Basic review of cardiac, lung, liver and beyond?

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The big question is?

How do you make a presentation like this exciting and not BORING?

Clinically relevant?

Be honest, how many of you felt that way when you saw the agenda?

Financial Disclosure

There is no financial conflicts with this presentation.

Lecturing about a topic does not constitute endorsement of any product. Please take the time to research each topic for more information.

Mentioning a product or company does NOT represent endorsement.
Clinical practice improvement

Annotating Continuous Quality Improvement in Clinical Practice

60%
25%
15%
0%

Respiratory System
Anesthesia
Muscular System
Skeletal System

FACTOR V LEIDEN

FACTOR V VARIENT LEADS TO HYPER—COAGULABILITY

FACTOR V NOT DEGRATED BY ACTIVATED PROTEIN C

30% WITH DVT OR PE HAVE FACTOR V LEIDEN

FACTOR V Leiden Thrombophilia
Specifically, Factor V Leiden Mutation, which results in abnormal activation of prothrombin. This predisposition to vascular disease is due to the presence of a variant of factor V, a blood clotting enzyme. It is a common cause of inherited venous thrombosis.
Thromboprophylaxis

SCIP

Surgery Patients with Recommended Venous Thromboembolism Prophylaxis Ordered

Surgery Patients Who Received Appropriate Venous Thromboembolism Prophylaxis Within 24 Hours Prior to Surgery to 24 Hours After Surgery

Caprini Risk assessment

Caprini Risk Factor Assessment

Thrombosis Risk Factor Assessment

Choose All That Apply

Risk Factor Assessment 4 Points

Risk Factor Assessment 4 Points

Risk Factor Assessment 4 Points

Risk Factor Assessment 4 Points
The function of the cardiovascular system is to deliver oxygen and nutrients and to remove carbon dioxide and other waste products.

CARDIOVASCULAR Response to Hypothermia

Systemic and pulmonary vasoconstriction; increased arterial BP; increased risk for ventricular dysrhythmias, MI, and cardiac mortality in postoperative patients; Decreased myocardial contractility → decreased cardiac output; Altered electrical conduction → bradycardia

- 44% lower rate of myocardial infarction in normothermic patients
Stents- History

Percutaneous transluminal coronary angioplasty (PTCA) by Gruntzig in 1977

Puel and Sigwart, in 1986, deployed the first coronary stent to act as a scaffold

In 2001, drug-eluting stents (DES) were introduced as a strategy to minimize restenosis.

Types

Bare metal stents:
- Traditional method
- May have an increased rate of re-narrowing due to growth of scar tissue in the stent, a condition called restenosis.

Drug-eluting stents:
- Combat Restenosis
  - Coated with medications that are slowly released to block the body’s ability to form scar tissue around the stent. The medication is delivered directly to the site of the artery blockage.

Diagram
Bridge Therapy

Integrillin—GP IIb IIIa inhibitor
Prevent activation and aggregation

Heparin
Prevent thrombin formation

IV therapy; hold 6 hours prior to surgery

Aspirin (ASA, acetylsalicylic acid)

The main mechanism of action of NSAIDs was clarified by Sir John Vane in 1971 who noted the inhibition of prostaglandin synthesis by aspirin.

Irreversibly acetylates platelet COX-1. Lasts for the life of the platelet (8-12 days)

Newsome 2008
Eisenberg 2010
Dimitrova 2012

Aspirin is recommended as a lifelong therapy that should NEVER be interrupted for patients with cardiovascular disease. Clopidogrel therapy is mandatory for six weeks after placement of bare-metal stents, three to six months after myocardial infarction, and at least 12 months after placement of drug-eluting stents.
Conduction System

**Intrinsic conduction system (nervous system)**
Heart muscle cells contract, without nerve impulses, in a regular, continuous way

**Special tissue sets the pace**
- Sinoatrial node (right atrium)
  - Pacemaker
- Atrioventricular node (junction of right and left atria and ventricles)
- Atrioventricular bundle (bundle of His)
- Bundle branches (right and left)
- Purkinje fibers

Cardiovascular Effects of Phenylephrine

Phenylephrine is a postsynaptic α-receptor agonist with little effect on the β receptors of the heart.

