“Blood Gas Analysis In The Univentricular Patient: The Need For A Different Perspective.”
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Objective

• The participant will be able to interpret blood gas tests taken from patients with the unique anatomical configuration of a univentricular heart.

• No disclosures

Blood Gas Compensation Mechanisms In Critically Ill Patients?

• Pulmonary compensation
  – For acidosis
    • Patient spontaneously hyperventilates
  – For alkalosis
    • Patient spontaneously hypoventilates

• Critical cardiopulmonary patients on positive pressure ventilation often have no spontaneous control over their ventilation!

Blood Gas Compensation Mechanisms
In Critically Ill Patients?

• Renal compensation
  – For acidosis
    • Patient spontaneously retains bicarbonate
  – For alkalosis
    • Patient spontaneously excretes bicarbonate

• Critical cardiopulmonary patients are frequently on diuretics if not in complete renal failure!

VENOARTERIAL CO2 GRADIENT

• A normal ABG in the critically ill cardiopulmonary patient provides false reassurance of a normal patient physiology

• Two assessments:
  – Cardiac index calculation
  – Intracellular CO2 retention
Cardiac Index = k x p(v-a)CO2
k = 12.9 adult, k = 18 infant

- pH / pCO2 / pO2 / Base
- ABG: 7.32 / 46 / 263 / -3.2 (Normal for bivent patient)
- VBG: 7.30 / 52 / 39 / -1.7
- p(v-a)CO2 = 6
  - Adult cardiac index (CI): 12.9 / 6 = 2.15 L/min
  - Infant CI: 18 / 6 = 3.0 L/min
- ABG: 7.35 / 43 / 55 / -1.9 (Normal for univent patient)
- VBG: 7.19 / 74 / 20 / -1.7
- p(v-a)CO2 = 31
  - Adult CI: 12.9 / 31 = 0.42 L/min
  - Infant CI: 18 / 31 = 0.58 L/min

INTRACELLULAR CO2 RETENTION:
PERFUSED CAPILLARY DENSITY (PCD)

Table 3. Shock Induced Intracellular Hypercapnea in Various Organs*

<table>
<thead>
<tr>
<th>Species</th>
<th>Organ</th>
<th>Intracellular pCO2 mmHg Before Shock</th>
<th>Intracellular pCO2 mmHg After Shock</th>
</tr>
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<tbody>
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<td>dog</td>
<td>muscle</td>
<td>31</td>
<td>53</td>
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<tr>
<td>dog</td>
<td>muscle</td>
<td>51</td>
<td>85</td>
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<td>stomach</td>
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<td>53</td>
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<td>small gut</td>
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<td>95</td>
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<td>liver</td>
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<td>heart</td>
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<td>heart</td>
<td>66</td>
<td>146</td>
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<tr>
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<td>heart</td>
<td>70</td>
<td>150</td>
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<td>heart</td>
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<td>pig</td>
<td>heart</td>
<td>66</td>
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</tr>
<tr>
<td>dog</td>
<td>heart</td>
<td>66</td>
<td>146</td>
</tr>
<tr>
<td>Average</td>
<td>Intracellular pCO2 mmHg Before Shock</td>
<td>50 +/- 16</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>Intracellular pCO2 mmHg After Shock</td>
<td>188 +/- 132</td>
<td></td>
</tr>
</tbody>
</table>

*Data from Johnson and Weil, 1991.
Brain Intracellular $pCO_2 = pCO_2 + [4 \times (v-a)CO_2]$

- $pH / pCO_2 / pO_2 / Base$
- ABG: 7.32 / 46 / 263 / -3.2
- VBG: 7.30 / 52 / 29 / -1.7
- $p(v-a)CO_2 = 6$
- $46 \text{ mmHg} + [4 \times 6 \text{ mmHg}] = 70 \text{ mmHg brain } pCO_2$

- ABG: 7.35 / 43 / 55 / -1.9
- VBG: 7.19 / 74 / 20 / -1.7
- $p(v-a)CO_2 = 31$
- $43 \text{ mmHg} + [4 \times 31 \text{ mmHg}] = 167 \text{ mmHg brain } pCO_2$

