"Blood Gas Analysis In The Univentricular Patient: The Need For A Different Perspective."

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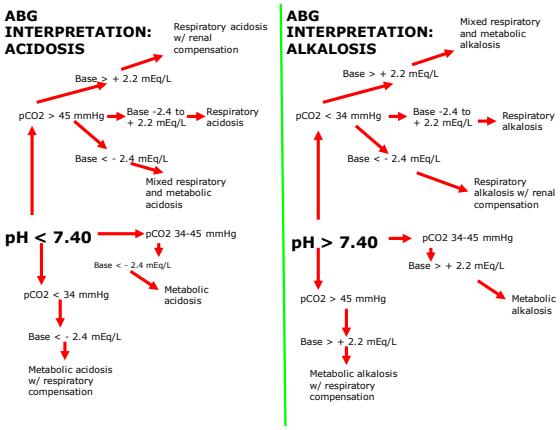
Objective

- 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?

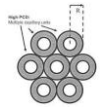
- Pulmonary compensation
  - For acidosis
    - Patient spontaneously hyperventilates
  - For alkalosis
    - Patient spontaneously hypoventilates
- Critical cardiopulmonary patients on positive pressure ventilation often have no spontaneous control over their ventilation!

Blood Gas Compensation Mechanisms In Critically Ill Patients?

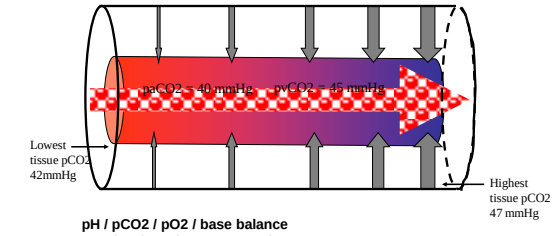
- Renal compensation
  - For acidosis
    - Patient spontaneously retains bicarbonate
  - For alkalosis
    - Patient spontaneously excretes bicarbonate
- Critical cardiopulmonary patients are frequently on diuretics if not in complete renal failure!

VENOARTERIAL CO2 GRADIENT

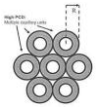
- A normal ABG in the critically ill cardiopulmonary patient provides false reassurance of a normal patient physiology
- Two assessments:
  - Cardiac index calculation
  - Intracellular CO2 retention



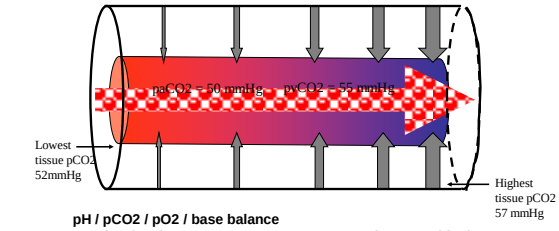
NORMAL BLOOD GASES:
Arterial normocarbia: paCO2 = 40 mmHg
Venous normocarbia: pvCO2 = 45 mmHg
Normal CO2 gradient = 5 mmHg



pH / pCO2 / pO2 / base balance
7.32 / 46 / 263 / -3.2 : Normal ABG
7.30 / 52 / 39 / -1.7 : Normal VBG
CO2 Gradient = 6 mmHg: No complications



RESPIRATORY ACIDOSIS:
Arterial hypercarbia: paCO2 = 50 mmHg
Venous hypercarbia: pvCO2 = 55 mmHg
Normal CO2 gradient = 5 mmHg



pH / pCO2 / pO2 / base balance
7.33 / 56 / 87 / +1.9 : ABG Hypercapnea – respiratory acidosis
7.31 / 65 / 44 / +3.7 : VBG Hypercapnea
CO2 Gradient = 9 mmHg: No complications

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)

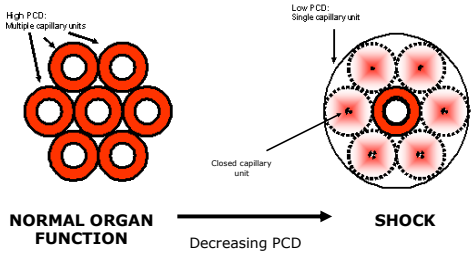
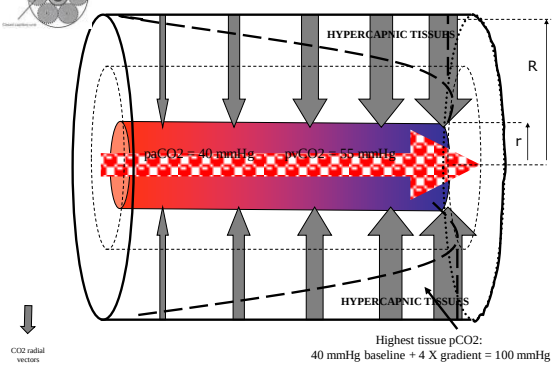


