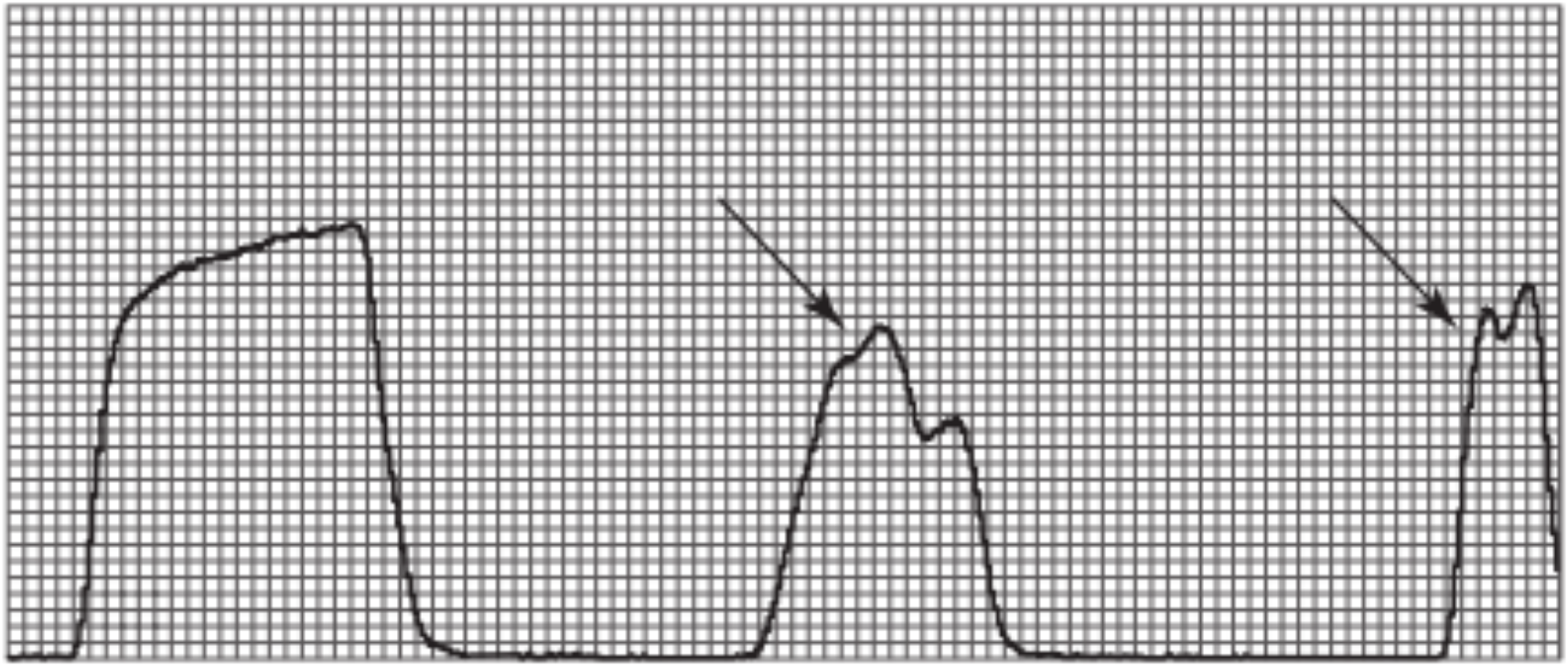
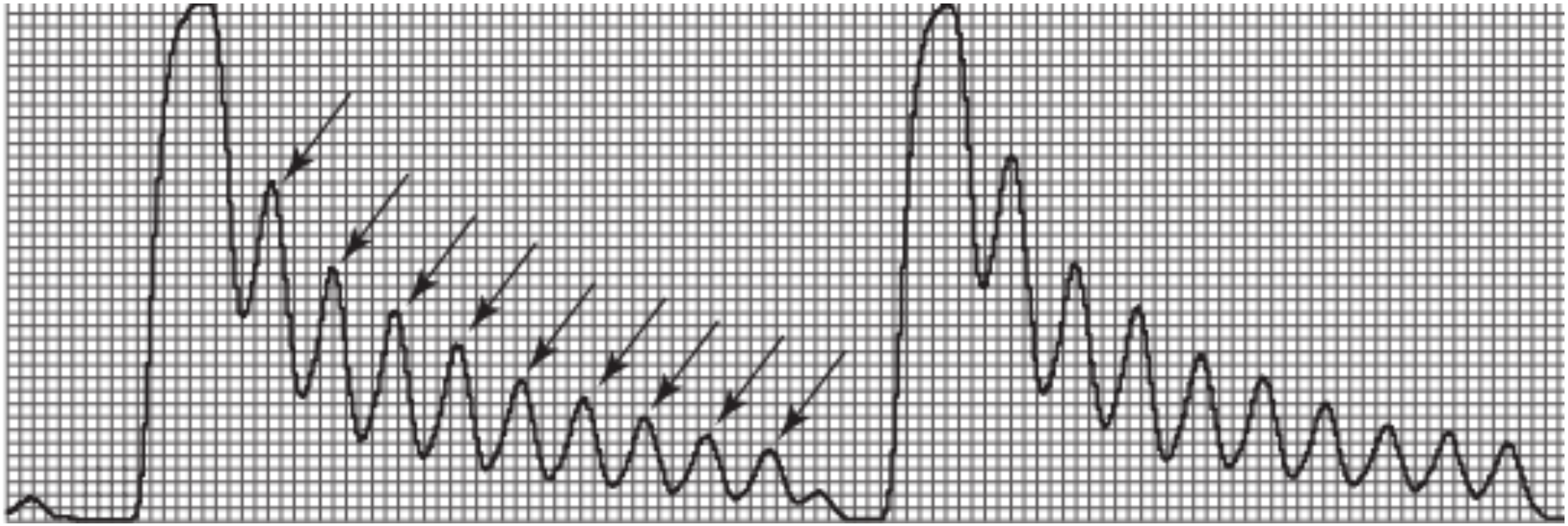
Question 4

