



# RESPIRATORY MEETING, WISCONSIN

HEART ↔ LUNG INTERACTIONS  
4/26/2011



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CRCCS

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## INTRODUCTION

Dr. Stephen Hales in the 17<sup>th</sup> century measured the BP of a horse by inserting a glass tube into the carotid artery. He demonstrated that the heart pumped and BP changed with respirations. Yes, the horse died.



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## Cardiopulmonary Interactions

- Geography and Function
- Autonomic Tone
- Venous Return
- Pulmonary Vascular Resistance
- Ventricular Function
- Clinical Implications

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**GEOGRAPHY**

7 year old boy with ARDS requiring 20 cm of PEEP with secondary myocardial dysfunction.



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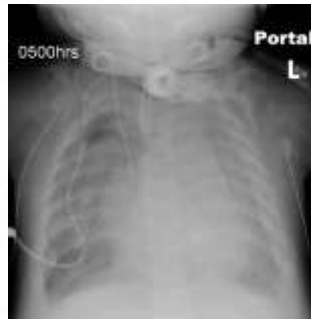
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**GEOGRAPHY**

10 month old with critical mitral valve stenosis with secondary pulmonary edema pleural effusions.



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**GEOGRAPHY**

22 month old with status asthmaticus emergently intubated for acute respiratory failure complicated with bilateral tension pneumothoraces.



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**FUNCTION**

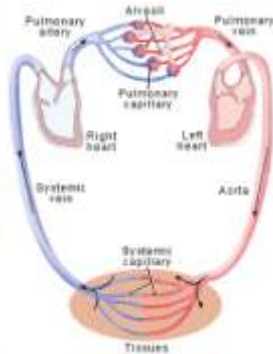
**Oxygen delivery**

**Cardiac performance**

- Heart rate
- Preload
- Afterload
- Contractility

**Pulmonary performance**

- Respiratory rate
- System compliance
- Airway resistance
- Gas exchange



[www.lib.mcg.edu/~sections/echb/s4cht\\_16.htm](http://www.lib.mcg.edu/~sections/echb/s4cht_16.htm)

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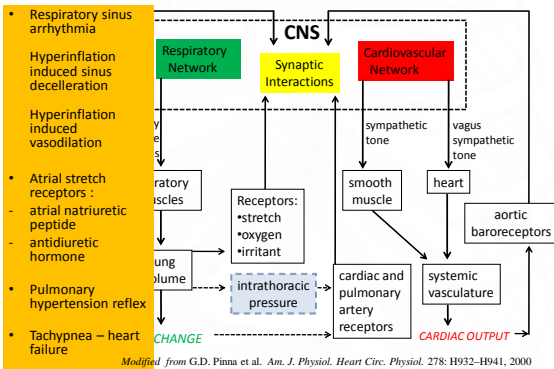
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**FUNCTION**

**AUTONOMIC RESPONSES**



Modified from G.D. Pinna et al. *Am. J. Physiol. Heart Circ. Physiol.* 278: H932-H941, 2000

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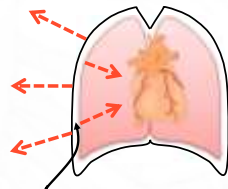
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**FUNCTION**

- chest wall
- thoracic cavity
- intra-thoracic pressure (ITP)
  - lung elastic recoil pressure
  - rib cage pulls outward
  - balance = subatmospheric pressure
  - functional residual capacity (FRC):

*Equilibrium volume when the elastic recoil of the lung is balanced by the tendency of the chest wall to spring out.*



ITP = - 5 cm H2O (3.7 mm Hg)

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**PULMONARY VASCULAR RESISTANCE**



$$\text{cardiac output (Q)} = \frac{\overline{\text{PAP}} - \text{PLa}}{\text{PVR}}$$

$$\text{PVR} = \frac{\overline{\text{PAP}} - \text{PLa}}{\text{Q}}$$

- **RV** compliant low pressure pump
- **PVR** approximates RV afterload
- **PVR** has many determinants:
  - ✓ developmental moment
  - ✓ lung volume
  - ✓ gas exchange
  - ✓ acid – base balance
  - ✓ medications (including NO)

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**IN UTERO**

- Fetal fluid compresses capillaries.
- PVR is almost immeasurably high.
- 90% of the cardiac output is directed away from the lungs.

**DEVELOPMENTAL FAILURE**

- Pulmonary hypoplasia
- Alveolar capillary dysplasia

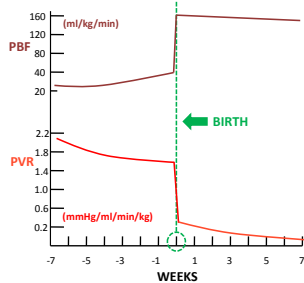
**BIRTH**

- Expulsion of fetal fluid.
- First breath, rhythmic respirations, ↑ oxygen tension : ↓ PVR.
- Ductus closes; foramen closes; systemic vascular resistance ↑.