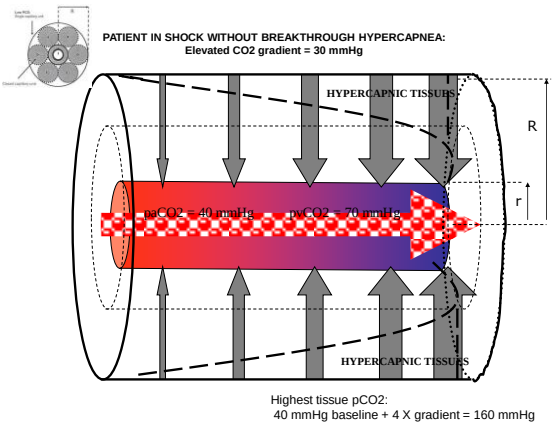
Table 3. Shock Induced Intracellular Hypercapnea in Various Organs*

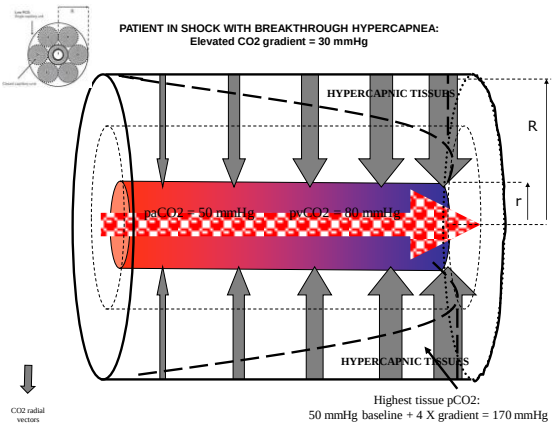
Species	Organ	Intracellular pCO2 mmHg Before Shock	Intracellular pCO2 mmHg After Shock
dog	muscle	31	53
dog	muscle	51	65
pig	stomach	33	53
rabbit	small gut	25	99
human	brain	58	80
rat	brain	49	149
rat	brain	80	389
dog	kidney	41	102
dog	kidney	42	122
dog	kidney	60	320
human	heart	65	182
dog	heart	33	248
pig	heart	61	359
dog	heart	66	416
	Range	25-80	53-416
	Average	50 +/- 16	188 +/- 132

*Data from Johnson and Weil, 1991.

PATIENT IN SHOCK WITH VENOUS HYPERCAPNEA:
Elevated CO2 gradient = 15 mmHg







Brain Intracellular pCO2 = paCO2 + [4 X p(v-a)CO2]

- **pH / pCO2 / pO2 / Base**

- **ABG: 7.32 / 46 / 263 / -3.2**
- **VBG: 7.30 / 52 / 39 / -1.7**
- **p(v-a)CO2 = 6**
- **46 mmHg + [4 x 6 mmHg] = 70 mmHg brain pCO2**

- **ABG: 7.35 / 43 / 55 / -1.9**
- **VBG: 7.19 / 74 / 20 / -1.7**
- **p(v-a)CO2 = 31**
- **43 mmHg + [4 x 31 mmHg] = 167 mmHg brain pCO2**

Effect of Hyperventilation On Cardiac Index and Brain pCO2

Before hyperventilation
- **ABG: 7.35 / 43 / 55 / -1.9**
- **VBG: 7.19 / 74 / 20 / -1.7**
- **p(v-a)CO2 = 31**
  - **Adult cardiac index: 12.9 / 31 = 0.42 L/min**
  - **Infant cardiac index: 18 / 31 = 0.58 L/min**
- **43 mmHg + [4 x 31 mmHg] = 167 mmHg brain pCO2**

After hyperventilation
- **ABG: 7.45 / 33 / 55 / -1.9**
- **VBG: 7.29 / 64 / 20 / -1.7**
- **p(v-a)CO2 = 31**
  - **Adult cardiac index: 12.9 / 31 = 0.42 L/min**
  - **Infant cardiac index: 18 / 31 = 0.58 L/min**
- **33 mmHg + [4 x 31 mmHg] = 157 mmHg brain pCO2**
- **↑ Risk of cerebral vasoconstriction**

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

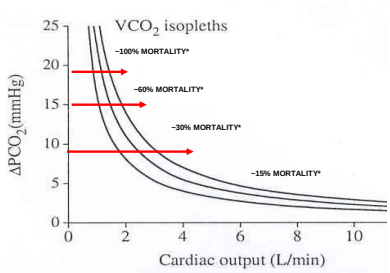
- **ABG: 7.35 / 32 / 154 / -7 :Hypocapnea - Part. Resp. Comp. Metabolic Acidosis**
- **VBG: 7.29 / 57 / 37 / 0 :Hypercapnea - respiratory acidosis?**
- **CO2 Gradient = 25 mmHg :Large brain hemorrhage**