Effect of Hyperventilation On Cardiac Index and Brain $pCO_2$

Before hyperventilation
- ABG: 7.35 / 43 / 55 / -1.9
- VBG: 7.19 / 74 / 20 / -1.7
- $p(v-a)CO_2 = 31$
  - Adult cardiac index: 12.9 / 31 = 0.42 L/min
  - Infant cardiac index: 18 / 31 = 0.58 L/min
- $43 \text{ mmHg} + [4 \times 31 \text{ mmHg}] = 167 \text{ mmHg brain } pCO_2$

After hyperventilation
- ABG: 7.45 / 33 / 55 / -1.9
- VBG: 7.29 / 64 / 20 / -1.7
- $p(v-a)CO_2 = 31$
  - Adult cardiac index: 12.9 / 31 = 0.42 L/min
  - Infant cardiac index: 18 / 31 = 0.58 L/min
- $33 \text{ mmHg} + [4 \times 31 \text{ mmHg}] = 157 \text{ mmHg brain } pCO_2$
- ↑ Risk of cerebral vasoconstriction

LETHAL VENOARTERIAL CO2 GRADIENTS
(pH / pCO2 / pO2 / Base)

- VBG: 7.29 / 57 / 37 / 0 :Hypercapnea - respiratory acidosis?
- CO2 Gradient = 25 mmHg :Large brain hemorrhage
- ABG: 7.35 / 43 / 55 / -1.9 :Normal
- ABG: 7.19 / 74 / 20 / -1.7 :Moderate hypercapnea - respiratory acidosis?
- CO2 Gradient = 31 mmHg :Refractory pulmonary hemorrhage
- ABG: 7.57 / 41 / 97 / +13.7 :Metabolic alkalosis
- VBG: 7.48 / 55 / 33 / +14.5 :Hypercapnea – metabolic alkalosis?
- CO2 Gradient = 14 mmHg :Seizures, failure to improve
- ABG: 7.31 / 48 / 375 / -2 :Breakthrough hypercapnea - not respiratory acidosis
- VBG: 6.90 / 106 / 27 / -7 :Severe hypercapnea
- CO2 Gradient = 58 mmHg :Large brain hemorrhage

AVERAGE VENOARTERIAL CO2 GRADIENT VS SURVIVAL IN ECMO PATIENTS

Lamia B, Minerva Anestesiol 2006
* CMH survival to discharge vs. average CO2 gradient on ECMO: n = 454, p < 0.05
• Definition
  – Single ventricle physiology is characterized by equal oxygen saturations in the aorta and pulmonary artery

• Anatomy
  – Any valvar defect that causes stenosis or atresia can lead to single ventricle physiology

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Tricuspid Atresia

- NO NEED FOR B-T SHUNT
- MUST HAVE LARGE ASD
- NO NEED TO KEEP PDA OPEN
- NO NEED FOR PA BANDS IF VSD IS RESTRICTIVE

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Pulmonary Atresia and Intact Ventricular Septum

- MUST HAVE LARGE ASD
- MODIFIED BTS
- PDA OPEN WITH PGE

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Tetralogy Of Fallot With Pulmonary Atresia

- MODIFIED BTS
- PDA OPEN WITH PGE
- NO NEED FOR LARGE ASD

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Hypoplastic Left Heart Syndrome

- RA MIXED SVC, IVC, AND PULMONARY BLOOD VBG
- 7.50 / 35 / 55 / +4.3
- SVO2 = 78%

- SVC UNMIXED CEPHALIC BLOOD VBG
- 7.39 / 51 / 28 / +3.7
- SVO2 = 61%

- L/R RADIAL, UAC OR FEMORAL ARTERY ABG
- 7.48 / 36 / 56 / +3.1
- SaO2 = 80%

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Oxygen saturations in a balanced circulation
Single Ventricle Reconstruction