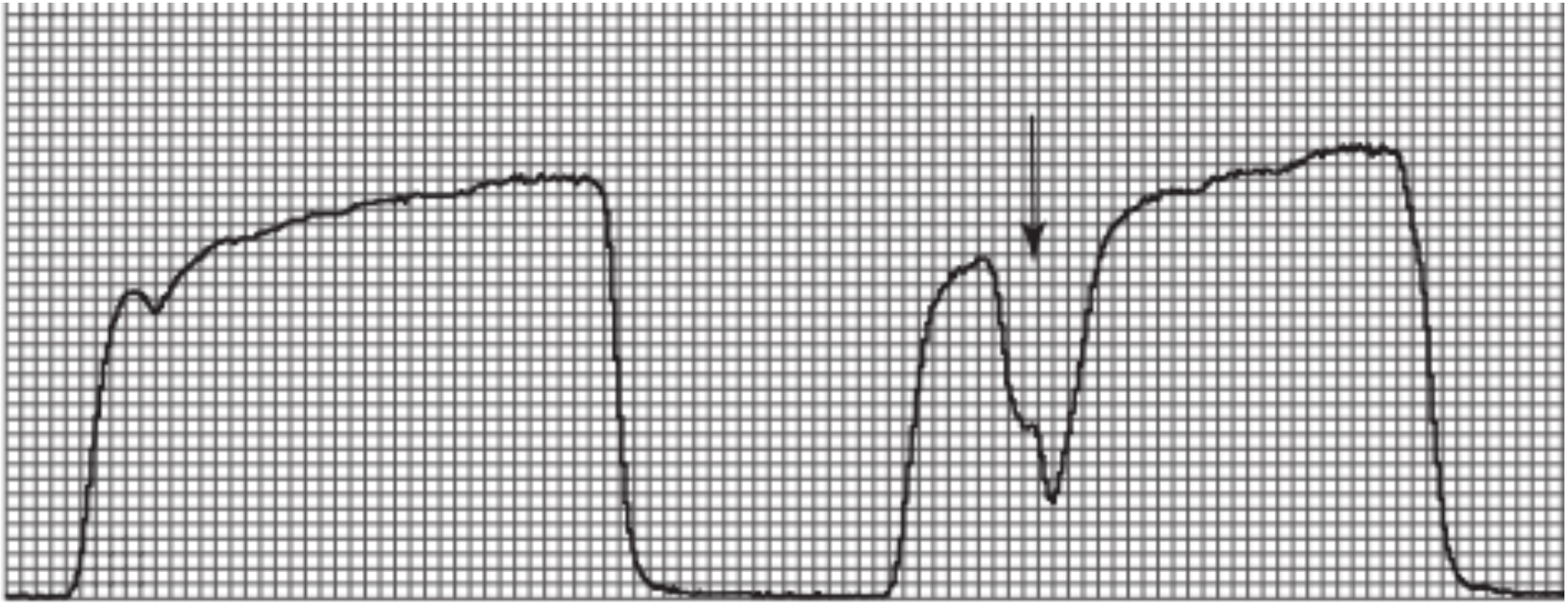
What causes this capnograph?

