Cardiopulmonary Physiology andAnesthesia

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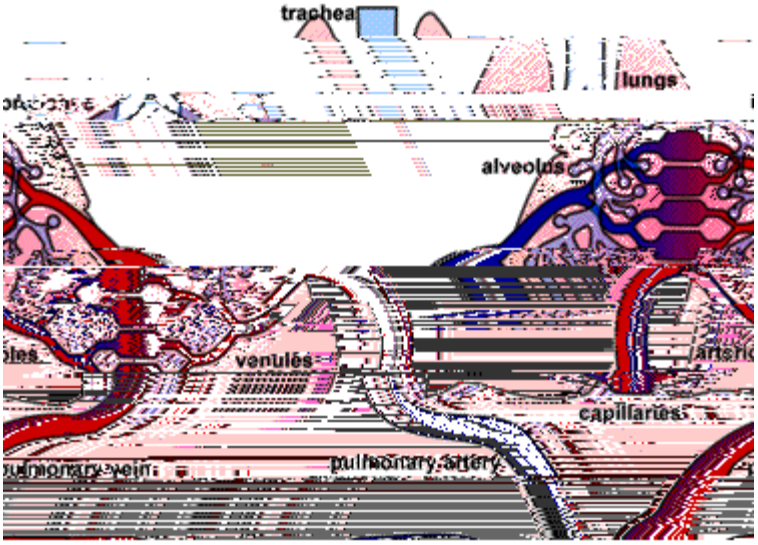
The vascular System

- Two separate circulations in series
  - systemic circulation and pulmonary circulation
  - As blood flows through the vascular beds, pressure and velocity drop, and overall cross-sectional area increases

Functional anatomy of the heart & lungs



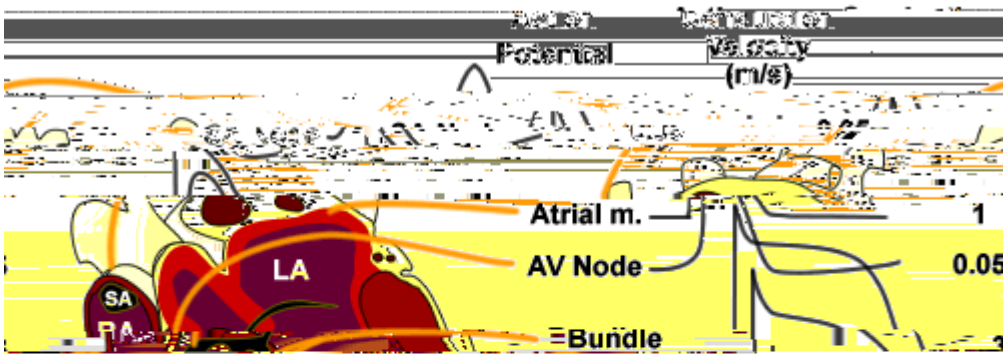
Electrophysiology

- Cardiac electric activity
  - electrical activity required for mechanical activity to occur
  - excitation-contraction coupling
  - altered by many anesthetic drugs

- Ion transport
  - distribution of sodium, potassium, and chloride responsible for electrical potential across cardiac cellular membranes
  - normal ion transport of ions required for normal electrical activity

- Electrocardiogram
  - an algebraic sum of all the action potentials produced by each cardiac cell
  - essential to understand the origin of the components of the normal ECG
  - P-wave: atrial depolarization
  - PR interval: conduction through the atria and AV node (affected by parasympathetic tone)
  - QRS complex: ventricular depolarization
  - QT interval: entire ventricular depolarization and repolarization
  - ST segment: entire ventricular depolarization: the pause between ventricular muscular firing and ventricular muscular repolarization
  - T-wave: ventricular repolarization

Source of the ECG tracing



- Excitability
  - ability of the heart to initiate an action potential in response to an inward current (depolarization)
  - absolute refractory period (ARP): upstroke of the AP to right after the plateau
  - efferent RP (ERP): slightly longer than ARP, time during which no AP can be initiated
  - relative RP (RRP): following ARP, when repolarization is complete, a period during when AP can be elicited, but more than the usual inward current is required
  - cardiac muscles is different than that of skeletal muscles or nerves
  - lower resting membrane potential (-90 vs -65 mV)
  - greater action potential (130 vs 80 mV)
  - longer duration (150-300 vs 1 msec)

- Pacemaker action potential (Sinoatrial node)
  - Phase 0: *upstroke*: much less steep, inward Ca^{++} current; upstroke occurs with rapid Na^{+} influx
  - Phase 3: *repolarization*: K^{+} conductance
  - Phase 4: *slow depolarization*: accounts for SA node pace maker activity (automaticity), Na^{+} conductance
  - There is no phase 1