Anesthesia techniques & volume status and cardiac status

**IV induction agents**
- Propofol: ↓ SVR, cardiac contractility, preload
  - BP effects less pronounced in Euvolemic pts
- Ketamine
  - ↑ BP/HR/C0 2 stimulation of SNS & inhibition of norepinephrine reuptake
  - Direct myocardial depressant effects unmasked by exhaustion of catecholamine stores (CNS, end-stage shock) ~ paradoxotal ↓ BP

Static vs. Dynamic
Patients do better with increased stroke volume

The Heart: Regulation of Heart Rate

Stroke volume usually remains relatively constant
- Starling's law of the heart – the more that the cardiac muscle is stretched, the stronger the contraction

Changing heart rate is the most common way to change cardiac output

Normal Valve Function
- Maintain forward flow and prevent reversal of flow.
- Valves open and close in response to pressure differences (gradients) between cardiac chambers.
- Incompetent valve = backflow and repump
- Stenosis = stiff= heart workload increased
Mitral Valve

- Mitral Stenosis
  - Rheumatic
  - Congenital
  - Prosthetic valve stenosis
  - Mitral Annular Calcification
  - Left Atrial Myxoma
- Acute Mitral Regurgitation
  - Infective endocarditis
  - Papillary muscle rupture
  - Mitral valve prolapse
  - Chordal rupture
  - Cardiomyopathy
- Chronic Mitral Regurgitation
  - Ischemic heart disease
  - Papillary muscle dysfunction
  - Ventricular septal defect
  - Mitral valve prolapse
  - Infective endocarditis
  - Rheumatic
  - Anesthetic
  - Mitral annular calcification
  - Cardiomyopathy
  - LV dilation

Mitral Stenosis - Pathophysiology

- Restriction of blood flow from LA → LV during diastole.
  - Normal MVA: 4-6 cm²
  - Mild MS: 2-4 cm²
  - Severe MS: < 1.5 cm²
- MV Pressure gradient —
  - MV grad = MV flow/MVA.
- Flow = CO/DFP (diastolic filling period).
- As HR increases, diastole shortens disproportionately and MV gradient increases.

Mitral Regurgitation

Anesthesia mgmt goals of mitral regurgitation?

FAST, FULL, FORWARD
- Avoid sudden decreases in HR
- Avoid sudden increases in SVR
- Monitor V-wave size as reflection of regurgitant flow
- Minimize drug-induced myocardial depression

Induction considerations with mitral regurgitation?

- Avoid excessive and abrupt changes in SVR or decreases in HR
- SLOW, FULL, TIGHT
- Avoid slow heart or rapid ventric response rate during a fib (will lead to HTN)
- No increases in central blood volume
- No drug-induced decreases in SVR
- Avoid Hypoxemia and/or Hypocarbia that may exacerbate pain HTN and cause RVF
Aortic Stenosis Overview:

**Normal Aortic Valve Area:** 3-4 cm²

**Symptoms:** Occur when valve area is 1/4th of normal area.

**Types:**
- Supravalvular
- Subvalvular
- Valvular

Goals of management of anesthesia of aortic stenosis.
- Avoid events that would further decrease cardiac output!
- Maintain NSR and avoid bradycardia!
- Avoid sudden increases or decreases in SVR (do NOT do regional anesthesia r/t sympathectomy)
- Optimize intravascular fluid volume to maintain venous return and LV filling

Pathophysiology of Aortic Stenosis

- A pressure gradient develops between the left ventricle and the aorta. (Increased afterload)
- LV function initially maintained by compensatory pressure hypertrophy
- When compensatory mechanisms exhausted, LV function declines.