**TRANSITION FAILURE**

- Persistent pulmonary hypertension of the newborn.

**PULMONARY VASCULAR RESISTANCE: Developmental Moment**




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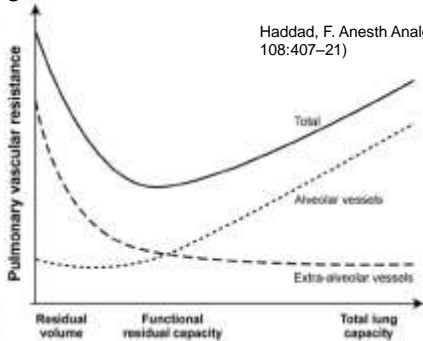
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**PULMONARY VASCULAR RESISTANCE: Lung Volume**



Haddad, F. Anesth Analg 2009; 108:407–21)

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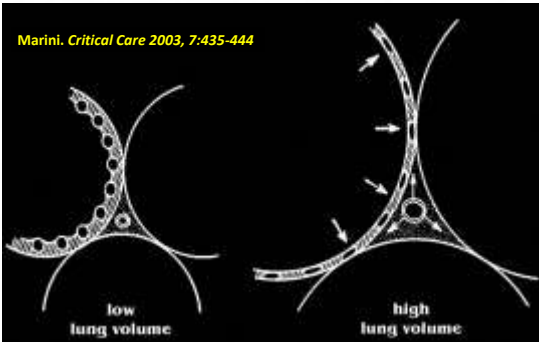
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**PULMONARY VASCULAR RESISTANCE: Lung Volume**




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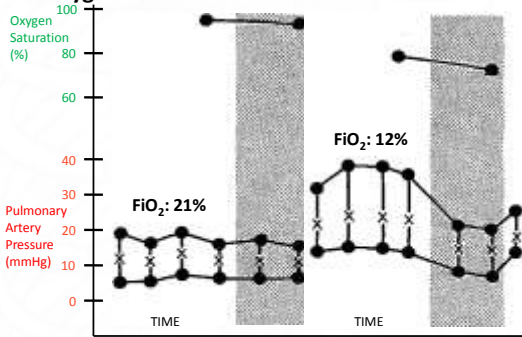
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**PULMONARY VASCULAR RESISTANCE:**

**Oxygen**



Cournand: Nobel Lecture 1956

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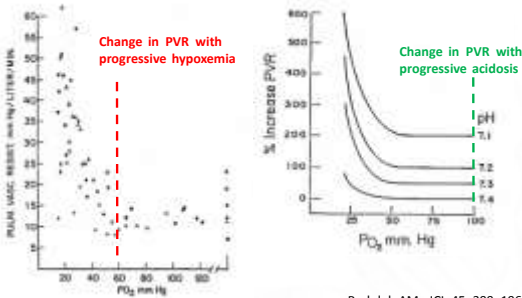
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**PULMONARY VASCULAR RESISTANCE:**

**Oxygen and pH**



Rudolph AM. *JCI*: 45; 399, 1966

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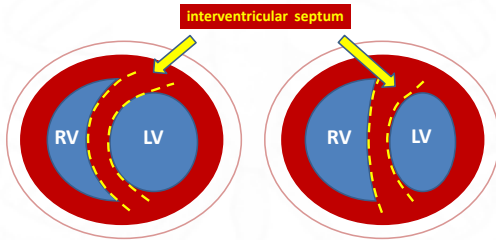
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**LV FUNCTION: Ventricular Interdependence**



↑PVR (afterload) and/or ↑ venous return (labored breathing) ⇒  
 ↑RVEDV ⇒ shift of interventricular septum ⇒ ↓ LV compliance ⇒  
 ↓ LVEDV ⇒ ↓ **CARDIAC OUTPUT**

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**LV FUNCTION: Left Ventricular Afterload**

- LV afterload is defined as the maximal LV systolic wall tension:
  - LVEDV
  - ITP
  - arterial impedance
- LV ejection pressure parallels LV afterload:

$$LVEjp = LVsys - ITP$$

spontaneous breathing     110 = 70 - (-40)  
 positive pressure vent     45 = 70 - (25)

★ + pressure ventilation ↓ LV afterload ★

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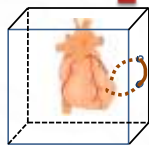
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**LV FUNCTION: Left Ventricular Afterload**

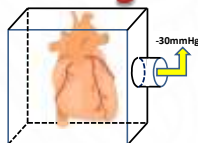


The heart resides in a "box": the thorax. When ITP is negative (and/or excessive) LV contraction is impaired.