- Non-pacemaker action potential (atria and ventricles)
  - Phase 0: *upstroke*: rapid Na^{+} influx depolarizes membrane
  - Phase 1: *initial repolarization*: transient outward movement of K^{+}
  - Phase 2: *plateau*: large but slow inward movement of Ca^{++}, K^{+} conductance, Na^{+} influx
  - Phase 3: *repolarization*: large outward movement of K^{+}
  - Phase 4: *resting*: K^{+} equilibrium

Cardiac Transmembrane Potentials

- Diastolic depolarization - pacemaker potentials
  - SA node, AV node, atrial and ventricular Purkinje network causes the unique automaticity of the heart
  - resting potential gradually depolarizes toward a threshold potential, when reached, an action potential is triggered
  - cardiac tissue with the more rapid rate of rise of phase 4 is the pacemaker and determines heart rate (usually SA node)

Diastolic Depolarization

- Automaticity increased by:
  - increased heart rate

Steps in excitation–contraction coupling

- mainly mediated by calcium
- AP promotes inward Ca current at cell membrane and T-tubule during the plateau
- Ca triggered Ca release from SR : L Q W U D F H X O O D U & D 9
- Ca binds to troponin C which removes inhibition of actin–myosin interaction by troponin I and tropomyosin
- myocardial cell contracts (magnitude depends on Ca concentration)

Cardiac cycle

- Atrial systole (phase 1)
  - preceded by P wave
  - creates fourth heart sound

- “blip” following closure of aortic valve corresponds to ‘dicrotic notch’ or ‘incisura’

- Rapid ventricular filling (phase 6)
  - when ventricular pressure decreases below atrial pressure, mitral valve opens, leading to left ventricular filling
  - 3rd heart sound is heard

- Reduced ventricular filling (diastasis) (phase 7)
  - ventricular filling continues, but at a lower rate
  - increased heart rate decreases the time for ventricular filling

- Heart sounds during cardiac cycle
  - First heart sound (S1) is related to mitral and tricuspid valve closure
  - The closure of the aortic and pulmonic valves contribute to the second sound (S2) production
  - The physiologic third heart sound (S3) is a low-pitched vibration occurring in early diastole during the time of rapid ventricular filling. Most of the time, it is non-audible for human ears
  - The physiologic fourth heart sound (S4) is a very soft, low-pitched noise occurring in late diastole, just before S1. S4 generation is related to the ventricular filling by atrial contraction.

Cardiac performance

- Ultimate goal of the heart is to provide adequate quantities of oxygenated blood to peripheral tissues – cardiac output is the critical variable
- Determinants of cardiac output

$$CO = HR \times SV$$

$$CO = \frac{BP}{SVR}$$

- SV (stroke volume) determined by cardiac contractility, preload and afterload
  - Cardiac contractility (inotropy): intrinsic ability of the heart to generate force; relates directly to physiochemical processes such as availablility of intracellular calcium; decreases in cardiac contractility is the key to heart failure following administration of negative inotropes (many anesthetics)
  - Preload: Frank-Starling relationship (increased ventricular volume increases the force of cardiac contraction)
  - Afterload: inverse relationship with cardiac output and direct correlation with myocardial oxygen consumption