- **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 / ? :Severe hypercapnea**
- **CO2 Gradient = 58 mmHg :Large brain hemorrhage**

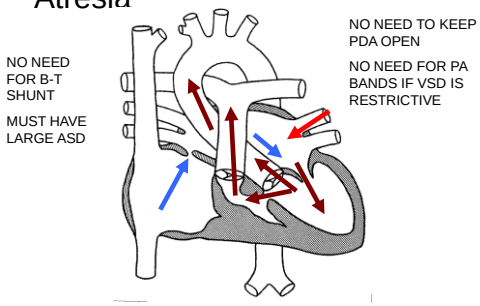
AVERAGE VENOARTERIAL CO2 GRADIENT VS SURVIVAL IN ECMO PATIENTS



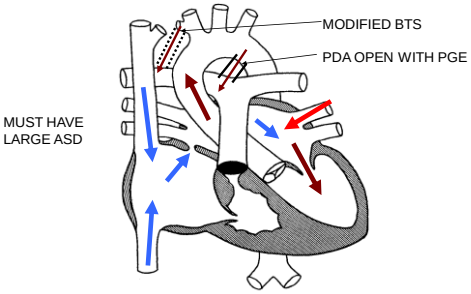
Lamia B, *Minerva Anestesiol* 2006
*** CMH survival to discharge vs. average CO2 gradient on ECMO: n = 454, p < 0.05**

- 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

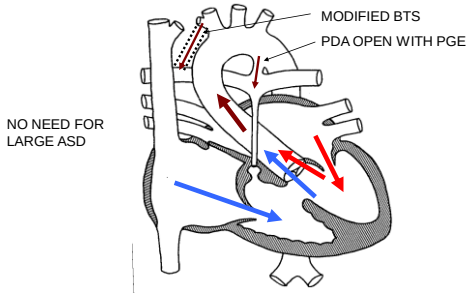
Tricuspid Atresia



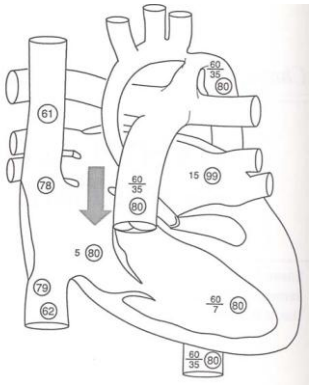
Pulmonary Atresia and Intact Ventricular Septum



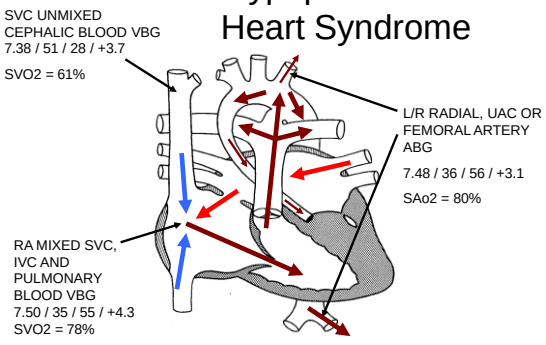
Tetrology Of Fallot With Pulmonary Atresia



Oxygen saturations in a *balanced* circulation



Hypoplastic Left Heart Syndrome



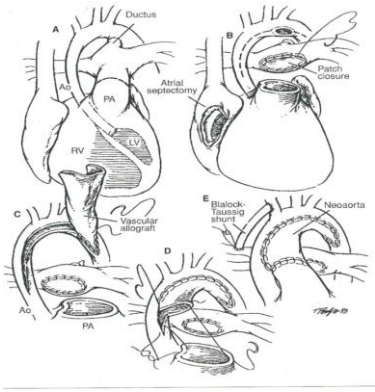
Single Ventricle Reconstruction

- 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

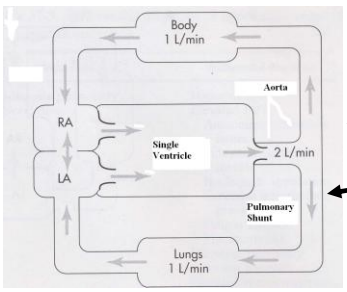
Single Ventricle With High Pressure Pulmonary Shunt