- 1. Increased dead space
- 2. exhausted CO2 absorbent
- 3. Inadequate fresh gas flow
- 4. all of the above









Recovery

- Continue monitoring until patient awake and alert
- Avoid quick stimulation and extubation

CPR

- Identification of CPA
- Start CPR immediately if CPA can not be ruled out
- Designate leader for CPR

Basic Life Support

- Immediate initiation of chest compressions with intubation and ventilation being performed simultaneously
- Ventilation rate – 10 breaths/min without interruption to chest compressions
- Chest compressions – compress 1/3 to ½ width in lateral recumbency, rate of at least 100 compressions/minute, allowing **full recoil**
- 2-minute cycles of uninterrupted compressions with alternation of compressors between cycles. Keep interruptions in compressions to minimum

Advanced Life Support (ALS)

- Epinephrine 0.01mg/kg IV every 3-5 minutes
- Rapid defibrillation in animals with pulselss VT or VF with biphasic defibrillator
  - Defibrillation should follow a cycle of CPR in >4 minutes CPA
- Open chest CPR considered in select cases when appropriate care available
- Reversal of anesthetic agents and correction of major acid-base and electrolyte disturbances

Vasopressors and Vagolytic Therapy

- Goal of improving coronary perfusion pressure (CPP)
  - Defined as the difference between diastolic aortic and right atrial pressure
  - Vasopressors – increase aortic pressure by increasing peripheral vascular resistance and directing more of the intravascular volume to the central circulation
    - Inotropic and chronotropic effects likely less crucial and may be harmful (increase myocardial oxygen demand, exacerbating myocardial ischemia, and predisposing to arrhythmias)

Vasopressin Versus Epinephrine

- Vasopressin 0.8U/kg IV, with or without epinpehrine, is a reasonable intervention during CPR
- Several studies show no harm with vasopressin and some evidence suggest benefit
- Possible advantage of certain subgroups
  - Asystole, prolonged CPA, hypovolemia

Atropine

- Little evidence that use is harmful (at dose of 0.04mg/kg)
- Animals with high vagal tone, atropine use is reasonable
  - Gastrointestinal disease, brachycephalic patients, respiratory distress
- No real evidence for use without high vagal tone

Antiarrhythmic drug therapy

- No compelling evidence that supports the routine use of antiarrhythmic drugs
- Dogs with shock-resistant pulseless VT or VF, amiodarone may be best choice
- Limited, but conflicting evidence that lidocaine may also be a useful adjunctive with shock-resistant VF, especially with biphasic defibrillators

Steroids

- No clear evidence of benefit or harm
  - Studies in other disease suggest harmful effects, therefore routine use not recommended
- May be beneficial for specific patients with suspected insufficiency

Buffer Therapy

- Against the routine administration of sodium bicarbonate
  - However, dog studies show less appreciation of a detrimental effect
- May be considered in prolonged arrest, or pH based

Electrolyte Correction

- No evidence to suggest treatment of mild electrolyte disturbances
- Moderate-to-severe hyperkalemia influences myocardial function and should be treated
- Severe ionized hypocalcaemia may be treated and calcium therapy warranted in calcium channel blocker overdose
  - Hypocalcaemia progresses with CPA however benefit of treating has not been shown

Three-Phase Model of CPA

- 1st phase, electrical phase - Last about 4 minutes
  - minimal ischemia
- 2nd phase, circulatory phase – between 4-10 minutes
  - Energy depletion and potentially reversible cellular damage
- 3rd phase, metabolic phase – after 10 minutes
  - Ischemic injury

Open Chest CPR

- More effective in restoring ROSC and promoting good outcome in canine models of VF
- In cases of significant intrathoracic disease may be advisable
- May cause increased regurgitant fraction in dogs with mitral regurgitation

Intravenous Versus Intratracheal Drugs

- Should give venous
- Lack of venous or IO than can give IT (epinephrine, atropine, vasopressin)
  - 10-fold increase in dosage of epinephrine
  - Delivered via a catheter to level of carina or farther
  - Diluted in sterile water, saline if water not available

Question 5

What is the most important part of CPR?

- 1. Recognition of arrest
- 2. Adequate chest compressions
- 3. Intravenous drugs
- 4. Adequate breathing

Anesthetic-Related Arrests

- Consider lipid rescue when due to local anesthetic drugs or other lipophilic drugs

Monitoring

- Chest compressions initiated immediately for apneic, unresponsive patients
- ECG analysis may help rule out CPA, and used to evaluate for rhythms requiring specific therapy
- End-tidal CO2 should not be used as sole confirmation of endotracheal intubation
- Minimize pauses in compression to evaluate ECG
- EtCO2 monitoring useful to identify ROSC
- Monitor patient following ROSC

Major Points

- Don’t rely on pulses or Doppler
- Use laryngoscope along with visualization of chest wall movement for confirmation of endotracheal tube
- EtCO2 may help evaluate effectiveness of compressions and ROSC
  - Therefore decreasing time stopped evaluating rhythm

Post-Cardiac Arrest (PCA) Care

- Overview of general Post-Cardiac Arrest physiology and care

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Paradigms of Care During PCA

- Initial paradigm aims at pathophysiologic process that occur in the postresuscitaiton phase
- As ROSC progresses shift emphasis of treatment to underlying disease process, prognostication, and rehabilitation
- **Immediately after ROSC the focus is on prevention of recurrence of cardiac arrest and limitation of organ injury**

Silverstein, D., Small Animal Critical Care Medicine, 2e; 2014; p17-25

Postresuscitaiton Pathophysiology

- Ischemia and reperfusion (IR) injury
- PCA brain injury
- PCA myocardial dysfunction
- Persistent precipitating pathologic condition