- Usually, three initial surgeries in 2 years
  1. Modified Blalock-Taussig shunt: newborn
     a) Norwood Procedure for HLHS or mitral atresia
     b) Includes MBTS
  2. Bidirectional Glenn: 4-6 months
  3. Completion Fontan: 2 years
- Potential for multiple surgical revisions or transplantation throughout lifetime

Single Ventricle With High Pressure Pulmonary Shunt

- The single ventricle does double duty
  - pumps blood to body and lungs
- Pulmonary : Systemic blood flow balanced
  - Qp:Qs ratio of 1:1
  - High energy use by the heart, but most efficient for the blood flow distribution

Single Ventricle High Pressure Pulmonary Shunt

Norwood Reconstruction for Hypoplastic Left Heart Syndrome

Single Ventricle With Low Pressure SVC Pulmonary Shunt

- The single ventricle is unloaded
  - pumps blood only to body
  - lungs receive passive venous flow
- Pulmonary : Systemic blood flow
  - Qp:Qs ratio of 1:2
  - less energy used by the heart, but arterial SAO2 still ~ 80%

LOW PRESSURE SHUNT Qp:Qs = 1:2

- MBTS REMOVED
- SVC CONNECTED TO THE RPA
- ALL CEPHALIC VENOUS BLOOD FLOW GOES TO THE LUNGS

ABG (L/R RADIAL OR FEMORAL ARTERY BLOOD)
SAO2 ~ 80%
RA VBG (MIXED SVC, IVC AND PULMONARY BLOOD)
SVO2 ~ 75%
HIGH PRESSURE MODIFIED B-T SHUNT
SVC VBG (UNMIXED CEPHALIC BLOOD)
SVO2 ~ 50%
**Single Ventricle**

**SVC Low Pressure Shunt**
- ABG L/R RADIAL OR FEMORAL ARTERY
  - SASO2 ~ 80%
- ONLY ACCESS TO RA VIA FEMORAL VEIN
- LOW PRESSURE SHUNT
- SVC VBG (UNMIXED CEPHALIC BLOOD)
  - SVO2 ~ 50%
- ONLY ACCESS TO RA VIA FEMORAL VEIN

**LOW PRESSURE SHUNT** Qp:Qs = 1:1
- IVC CONNECTED TO THE RPA
- ALL SYSTEMIC VENOUS BLOOD FLOW GOES TO THE LUNGS

**Survival**
- Mortality after Norwood: 20 - 50%
- Mortality before second stage: 10 -15%
- Mortality after BDG: 2 - 5%
- Mortality after Fontan: 3 - 10%
- Overall survival: 50 - 70%

**Central Shunts**
- DRAW SVC VBG IF COMMON ATRIUM
- DRAW RA VBG IF ATRIAL SEPTUM INTACT
- DRAW R OR L ABG
Major Aorta to Pulmonary Collateral Arteries (MAPCAs)

MAPCAs can be a few large vessels or a plexus of hundreds of small vessels. Either can siphon a significant amount of systemic blood flow from the aorta.

The tissue composition of MAPCAs can be systemic, pulmonary or both. This makes the response of the pulmonary vascular bed to oxygen, CO2 or drugs unpredictable.

Use venocentral CO2 gradient and SV02 to assess cardiac index.

Long Term Concerns

- Oldest patients just now reaching late their 20’s
- Long term ventricular function, one ventricle doing the work of two
- Protein losing enteropathy from high systemic venous pressure
- Dysrhythmias from atrial distortion and abnormal conduction system
- Need for future cardiac transplantation

Univentricular Patients w/ Conditions Requiring Positive Pressure Ventilation

- Pulmonary infections may increase pulmonary vascular resistance
  - Dehydration quickly causes hemodynamic collapse
  - “Pop off” if present, reduces SAO2
  - If no pop-off, preload to the ventricle is reduced
  - Aggressive fluid infusion needed to maintain pulmonary blood flow
  - Risk of edema

- Aggressive positive pressure ventilator settings may be needed to improve oxygenation and/or CO2 removal
  - This reduces venous return to the heart
  - Cardiac output falls, precipitating shock
  - Aggressive fluid infusion needed to maintain ventricular preload
  - Risk of edema

Questions?