- 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

Norwood Reconstruction for Hypoplastic Left Heart Syndrome



Single Ventricle High Pressure Pulmonary Shunt



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

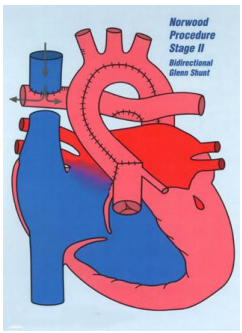
SVC VBG (UNMIXED CEPHALIC BLOOD)
SVO2 ~ 50%

Single Ventricle With Low Pressure SVC Pulmonary Shunt

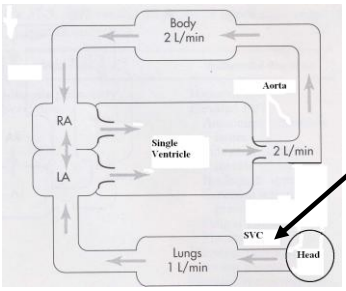
- The single ventricle is unloaded
  – pumps blood only to body
  – lungs receive passive venous flow
- Pulmonary : Systemic blood flow
  – Qp:Qs ratio of 1:2
  – less energy used by the heart, but arterial SAO2 still ~ 80%

LOW PRESSURE SHUNT Qp:Qs = 1:2

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



Single Ventricle
SVC Low Pressure Shunt



ABG L/R RADIAL OR FEMORAL ARTERY SAO2 ~ 80%

ONLY ACCESS TO RA VIA FEMORAL VEIN

LOW PRESSURE SHUNT

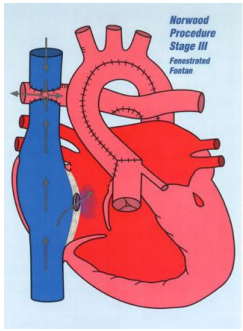
SVC VBG (UNMIXED CEPHALIC BLOOD) SVO2 ~ 50%

Single Ventricle
With Low Pressure SVC/IVC Pulmonary Shunt

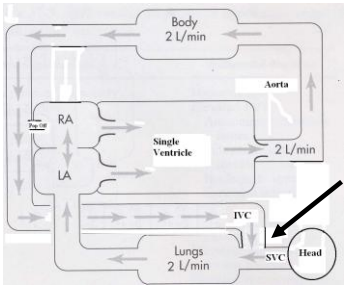
- The single ventricle is unloaded
  - pumps blood only to body
  - lungs receive passive systemic venous flow
- Pulmonary : Systemic blood flow
  - Qp:Qs ratio of 1:1
  - lest energy used by the heart, but arterial SAO2 ~ 95%, if not "popping off"
  - highest risk of pulmonary disease (flu, pneumonia, RSV, etc.) causing hemodynamic collapse
  - cardiac output most susceptible to positive pressure ventilation

LOW PRESSURE SHUNT Qp:Qs = 1:1

- IVC CONNECTED TO THE RPA
- ALL SYSTEMIC VENOUS BLOOD FLOW GOES TO THE LUNGS



Single Ventricle
SVC & IVC Fontan Circulation



ABG L/R RADIAL OR FEMORAL ARTERY SAO2 ~ 95% (except when "popping off")

NO ACCESS TO RA

LOW PRESSURE SHUNT

VBG (MIXED SVC+IVC BLOOD)

SVO2 ~ 65%

Survival

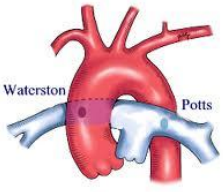
- Mortality after Norwood: 20 - 50%
- Mortality before second stage: 10 -15%
- Mortality after BDG : 2 - 5%
- Mortality after Fontan: 3 -10%
- Overall survival: 50 -70%

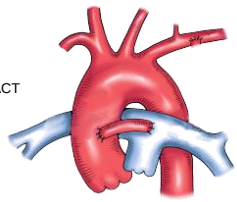
Central Shunts

DRAW SVC VBG IF COMMON ATRIUM

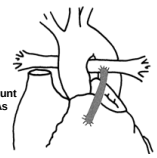
DRAW RA VBG IF ATRIAL SEPTUM INTACT

DRAW R OR L ABG



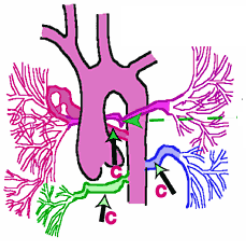


Modified Central Shunt



Sano Shunt
RV to PAs

Major Aorta to Pulmonary Collateral Arteries (MAPCAs)



Collateral Arteries feeding Pulmonary Circulation (C)

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 venoarterial CO2 gradient and SVO2 to assess cardiac index.

The Royal Children's Hospital Melbourne http://www.rch.org.au/cardiology/health-info.cfm?doc_id=3542

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

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

Questions ?