Ischemia and Reperfusion (IR) Injury "Sepsis-Like" Syndrome

- Many characteristics similar to sepsis regarding inflammation, coagulation, and endothelium
- Systemic inflammatory response syndrome (SIRS)
- Impaired vasoregulation
- Coagulation abnormalities
- Adrenal suppression
- Impaired tissue oxygen delivery and utilization
- Impaired resistance to infection
- Intravascular volume depletion

Ischemia and Reperfusion (IR) Injury "Sepsis-Like" Syndrome

- Similar treatment goals
  - Early hemodynamic optimization
  - Glycemic Control
  - Critical illness-related corticosteroid insufficiency (CIRCI)

Hemodynamic Optimization

- Initial efforts to prevent rearrest by ensuring optimal ventilation, oxygenation, and tissue perfusion
- Goal-directed therapy:
  - Adequate oxygenation – SpO2 94-98% (PaO2 80-100 mmHg)
  - Blood pressure – Mean arterial blood pressure 80 mmHg or higher
  - Lactate clearance - goal lactate <2.5
  - Adequate ventilation – EtCO2 = Dog 32-42; Cat 26-36

Glycemic Control

- Hyperglycemia common in humans and associated with worse outcome; Hypoglycemia also associated with worse outcome
- Hyperglycemia suspect secondary to catecholamines
- Recommend moderate glycemic control
  - Avoid iatrogenic hypoglycemia

Hopper, K; Brochers, A; Epstein, S; Acid base, electrolyte, glucose, and lactate values during cardiopulmonary resuscitation in dogs and cats. JVECC 24(2) 2014

Adrenal Dysfunction

- Steroids important for regulation of vascular tone and endothelial permeability
- Routine administration not recommended
- Should be considered when vasopressor-dependent
  - Low-dose hydrocortisone – 1mg/kg IV q6
  - No studies to show benefit in PCA phase

PCA Brain Injury

- Considered most common cause of death in humans
- Pathophysiology related to cerebral Ischemia/reperfusion
  - Most of the injury sustained during reperfusion, no during ischemia
  - Burst of reactive oxygen species (ROS) during reperfusion → propagates injury to neural cells
- Little evidence that brain edema or elevated ICP directly exacerbates PCA brain injury

Treatment Goals for Brain Injury

- Controlled re-oxygenation
- Therapeutic hypothermia
- Seizure control
- Mechanical Ventilation
- Airway protection

Controlled Reoxygenation

- Hyperoxemia soon after ROSC:
  - Increases oxidative brain injury
  - Increases neurodegeneration
  - Worsens functional neurologic outcome
  - Negatively affects overall survival
- Titrate to normoxemia
  - SpO2 = 94%-98%
  - PaO2 = 80-100 mm Hg

Therapeutic Hypothermia

- Protective effects
  - Reduction of mitochondiral injury and dysfunction
  - Decrease in cerebral metabolism
  - Reduction of Ca inflow into cells and neuronal excitotoxicity
  - Reduced production of ROS
  - Reduced apoptosis
  - Suppression of seizure activity

Therapeutic Hypothermia

- Reduction of core body temperature to 90-93˚F
- Methods for cooling
  - Cooling blankets, ice packs, ice cold saline infusion, endovascular cooling devices
- Should be instituted immediately
- Recommended period for hypothermia unknown
  - Recommend 24-48hours
- Slow rewarming (0.5-1 ˚F) per hour
- Likely requires sedation
  - Increased muscle tone/shivering detrimental

Seizure Control

- Seizure incidence in veterinary patients unknown
- Should be monitored for seizure activity
- Prophylactic anticonvulsants not recommended at this time

Mechanical Ventilation

- Maintain normal ventilation
- Target of:
  - Dog – $PaCO_2$ 32-42
  - Cat $PaCO_2$ – 26-36

Neurologic Exam and Prognosis

- Not evaluated in veterinary patients
- In humans:
  - Not reliable during first 24 hours
  - 24-72 hours
    - Presence of coma and absent PLRs significantly increases likelihood of poor outcome
  - >72 hours
    - Presence of coma and absent PLRs becomes reliable indicator that will fail to regain consciousness

PCA Myocardial Dysfunction

- Decreased cardiac output
  - Reduced systolic and diastolic ventricular function
    - Increased end-diastolic and systolic volume
    - Reduced left ventricular ejection fraction
- Unknown pathophysiology
  - Likely multifactorial and similar to IR injury
- Reversible and typically resolve within 48hours
- Therapeutic hypothermia showed to attenuate
- Dobutamine showed to improve function and cardiac output

