The Patient with Congenital Heart Disease
Coming for Non-Cardiac Surgery

Astrid G. Stucke, MD
Associate Professor, Pediatric Anesthesiology
Medical College of Wisconsin
Milwaukee, Wisconsin

How does the blood flow?

- Shunt (volume and direction)
- Valve function
- Ventricular function

AV Canal - Anatomy

3-mo old chld, 4 kg, complete AV canal for gastrostomy
- Defect of the endocardial cushion
- 5% of all heart defects
- Associated with Down Syndrome

All Schematics by Cincinnati Children's Hospital
AV Canal – valvar function?

- Rastelli classification
- Ventricular function?
- AV valve function?

AV Canal – Shunt direction?

SVR - PVR

AV Canal – Shunt direction?
AV Canal – Effects on PVR

SVR - PVR

- FiO₂
- PaCO₂
- Acidosis

AV Canal – Shunt direction?

SVR - PVR

- Preoxygenation?
- Respiratory Rate?
- Blood Pressure

Propofol reduces sympathetic tone and blood pressure

- 10 healthy volunteers (21-37y)
- Propofol infusion vs. placebo
- Invasive blood pressure measurement, forearm plethysmography, sympathetic activity (N. Peroneus)
- TCI for light (~1µg/ml) or deep sedation (~2µg/ml); BIS 70 or 50
Propofol reduces sympathetic tone and blood pressure

BP (mmHg)

sympathetic activity bursts/100 beats

vascular resistance forearm mmHg/ml/min/100 ml tissue

Anesthesiology 2005, 103: 20-4

Hemodynamic effects of sevoflurane and midazolam/fentanyl

- 54 children under 14 years for cardiac surgery
- No child with heart failure
- Echo before and during both anesthetic levels

- Halothane 1 und 1.5 MAC
- Sevoflurane 1 und 1.5 MAC
- Isoflurane 1 und 1.5 MAC
- Midazolam/Fentanyl 100/4 mg/ml und 200/6 ng/ml IV

Sevoflurane and midazolam/fentanyl lower blood pressure

HF

EF

CI

SVRI

MAP

Anesthesiology 2001, 94:223-9

*: p<0.05 vs. baseline
Sevoflurane reduces ejection fraction, but only in high concentrations

*Anesthesiology 2001, 94:223-9*

<table>
<thead>
<tr>
<th>Age</th>
<th>MAC</th>
<th>HR</th>
<th>MAP</th>
<th>CI</th>
<th>SVRI</th>
<th>EF</th>
</tr>
</thead>
<tbody>
<tr>
<td>60</td>
<td>0.1</td>
<td>110</td>
<td>65</td>
<td>40</td>
<td>2.3</td>
<td>0.8</td>
</tr>
<tr>
<td>60</td>
<td>0.2</td>
<td>110</td>
<td>65</td>
<td>40</td>
<td>2.3</td>
<td>0.8</td>
</tr>
<tr>
<td>60</td>
<td>0.3</td>
<td>110</td>
<td>65</td>
<td>40</td>
<td>2.3</td>
<td>0.8</td>
</tr>
<tr>
<td>60</td>
<td>0.4</td>
<td>110</td>
<td>65</td>
<td>40</td>
<td>2.3</td>
<td>0.8</td>
</tr>
<tr>
<td>60</td>
<td>0.5</td>
<td>110</td>
<td>65</td>
<td>40</td>
<td>2.3</td>
<td>0.8</td>
</tr>
</tbody>
</table>

*: p<0.05 vs. baseline

Sevoflurane...

- and Midazolam/ Fentanyl lower blood pressure
- reduces ejection fraction, but only > 1 MAC

Signs of heart failure

Left heart:
- Wet lung
- Poor weight gain
- Cold extremities

Right heart:
- Hepatomegaly

Goal of induction:
- Avoid hypotension
- Avoid negative inotropy
Effects of volatile anesthetics on cardiac function in heart failure

- 10 dogs with catheter for cardiac index measurements
- Heart rate per pacemaker (+10%)
- Measurements during apnea
- Congestive heart failure through pacing 240/min until LVED pressure >15mmHg + clinical failure
- Each anesthetic tested on subsequent days
- Systolic: regional contractility (SS%)
- Diastolic: LV isovolumetric relaxation (τ)

Eur J Anaesth 2004;212:797-806
Volatile anesthetics...

- Are negative inotrope above 1 MAC
- Slow diastolic relaxation
- Both effects are much more pronounced in cardiac failure.

Etomidate

- 12 children (~9y) for ASD closure/ SVT ablation
- Sedated with IV morphine + midazolam
- Catheter in pulmonary artery and aorta
- 0.3 mg/kg etomidate IV

Etomidate has no effect on hemodynamics

Etomidate...

- has no hemodynamic effects
- suppresses adrenal cortex for at least 1 day

Plasma Cortisol Level (µg/dl)

<table>
<thead>
<tr>
<th></th>
<th>pre</th>
<th>induct</th>
<th>CPB</th>
<th>postop</th>
<th>24h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate</td>
<td>20</td>
<td>40</td>
<td>60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>0</td>
<td>20</td>
<td>40</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Anatomy – Pulmonary Hypertension

- 11 m/o, 5kg boy, s/p TAPVR, now pulmonary HTN, supra-systemic RV pressure, severe RV-hypertrophy
- Comes for PICC-line
- Reduced exercise tolerance/severe dyspnea with feeds

Pulmonary Hypertension

Ventilatory strategy:
- High FiO₂
- Avoid increase in CO₂
- Avoid pulmonary-hypertensive crisis
- Avoid systemic hypotension
Ketamine – Pulmonary Hypertension