Aortic stenosis

**Grading and severity**
- Aortic valve area must be reduced to 25% of normal before significant circulatory changes occur.
- Grading of stenosis severity is as follows:
  - Normal valve area = 3-4cm²
  - Mild stenosis = 1.5-3cm²
  - Moderate stenosis = 1.0-1.5cm²
  - Severe stenosis ≤ 1.0cm²
- When stenosis is severe, the peak gradient across the aortic valve is usually > 60mmHg.
Aortic Regurgitation

Management of anesthesia goals with aortic regurgitation?

- maintain forward left ventricular stroke volume
- avoid sudden decreases in HR
- avoid sudden increases in SVR
- minimize drug-induced myocardial depression (consider balanced technique over high gas technique as opioids do NOT depress contractility)

Induction and maintenance considerations with aortic regurgitation:

Induction with drugs that will maintain forward left ventricular stroke volume

maintenance depends on degree of LV dysfunction

Little LV dysfunction: nitrous oxide plus volatile agent

Severe LV dysfunction: high opioid anesthetic

A rule of thumb for valve disease is that stenotic lesions are kept 'slow and tight', while regurgitant lesions are kept 'fast and full'. This means you will avoid excessive volume loading to avoid pulmonary edema, peripheral vasodilation to avoid hypotension and compensatory increases in heart rate, and tachycardia

Blood Vessels? Huh....
Von Willebrand Factor

2 components
(separate genetic control)

Von Willebrand factor (vWF)---9 different types

Desmopressin (DDAVP)
DDAVP releases vWF from endothelial cells

Increases Factor VIII activity in patients with hemophilia and von Willebrand's disease

Can be given IV or intranasally

• 0.3 mcg/kg IV, or 150 mcg per nostril

Response to DDAVP varies considerably

Coagulation Abnormalities

Bleeding associated w/synthetic colloids widely reported

Dextran produces dose-related ↓ platelet aggregation & adhesiveness

Hetastarch can lead to ↓ factor VIII & von Willebrand factor

Coag studies & bleeding times not affected by infusions of up to 1L

• Best avoided in pts w/known coagulopathy
• Voluven: short half life, less ↓ in coag factors
What organ controls body temperature?

The hypothalamus,

The hypothalamus sends signals to various parts of the body, such as the glands and nervous system, when its core internal temperature begins to fluctuate too high or low. This area of the brain contains the components to control the body’s temperature along with the key temperature sensors. Therefore, when a person gets too hot, the hypothalamus sends a signal to start sweating. It is also what makes people shiver in the winter if they get too cold.

Anesthesia and Temperature

Muscle relaxants: Inhibit shivering, so decreases tolerable range of temp/increases minimum tolerable ambient temperature

-Anticholinergics: Inhibit sweating, decreases maximum tolerable temp
-No behavioral regulation available

-GA agents cause elevated warm-response thresholds, and reduced cold response thresholds, inter threshold range now 2-4 degrees C
-Propofol, alfentanil, precelex: Slightly linear increase in sweating threshold, marked linear decrease in vasoconstriction and shivering thresholds
HYPOTHERMIA

50% to 90% of surgical patients (approximately 14 million) experience inadvertent perioperative hypothermia each year.

Between 30-40% of all surgical patients are hypothermic upon admission to PACU.

Inadvertent hypothermia has been called as the most frequent, preventable complication of surgery.

HYPOTHERMIA

Surgical site infections:

Most common healthcare-associated infections

Average per patient cost: $11,087-$34,670 (adjusted to 2007 dollars)

8000 associated deaths each year (aggregate estimated cost at $3.2-$10 billion)

Patient Satisfaction Scores:

SUMMARY OF OUTCOMES OF NORMOTHERMIA

<table>
<thead>
<tr>
<th>Decreased:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Time spent in ICU</td>
<td>43%</td>
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<tr>
<td>Need for mechanical ventilation</td>
<td>40%</td>
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<tr>
<td>Need for blood transfusion</td>
<td>40%</td>
</tr>
<tr>
<td>PRBC</td>
<td>85%</td>
</tr>
<tr>
<td>FFP</td>
<td>79%</td>
</tr>
<tr>
<td>Platelets</td>
<td>78%</td>
</tr>
<tr>
<td>Surgical site infections</td>
<td>64%</td>
</tr>
<tr>
<td>Postop MI</td>
<td>44%</td>
</tr>
<tr>
<td>Mortality rates</td>
<td>55%</td>
</tr>
</tbody>
</table>
Physiologic Thermoregulation

Thermoregulation under anesthesia

Heat redistribution phenomenon
Surgical Site Infections

- Vasoconstriction → ↓ blood flow to tissues → low oxygen tension → conducive for bacterial contamination; Decreased collagen synthesis → slower healing and weaker scar tissue; Impaired immunity

64% lower wound infection rate in normothermic patients

The pulmonary system

Respiratory System
The Functions of the Nose

• Filter the air
• Humidify the air
• Warm the air
• Do we need a HME!

Alveoli

• Ca. 300 million alveoli
• Between 75 µ to 300 µ in diameter
• Most gas exchange takes place at alveolar-capillary membrane

Anatomic Arrangement of Alveoli

• 85-95% of alveoli covered by small pulmonary capillaries

New ventilation efforts, closer to 6 ml/kg with peep at least 4

Recruitment maneuver
Cystic Fibrosis — Defect in Chromosome 7

- Hereditary diseases of exocrine glands of pulmonary and gastrointestinal systems.
- Thick and viscous secretions and decreased ciliary activity lead to pneumonia and wheezing.
- Dehydration and electrolyte abnormalities.

Ivacaftor (FDA 2012) works only in a specific mutation (~5% of patients).

The lungs...
Fospropofol (Lusedra) over 200 gene variations that stop the conversion

University of Linköping, Sweden 2016

Detailed Description
Propofol, [2,6-dialpropylphenol] is a short-acting anesthetic drug used for induction and maintenance of anesthesia. The aim of this study is to evaluate plasma concentrations of propofol in relation to depth of anesthesia, measured by continuous EEG and to correlate plasma concentrations with genetic analyses of liver enzymes responsible for drug elimination. Our hypothesis is that there is an individual requirement of propofol plasma concentration depending on genetic differences in drug elimination. 200 patients, ASA classification I, planned for elective surgery of a duration of at least 30 minutes will be included in this study.

Protect the lungs.....
What is Mendelson’s Chemical Pneumonitis??

What is that based on?
- Increased gastric fluid volume
- Increased gastric fluid acidity
- Decreased competency of lower esophageal sphincter
- Increased risk of aspiration

Aspiration prophylaxis recommended for C/S
- 0.2 M Sodium citrate 30 mins PO
- Ranitidine 50mg IV
- Metoclopramide 10mg IV

Sodium Citrate/Bictra

30 cc PO, tastes terrible

Have patient drink in one shot

The only drug capable of helping in the short term with minimizing aspiration pneumonitis

Metoclopramide

A dopamine antagonist—think deep.

How would this help our OH patient beyond the stomach issue? Dopamine receptors do what after delivery?

A prokinetic that increases lower esophageal sphincter tone, stimulates upper GI motility and increases GI transit time.

No effect on H+ ions
Uses of H2 antagonists

There are others…. But, Zantac cheaper and less interaction with P-450

Chemoprophylaxis to increase gastric fluid pH, but have NO influence on the gastric fluid currently present in the stomach (they are not antacids)

(the only way to deal with that is sodium citrate)

The Liver makes six blood clotting factors: I (fibrinogen), II (Prothrombin), IV, V, VI, and VII.
Calcium

- Intense positive inotropy, increased BP, decreased LVEDP, decreased SVR and heart rate.
- Commercial availability:
  - 10% calcium chloride
  - 10% calcium gluconate
- Adult dosing:
  - calcium chloride: 200-1000 mg IV
  - calcium gluconate: 600-3000 mg IV

Our friend the LIVER

Over 50 human P450 enzymes have been identified and are classified according to their number of shared amino acid sequences. Families are given the nomenclature CYP1, CYP2 .... and are further divided into sub-families CYP1A, CYP1B etc.