Persistent Precipitating Pathologic Condition

- Specific to patient

Cardiovascular Support

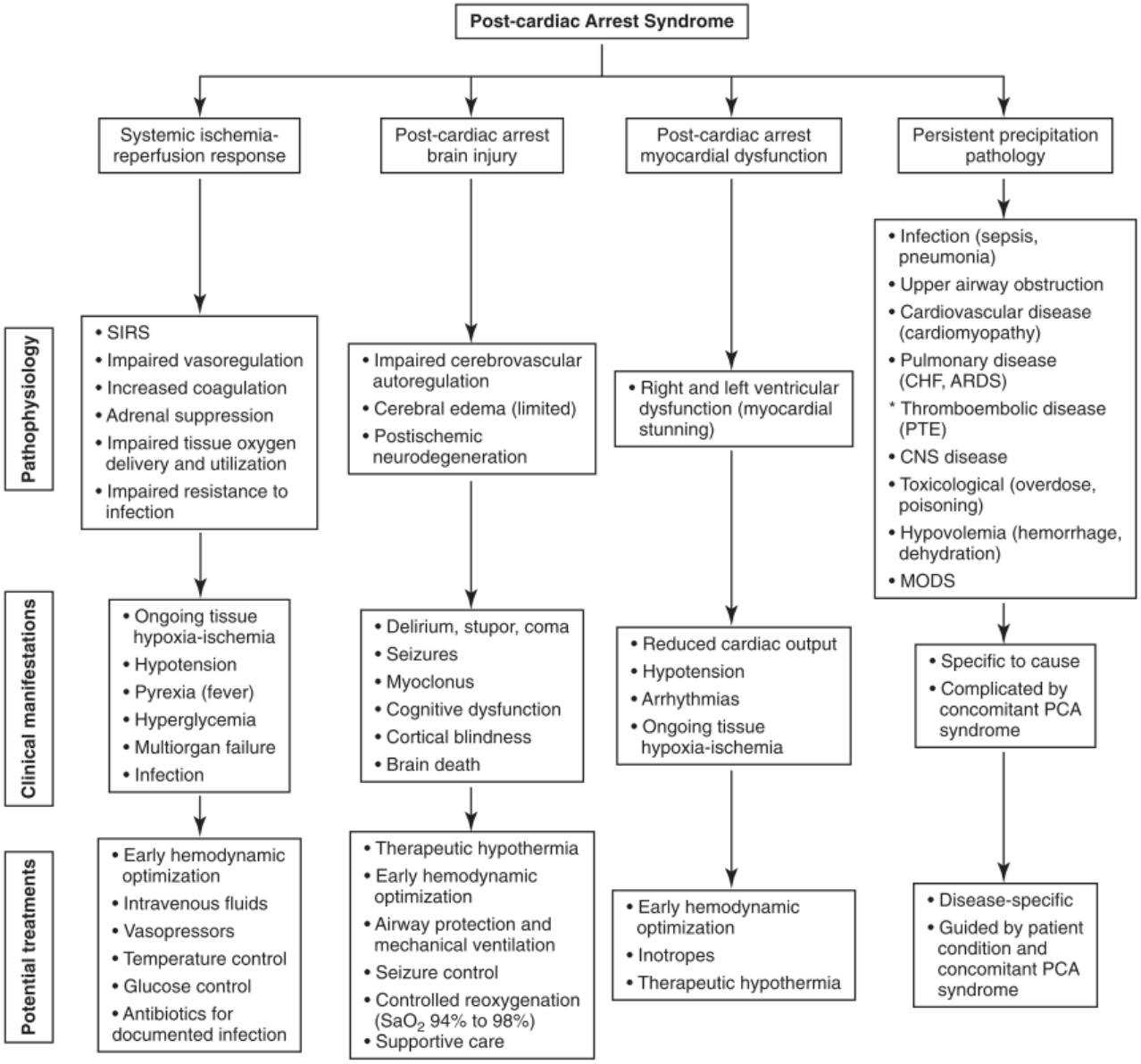
- Treatment with fluid, vasopressors, inotropes administration
- Goal Directed Therapy
  - Blood pressure, lactate
- Treatment tailored to patient:
  - Hypovolemia – Fluids
  - Vasodilation – Vasopressor (Norepinephrine, vasopressin)
  - Decreased contractility – Inotropes (Dobutamine, Dopamine)

Neuroprotective Stratagies

- Cerebral edema common but increased ICP rare in humans
- Not good evidence to support the use of routine osmotic agents in post-arrest patients
- In patients with signs consistent with cerebral edema mannitol or hypertonic saline may be considered

Ventilation

- Routine mechanical ventilation not recommend in current RECOVER guidelines post-arrest
- Benefits of mechanical ventilation
  - Airway protection
  - Decreased work/oxygen demand of patient
  - Controlled reoxygenation
  - Can better insure adequate ventilation
    - Maintain normoxemia and normocarbia
    - Helps avoid hypoventilation or hyperventilation