- 15 Children, 3mo-18y
- PAP >25 mmHg
- Idiopathic or ASD/ VSD/ PDA

- Catheter under spontaneous ventilation, 
  ÷ MAC sevoflurane
- Ketamine 2 mg/kg + 10 µg/kg/min

PVRI (Wood Units/m²)

pre 5 min 10 min 15 min

Ketamine
2 mg/kg in 5 min
+ 10 µg/kg/min

Ketamine - Inotropy

Atrial and ventricular muscle in heart failure

Contractility

Relaxation
Ketamine - CI

- 11 Children for cardiac cath
- No shunt-lesions
- Midazolam + thiopental for catheter insertion, then ketamine infusion until end of study
- Spontaneous ventilation (etCO₂ unchanged)

<table>
<thead>
<tr>
<th>Control</th>
<th>Ketamine 50-75 µg/kg/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SWAP (mmHg)</td>
<td>70.77 ± 14.03</td>
</tr>
<tr>
<td>PMAP (mmHg)</td>
<td>19.84 ± 16.13</td>
</tr>
<tr>
<td>DP (cmH₂O)</td>
<td>6.29 ± 5.40</td>
</tr>
<tr>
<td>Qs/Qr (L/min)</td>
<td>5.25 ± 2.85</td>
</tr>
</tbody>
</table>

Ketamine – Cardiac Index

Ketamine/midaz vs. sufentanil/ midaz; ketamine 2.4 mg/kg/h

Ketamine – spontaneous ventilation

<table>
<thead>
<tr>
<th></th>
<th>Vt (ml/kg)</th>
<th>F (l/min)</th>
<th>Ti (s)</th>
<th>Te (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine</td>
<td>8.1±1.5</td>
<td>26±3</td>
<td>1.2±0.3</td>
<td>1.3±0.2</td>
</tr>
<tr>
<td>Halothane</td>
<td>4.9±1.1</td>
<td>18±3</td>
<td>0.8±0.1</td>
<td>1.0±0.2</td>
</tr>
</tbody>
</table>

*J Cardiothor Vasc Anesth 2003; 17:686-90

Can J Anaesth 1988; 35:368-74
Ketamine...

- Does not elevate PVR
- Is negative inotrope but “usually” maintains cardiac index and blood pressure
- Maintains spontaneous ventilation and FRC in anesthetic concentrations

Dexmedetomidine...  

- Peripheral vasculature: increase in BP ($\alpha_{2B}$)
- Central $\alpha_{2A}$-receptors: decrease in sympathetic tone (BP and HR)
- Blunts increase in sympathetic tone with stimulation
- Decrease in HR but not conduction
Dexmedetomidine...

- Does not depress breathing
- Does not lead to UAO
- Simulates non-REM sleep
- Does not reliably provide amnesia

Dexmedetomidine – Pulmonary Hypertension


<table>
<thead>
<tr>
<th>Baseline</th>
<th>Dexmed</th>
<th>Change</th>
</tr>
</thead>
</table>
| HR       | 86      | 72     | +14 %
| MAP      | 57      | 72     | +25 %
| PAP      | 24      | 30     | +25 %
| PVR/SVR  | 86/105  | 81/135 | +13 %

Anatomy – Fontan Palliation

- 12 y/o boy
- Tricuspid atresia
- S/p palliation with mBT-shunt, Glenn, Fontan
- Now: mild PLE, Pox: 93%, limited exercise tolerance
- Echo: mildly LVH, mild MI, fenestration not visible
- Appendicitis

Anatomically acute left to right shunt

HD: Hypotension
MAP: Mean Arterial Pressure
PAP: Pulmonary Artery Pressure
PVR: Pulmonary Vascular Resistance
SVR: Systemic Vascular Resistance
HR: Heart Rate

* Significant compared to baseline (p<0.01, 95% confidence interval +0.01)

https://www.anesth-analg.com/content/117/2/953

Fontan
Glenn
Fenestration
Fontan – special considerations

- Negative inotropy or reduced preload poorly tolerated
- Ventricular preload dependent on intrathoracic pressure
- Loss of spontaneous ventilation poorly tolerated

Venous return in children with Fontan palliation

- Spontaneous ventilation promotes venous return, especially in the inferior vena cava
- Light exercise enhances this phenomenon

Induction of the “Fontan”

- Induction with...?
- Rapid sequence induction?
- Epinephrine infusion?
- Transition to positive pressure ventilation
- Invasive blood pressure measurement?
- NIRS?
Why we like NIRS:

- Near Infrared Spectrometry
- Indicator of tissue perfusion
- \[ \text{MVO}_2 = \text{SaO}_2 - \text{VO}_2 / \text{Hb} \times \text{CO} \]
- After induction: \( \text{VO}_2 \) down => \( \text{MVO}_2 \) up

Why we like NIRS:

- \( \text{MVO}_2 = \text{SaO}_2 - \text{VO}_2 / \text{Hb} \times \text{CO} \)
- With hyperventilation:
  - Cerebral perfusion down => \( \text{MVO}_2 \) (cerebral) down

Why we like NIRS:

- \( \text{MVO}_2 = \text{SaO}_2 - \text{VO}_2 / \text{Hb} \times \text{CO} \)
- With aortic cross clamp:
  - Somatic perfusion down => \( \text{MVO}_2 \) (somatic) down
Why we like NIRS:

- $\text{MVO}_2 = \text{SaO}_2 - \text{Hb} \times \text{CO}$
- Your problem?
- $\text{SaO}_2$?
- $\text{Hb}$?
- $\text{CO}$?

Discussion