Sub-families can be further divided into iso-forms CYP1A1, CYP1A2 etc. Most drugs are metabolized by more than one enzyme and genetic polymorphisms do exist.

Pharmacodynamic Interactions: Warfarin

- Warfarin (coumadin) is a commonly prescribed oral anti-coagulant.
- Common cause of adverse drug reactions: too much drug results in increased bleeding, too little results inadequate anti-coagulation (leading to possible stroke, DVT etc.)
- Need for monitoring
Coagulation made easy

Rather than thinking about the intrinsic and the extrinsic pathways,
Think about the PTT and the PT pathways

Coagulation Factors—Vitamin-K dependent factors (II, VII, IX, X)(S and C)

<table>
<thead>
<tr>
<th>FACTORS</th>
<th>PLASMA t ½ (hrs)</th>
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</thead>
<tbody>
<tr>
<td>FIbrinogen (II)</td>
<td>72-120</td>
</tr>
<tr>
<td>Prothrombin (III)</td>
<td>60-70</td>
</tr>
<tr>
<td>V</td>
<td>12-16</td>
</tr>
<tr>
<td>VII</td>
<td>2-6</td>
</tr>
<tr>
<td>VIII</td>
<td>8-12</td>
</tr>
<tr>
<td>IX</td>
<td>18-34</td>
</tr>
<tr>
<td>X</td>
<td>50-60</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FACTORS</th>
<th>PLASMA t ½ (hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>XI</td>
<td>52</td>
</tr>
<tr>
<td>XII</td>
<td>60</td>
</tr>
<tr>
<td>Protein C</td>
<td>6</td>
</tr>
<tr>
<td>Protein S (total)</td>
<td>42</td>
</tr>
<tr>
<td>Tissue factor</td>
<td>--</td>
</tr>
<tr>
<td>Thrombomodulin</td>
<td>--</td>
</tr>
<tr>
<td>Antithrombin</td>
<td>72</td>
</tr>
</tbody>
</table>

Coagulation Mechanism

Our Friend Coumadin

Vitamin-K dependent factors (II, VII, IX, X)(S and C)
**VKORC1 or VKOR**

- Drug target: vitamin K epoxide reductase complex subunit 1 (VKORC1 or VKOR)
- Vit K needs to be converted from inactive epoxidized form to active reduced form
- Warfarin binds to VKORC1 near its catalytic site, inhibiting the reduction reaction.
- VKORC1 variants are associated with warfarin resistance in humans

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**Laboratory Monitoring, Thromboelastography (TEG)**

Continuous profiles during all phases of clot formation
Provides more accurate picture of in vivo coagulation process to evaluate:

- Reflects balance of the hemostatic system

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**Hemostasis Monitoring: TEG Hemostasis System**

Whole blood test
Measures hemostasis
- Clot initiation through clot lysis
- Net effect of components

TEG system
- Laboratory based
- Point of care
- Remote, can be networked
- Flexible to institution needs
Thromboelastogram (TEG)
Reflects balance of the hemostatic system

Utility of TEG Analysis

- Demonstrates all phases of hemostasis
  - Initial fibrin formation
  - Fibrin-platelet plug construction
  - Clot lysis

Identifies imbalances in the hemostatic system
- Risk of bleeding
- Risk of thrombotic event

Changes in Obesity to remember?
Pickwickian syndrome
“Obesity Hypoventilation Syndrome”

Severe obesity & respiratory compromise
↑ PaCO₂
↓ PaO₂
Polythemia
Sleep apnea
Pulmonary HTN
CHF
Predisposition to upper airway obstruction

Physiological Changes

Restrictive ventilation
- ↓ in FRC and TLC
- V/Q mismatch
- R-L shunting
- Hypercarbia
- ↓ chest wall compliance
- ↑ work of breathing
- Airway closure

Metabolic & mechanical effects on ventilation
- ↑ VO₂
- ↑ CO₂ production
- ↑ Vₐ
- ↑ work of breathing
- ↓ chest wall compliance
- ↓ lung volumes
- ↓ PaO₂

C-V & pulmonary abnormalities in the morbidly obese
Pediatric changes....??