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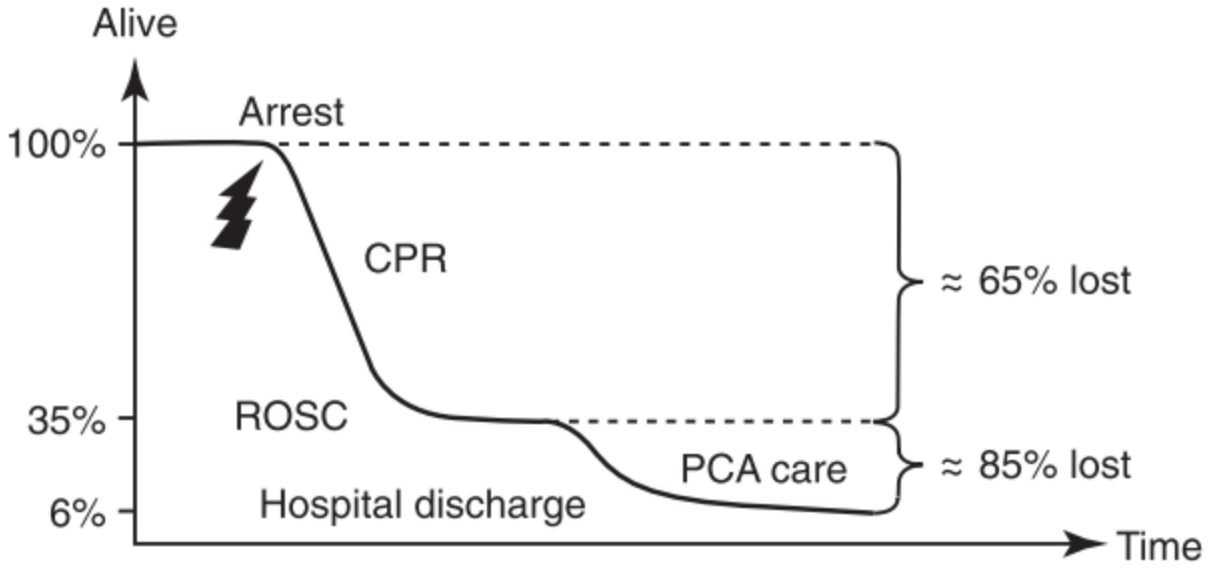
Prognosis

- Veterinary Medicine Arrest Patients
  - 58-65% mortality
  - 5-6% survival to discharge