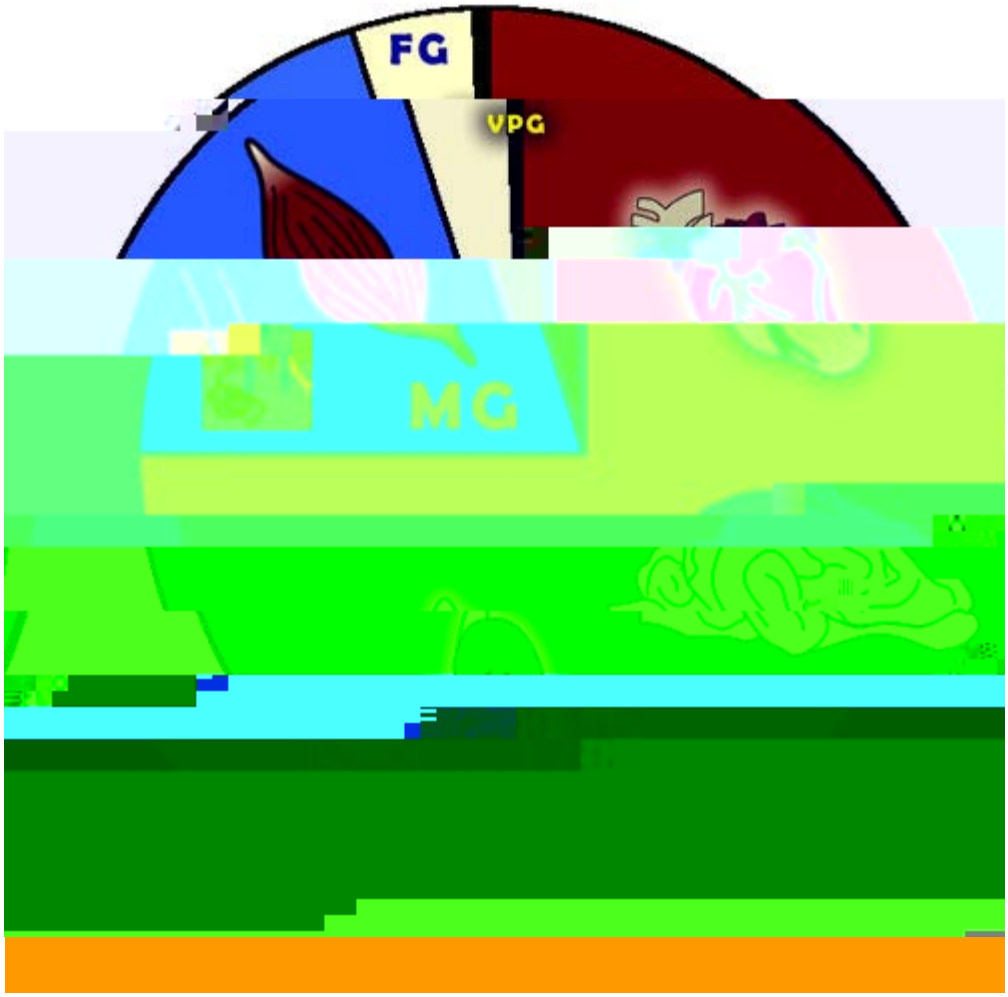
Determinants of Cardiac Output

Cardiac output and organ distribution

- Total blood volume (ml/kg):
  - Cats: 60-70
  - Dogs: 80-90
  - Horses (racing): 100
  - Horses (draft): 70
  - Cows: 60
  - Pigs: 60
  - Sheep, goats: 60
  - Humans: 80
- Vessel Rich Group (VRG): 75% of the CO
  - Brain
  - Heart
  - Kidney
  - Liver
  - Lungs
- Muscle Group (MG): 20% of the CO
  - Muscle

- Skin
- Fat Group (FG): 5% of the CO
  - Fat
- Vessel Poor Group (VPG): <1% of the CO
  - Bone, teeth
  - Tendons
  - Ligaments

Distribution of cardiac output



Blood pressure

- Arterial blood pressure is frequently assessed during anesthesia, either directly or indirectly

- Provides a rapid means to assess cardiac function
- Factors that determine blood pressure:
  - heart rate and stroke volume (CO)
  - vascular resistance
  - arterial compliance
  - blood volume
- All of the above factors can change dramatically during the course of anesthesia and surgery, either due to the affects of anesthetic drugs or surgical manipulations
- Blood pressure does not truly indicate tissue perfusion and one must use clinical judgment to correctly interpret blood pressure measurements (e.g. blood pressure can increase while CO decreases under the effects of several anesthetic drugs)

Relationship between pressure, flow velocity, and cross sectional area within the vascular system

Nervous, humoral, and local control of the cardiovascular system

The autonomic nervous system significant regulator of CV function

- Sympathetic and parasympathetic outflow affect heart rate, inotropy, and vascular tone to affect cardiac output, blood pressure, and distribution of blood flow
- Parasympathetic (vagus) effects
  - the vagus nerves inhibit the cardiac pacemaker, atrial myocardium and AV conduction tissue, acetylcholine serves a neurotransmitter at muscarinic receptors
  - has negative chronotropic effects (decreased heart rate)
  - has negative dromotropic effects (decreased conduction velocity)
  - has negative inotropic effects (decreased contractility)
- Sympathetic effects
  - innervation is throughout the hear-14(cem9)-2(hr)3(oug)10(hout)-12(t)-2(he)a458 h,r lh3

- usually released from the hypothalmus in response to increases in plasma solute

Autoregulation

- Ability of blood vessels to adjust flow in response to local metabolic needs and maintain flow in spite of extreme changes in perfusion pressure
- Most tissues regulate flow at a local level by responding to release of metabolites and tissue mediators (eg, histamine, carbon dioxide, NO, H^+)
- The heart, brain, and kidney demonstrate tight autoregulation

Clinical notes

- Most anesthetics depress cardiovascular performance ranging from hypotension,

Respiratory System

Respiration

- Total process where oxygen is supplied to and used by cells, and carbon dioxide is eliminated

Ventilation

- Movement of gases in and out of the alveoli
- Varies with metabolic needs of the animal