Always keep in Mind...

Cardiac output is dependent on?

When compared to adults:
- Non-compliant Left ventricle
- Residual Fetal Circulation
- Large Head, Large Tongue
- Anterior and Cephalad Larynx
- Long Epiglottis, Short Trachea and Neck
- Prominent Tonsils and Adenoids
- Immature Hepatic Biotransformation
- Decreased Protein-Binding
- Rapid induction and Recovery
- Large Volume of distribution for water soluble drugs
- Immature Neuromuscular junction
Children have a higher cardiac output and oxygen consumption per kilogram than adults.

They support this higher output with a higher baseline heart rate.

Infants are heart rate dependent for their cardiac output. In other words, they have a fixed stroke volume, and must increase their heart rate to increase cardiac output.

They may respond to stress, such as hypoxia, by becoming bradycardic, and therefore decreasing CO. This can make resuscitation quite difficult.

Normal vital signs for children include higher heart rates and lower blood pressures than adults.

They need a high heart rate.

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### Cardiovascular

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Normal vital signs for children include higher heart rates and lower blood pressures than adults.

**Table 3. Normal Vital Signs For Age Of Pediatric Patients.**

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart Rate (bpm)</th>
<th>Respiratory Rate (bpm)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
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</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>90-180</td>
<td>20-50</td>
<td>60-11</td>
<td>27-11</td>
</tr>
<tr>
<td>1-6 months</td>
<td>100-180</td>
<td>20-40</td>
<td>80-10</td>
<td>45-13</td>
</tr>
<tr>
<td>6-11 months</td>
<td>100-150</td>
<td>25-35</td>
<td>90-16</td>
<td>65-15</td>
</tr>
<tr>
<td>1 year</td>
<td>100-150</td>
<td>20-30</td>
<td>95-18</td>
<td>65-20</td>
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<tr>
<td>2 years</td>
<td>85-100</td>
<td>15-25</td>
<td>100-25</td>
<td>65-25</td>
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<tr>
<td>3-4 years</td>
<td>85-110</td>
<td>15-25</td>
<td>100-20</td>
<td>65-15</td>
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<tr>
<td>4-5 years</td>
<td>85-110</td>
<td>12-20</td>
<td>100-10</td>
<td>65-15</td>
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<tr>
<td>5-7 years</td>
<td>85-110</td>
<td>12-20</td>
<td>110-20</td>
<td>70-15</td>
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<tr>
<td>7-10 years</td>
<td>55-110</td>
<td>12-16</td>
<td>110-10</td>
<td>75-15</td>
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</table>

*Adapted from: Shnider SC, Practical Information. In: Textbook of Pediatric Emergency Medicine, 2nd ed. Also; Jvin DC. Multiple Trauma. In: Emergency Medical Concepts and Clinical Practice. 7th ed. EE. All rights reserved. See References A and I, respectively.*
Cardiac

Infants are born with an anatomically patent foramen ovale and ductus arteriosus

- The ductus closes in the first day of life
- The foramen ovale may remain probe patent for life, but physiologically closes in the first day of life
- This can be important, because bubbles in IV fluid can cross the PFO and go directly to the brain

Bubble trap? Downs Patient? Patent PFO?

Thoughts??

Oh those pediatric lungs.....

- Neonates Smaller FRC = faster induction
- Increased closing volumes and decreased FRC make the neonate prone to atelectasis and hypoxia
- Neonates are diaphragmatic breathers
- Intercostal muscles are underdeveloped
- Diaphragm is high
- Chest cavity is small

Minute volume is increased, corresponding with the increased cardiac output. This is supported by an increased respiratory rate.