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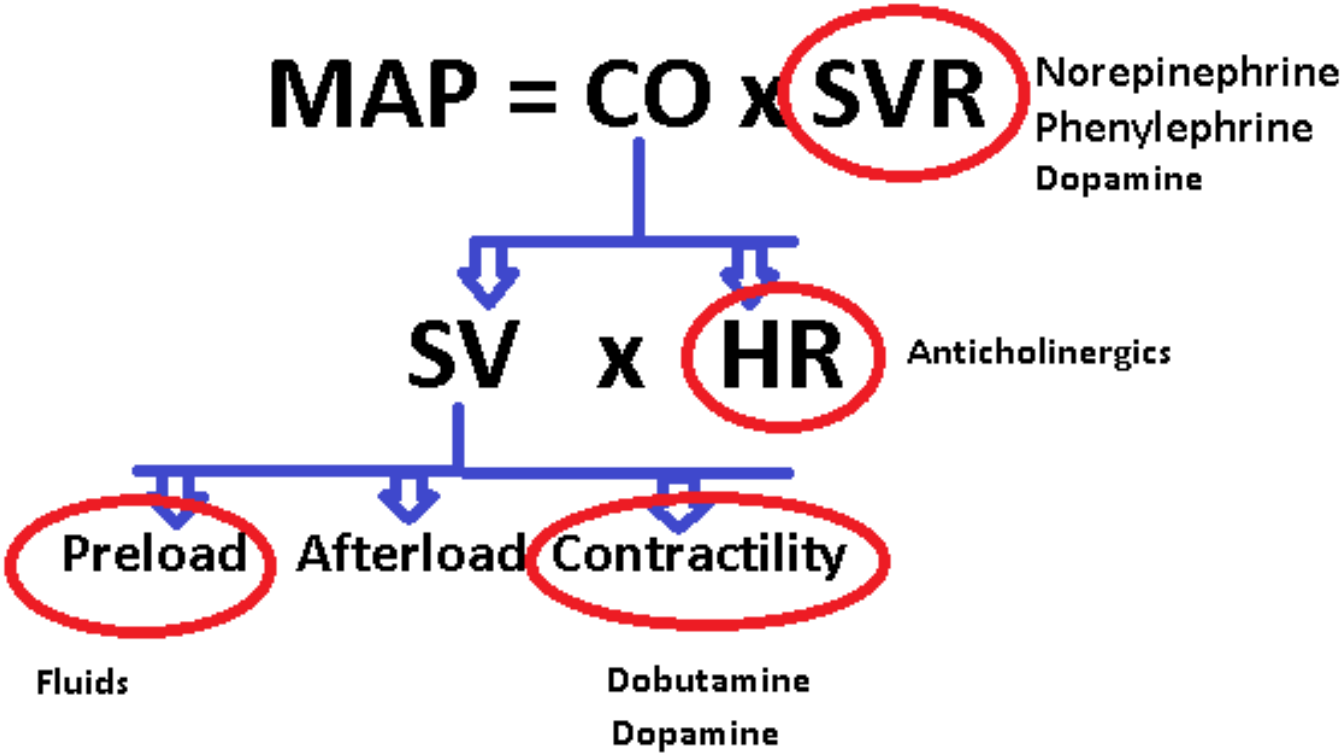
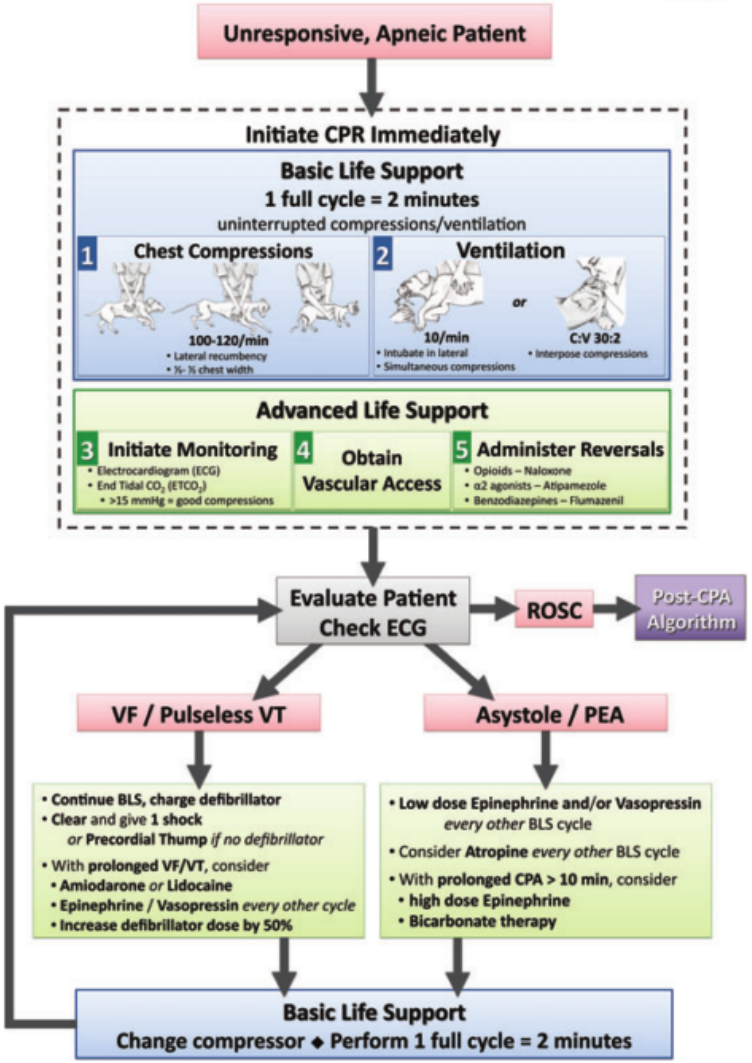
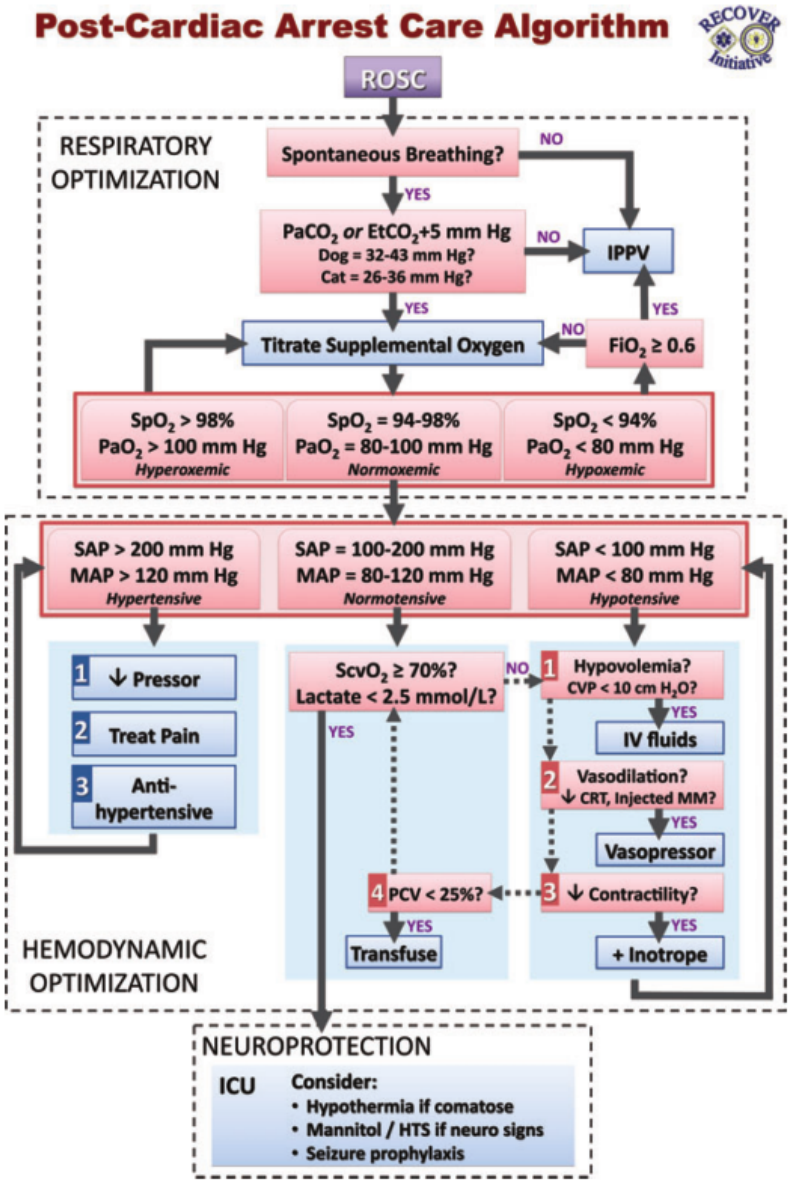
Approach to Hypotension