Terms

- Apnea: transient cessation
- Apneustic ventilation: long gasping inspirations with several subsequent ineffective exhalation
- Bradypnea: slow regular
- Dyspnea: labored
- Eupnea: ordinary and quiet
- Hyperpnea: fast ± deep, overrespiration
- Hypopnea: slow ± deep, underrespiration
- Polypnea: rapid, shallow panting
- Tachypnea: increased rate
- Hypoxia: any state in which oxygen in the lung, blood and/or tissue is low
- Hypoxemia: insufficient oxygenation of blood to meet metabolic requirement, PaO_2 < 70 mmHg at sea level
- Hypercapnia: elevated CO_2 tension in blood, $PaCO_2$ > 45 mmHg

- Vital capacity (VC): IRV +V_T + ERV, the maximum air expelled from the lungs after filling them to their maximum capacity, take maximum inspiration then take maximum expiration, the exhaled volume is VC
- Total lung capacity (TLC): VC + RV, the maximum volume to which the lungs can be expanded with the greatest possible inspiratory effort

Lung Volumes and Capacities

Components of the ventilatory system:

- neural control mechanisms
- bellows mechanism (chest wall and diaphragm)
- upper airway
- lung parenchyma

Control of respiration

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- normal ventilation relies on a slight negative pressure within the alveoli during inspiration to draw air into the lungs, and a slight positive pressure within the alveoli during expiration to move air back out of the lungs
- at inspiration, thoracic wall is expanded and diaphragm acts which leads to decrease in intrapleural pressure and increase in mouth pressure
- following inspiration, intrapleural space reduces which result in increased intrapleural pressure, and air flows reverse to the mouth
- assisted or controlled ventilation provides a positive pressure at the mouth to move air into the lungs this positive intrapleural pressure has significant cardiovascular effects

Ventilation : perfusion matching

- Matching of alveolar ventilation and capillary blood flow is influenced by gravity, and also that the pulmonary circulation is a low pressure system
- Anesthesia can cause significant abnormalities in ventilation perfusion matching (termed V/Q mismatching)
- Relationship between alveolar ventilation, hemoglobin oxygen saturation, oxygen content, and arterial partial pressure of carbon dioxide
- Hypoxic pulmonary vasoconstriction (HPV -0.002 Tw 1.5 0 (-1(t)-2Td ( )Tj 0.002 Tc -0.002 T

release of oxygen from hemoglobin, making reduced hemoglobin a CO_2 carrier that is 3.5 fold more effective than oxyhemoglobin. This adds another 25% of CO_2 transport.

- The carbon dioxide dissolved in plasma amount for 10% of CO_2.

Oxygen transport

Oxygen transport is determined by:

- Cardiac output (CO)
- Blood oxygen content (Ca_2O)
- The affinity of hemoglobin for oxygen (affinity determines the position of the hemoglobin dissociative curve - see below)

- Notice that oxygen dissociation in the plasma has little impact upon C_aO_2 because it is only 0.3, and hemoglobin carry majority of the oxygen (20.85)
- Majority of the oxygen are carried by hemoglobin
- Clinically, it is better to provide hemoglobin (blood transfusion) than providing 100% oxygen to an anemic patient (see the equation)

Relationship between alveolar ventilation, hemoglobin oxygen saturation, oxygen content, and P_aCO_2

- very little change in saturation and (oxygen content) above 70 mm Hg PO2
- marked change in saturation and (oxygen content) between 40-70 mm Hg PO2 (which is commonly found in metabolizing tissues)
- factors that affect the affinity of hemoglobin for oxygen:
  - 2,3-DPG - enhances dissociation of oxygen by competing for oxygen binding sites; decreases 2,3-DPG levels reduce the ability of hemoglobin to deliver oxygen to tissues
  - Carbon dioxide and lactate (metabolic by products) - enhances dissociation of oxygen
  - increased temperature - enhances dissociation of oxygen
- Plasma oxygen
  - only a small component of the total amount of oxygen carried by blood (0.3 ml/dl at 100 mm Hg PO2)
  - high inspired oxygen contents can increase the amount of plasma oxygen modestly (1.8 ml/dl at 650 mm Hg PO2), which results in about a 10% increase in the total oxygen content of blood at normal hemoglobin levels

- Anemia and reduced blood hemoglobin levels dramatically reduces the oxygen carrying capacity of blood, even with 100 % oxygen saturation

Estimation of normal PaO_2 using FiO2

- Clinically FiO_2 can be related to estimate PaO_2 as measured by the blood gas analyzer
- If FiO_2 is

Clinical notes

- Supplemental oxygen is usually a good idea even just with sedation
- Intubation and control of the patent airway is usually a good idea
- Controlled ventilation may be considered, depending on anesthetic combination used - however, it is almost always a good idea to have the ability to provide assisted or controlled ventilation whenever general anesthesia is utilized