Crying is good!

Total volume and dead space are equivalent to adults on a per kilogram basis.

Children desaturate rapidly because of increased utilization of oxygen per kilogram, and because their FRC is decreased under anesthesia.

FRC decreases under anesthesia because of small alveoli, and a very compliant chest wall. (No rigid box effect like adults have.) Both of these things lead to increased ventilation.
Metabolism

Hydrolyzed in plasma by plasma Cholinesterases
- Plasma cholinesterase activity in infants < adults

Immature P450 system

Obstetrics and physiology... oh my.....

Physiological Changes

- Diaphragm is displaced cephalad by the uterus by about 4-6 cm
- This displacement causes the FRC to be decreased by 20-30%
- The total lung capacity is not changed as well as vital capacity
- This is related to a compensatory increase in thoracic anterior posterior diameter.
- They become chest breathers and not belly breathers
Physiological Changes

- The respiratory changes cause a respiratory alkalosis, this is actually compensated by a metabolic acidosis.
- The PaCO₂ will be around 30.
- The PaCO₂ will be higher in the non-pregnant patient.
- The Pao₂ will be higher in the pregnant patient.
- Increase in oxygen consumption and a increase in alveolar ventilation. Tidal volume increase by 30-40%.
- Respiratory rate will increase by 20%.

Physiological Changes – Respiratory system

- ↑ oxygen consumption ~ 20% (100% in labor) due to increased metabolic rate.
- ↑ minute ventilation ~ 50% (due to increased tidal volume).
- ↓ arterial pCO₂
- ↓ FRC causing a decrease in oxygen reserves

Physiological Changes

- Decrease in FRC may allow a very quick and fast desaturation.
- This in association with the FRC leads to quick maternal hypoxia and desaturation.
- This also leads to a very quick and fast uptake of inhalation anesthesia.
- The hyperventilation and physiological changes can lead to a maternal alkalosis that can lead uterine vasoconstriction and lead to a decrease in placental perfusion...putting the fetus at risk.
Physiological Changes—Cardiac, The Highlights!

Almost all the changes seen are due to high levels of progesterone and include:

- 35% ↑ Total Blood Volume
- ↑ heart rate 15 beats/min
- 40% ↑ CO
- 30% ↑ SV
- 15% ↓ SVR
- 500ml/min ↑ blood flow to uterus
- ↑ venous return from legs
- AORTOCAVAL COMPRESSION (mechanical)

Impact of cardiac changes

Patients with pre-existing cardiac disease may decompensate either during labor or immediately post delivery. This corresponds to the time of maximal ↑ CO

Approximately 400 – 600ml blood loss occurs at delivery
- Supine hypotensive syndrome

Cardiovascular Changes

Blood volume increased—plasma level increased—RBC volume increased
- CO, Stroke Volume, Ht all increased

The cardiac output increases nearly 40% during the first trimester because the heart rate increases and the after load decreases. This continues to peak until after delivery. Max CO is right after delivery and continues to adjust for the next several weeks.

Stroke volume increases post delivery. This is related to a unobstructed vena cava and aorta leading to the blood volume increase. The uterus no longer obstructs the major vessels
Cardiovascular Changes

The patient has a increase in RBC’s and is still anemic. This is related to the increase in plasma volume.

A increase in Blood volume does not increase blood pressure. Remember, the patient has a decrease in PVR.

At term the blood volume increases 1000ml due to vasodilatation.

The CO to the uterine vasculature is 700ml/min. The CO must be maintained and keeping the SBP greater than 100 will maintain the perfusion pressure.

Ultrasound….Here and more coming.....

New Ultrasound?---
“Another tool to distinguish us from the CRNA”
Ultrasound speeds up safety and how well and effective your block is...
Increase Public Relations and Productivity.

Spinal and Epidural Placement
Can I be excused?  ... my brain is full!

Questions

Thank you!
pstrube3000@yahoo.com