- Reassess after treatment for response
  - Adjust as needed
  - Expect response within 15 minutes

CPR Algorithm




Post-Cardiac Arrest Care Algorithm
RECOVER Initiative
ROSC
RESPIRATORY OPTIMIZATION
Spontaneous Breathing?
NO
YES
PaCO2 or EtCO2+5 mm Hg
Dog = 32-43 mm Hg?
Cat = 26-36 mm Hg?
NO
IPPV
YES
YES
Titrate Supplemental Oxygen
NO
FiO2 ≥ 0.6
SpO2 > 98%
PaO2 > 100 mm Hg
Hyperoxemic
SpO2 = 94-98%
PaO2 = 80-100 mm Hg
Normoxemic
SpO2 < 94%
PaO2 < 80 mm Hg
Hypoxemic
SAP > 200 mm Hg
MAP > 120 mm Hg
Hypertensive
SAP = 100-200 mm Hg
MAP = 80-120 mm Hg
Normotensive
SAP < 100 mm Hg
MAP < 80 mm Hg
Hypotensive
1 ↓ Pressor
2 Treat Pain
3 Anti-hypertensive
ScvO2 ≥ 70%?
Lactate < 2.5 mmol/L?
NO
YES
1 Hypovolemia?
CVP < 10 cm H2O?
YES
IV fluids
2 Vasodilation?
↓ CRT, Injected MM?
YES
Vasopressor
4 PCV < 25%?
3 ↓ Contractility?
YES
YES
Transfuse
+ Inotrope
HEMODYNAMIC OPTIMIZATION
NEUROPROTECTION
ICU
Consider:
• Hypothermia if comatose
• Mannitol / HTS if neuro signs
• Seizure prophylaxis

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CPR Emergency Drugs and Doses

	DRUG	DOSE	Weight (kg)	2.5	5	10	15	20	25	30	35	40	45	50
			Weight (lb)	5	10	20	30	40	50	60	70	80	90	100
				ml	ml	ml	ml	ml	ml	ml	ml	ml	ml	ml
Arrest	Epi Low (1:1000; 1mg/ml) every other BLS cycle x3	0.01 mg/kg		0.03	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
	Epi High (1:1000; 1 mg/ml) for prolonged CPR	0.1 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Vasopressin (20 U/ml)	0.8 U/kg		0.1	0.2	0.4	0.6	0.8	1	1.2	1.4	1.6	1.8	2
	Atropine (0.54 mg/ml)	0.04 mg/kg		0.2	0.4	0.8	1.1	1.5	1.9	2.2	2.6	3	3.3	3.7
Anti-Arrhyth	Amiodarone (50 mg/ml)	5 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Lidocaine (20 mg/ml)	2 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Reversal	Naloxone (0.4 mg/ml)	0.04 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Flumazenil (0.1 mg/ml)	0.01 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Atipamezole (5 mg/ml)	100 цg/kg		0.06	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
Defib Monophasic	External Defib (J)	4-6 J/kg		10	20	40	60	80	100	120	140	160	180	200
	Internal Defib (J)	0.5-1 J/kg		2	3	5	8	10	15	15	20	20	20	25

Figure 3: CPR drug dosing chart. Drugs are separated by indication and volumes are provided by body weight to reduce calculation errors. Defibrillator dosing is for a monphasic electrical defibrillator. Anti-arrhyth, antiarrhythmic drugs; CPR, cardiopulmonary resuscitation; Epi, epinephrine; Defib, electrical defibrillation.

Sodium Bicarbonate – 1 mEq/kg

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Questions?

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