2  
 LV free wall is fixed to the side of the box with an "elastic suture". Contraction of the LV is impaired.



3  
 After removing the suture, suction is applied to the box. LV contraction is impaired.




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**CLINICAL IMPLICATIONS: CPR**

Adhesive glove device (AGD) with active compression – decompression CPR in a pig model:

Carotid Blood Flow  
 AGD-CPR: 53.2 27.1%  
 S-CPR: 19.1 12.5%

Active decompression causes ITP to become more negative increasing venous return. Incomplete chest recoil impedes venous return and thus cardiac output.



Udassi JP et al. Circulation, 2009;120:S1451

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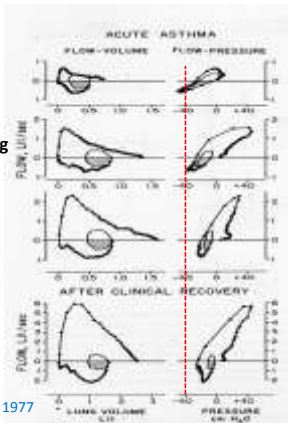
**CLINICAL IMPLICATIONS: ASTHMA**

Acute asthma

- spontaneous:
  - ↑ work of breathing
  - ↑ VR
  - ↑ hypoxia ⇒ ↑ PVR
  - ↑ PaCO<sub>2</sub> ⇒ ↑ PVR
  - ↑ RV dimension
  - ↓ LV dimension
- intubation
  - ↑↑ hyperinflation
  - ↑ PVR
  - ↓ VR
  - ↑ RV dimension
  - ↓ LV dimension

↓ CO

Stalcup & Mellins. NEJM 1977




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**CLINICAL IMPLICATIONS: ASTHMA**

20 year old with acute and severe asthma brought to ED by ambulance receiving bag-mask ventilation. Pt lethargic with poor respiratory effort. Oxygen saturation 80% with 100% FiO<sub>2</sub>. Poor intake for 48hrs. 2-3 vomiting episodes in the last 24hrs. Pt. arrested on intubation. CPR was unsuccessful.



Terminal asthma: massive hyperinflation; alveolar-capillary compression

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**CLINICAL IMPLICATIONS: CDH**

Newborn with acute respiratory failure upon delivery. Poor color, desaturation, and poor perfusion resulted in immediate intubation. Initial ABG: pH 6.95, PaCO2 96; PaO2 42. Pt "stabilized" with HFOV, pH correction, NO, and 100% FIO2 but ultimately required ECMO support and CDH repair. Infant requires chronic ventilation but has a good long term prognosis.



Diaphragmatic hernia: R pneumothorax; bowel in L pleural space.

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**CLINICAL IMPLICATIONS: AUTONOMIC DYSFUNCTION**

4yr old former 24 week pre-mature infant weaning off of chronic mechanical ventilation. At 6mo of age he had "BPD spells". Agitated, he would perform an expiratory breath hold, become cyanotic, bradycardic, and asystolic. A pace-maker was placed to offset these vagal events.




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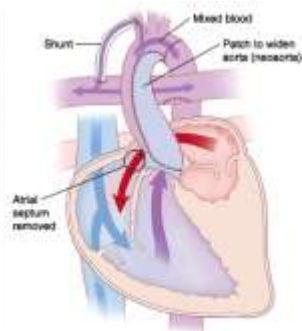
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**CLINICAL IMPLICATIONS: HLHS**

**HLHS**

- "too pink": ↑ PVR
- "too blue": ↓ PVR

↑ O2 sats ⇔ ↑ PBF ⇔  
 ↓ systemic delivery ⇔  
 ↑ acidosis ⇔ ↓ CO



Reddy VM. J Thorac CV Surg 1996;112:437-49

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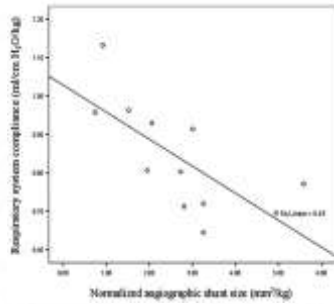
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**CLINICAL IMPLICATIONS:**  
**HLHS**

**Shunt Size and Respiratory Compliance**

Patients with single ventricle physiology. There was an inverse relationship between the normalized size of the shunt (both surgical and naturally occurring) and  $C_{rs}/kg$  ( $r = -0.60$ ,  $r^2 = 0.36$ ,  $p = 0.03$ ,  $n = 14$ ).



Iren L. Matthews, et al. PCCM. 2009;10:1:60-65.

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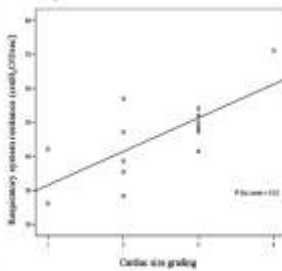
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**CLINICAL IMPLICATIONS:**  
**HLHS**

**Cardiac Size and Respiratory Resistance**

Patients with single ventricle physiology. There was a direct relationship between the cardiac size on CXR and respiratory system resistance.



Iren L. Matthews, et al. PCCM. 2009;10:1:60-65.

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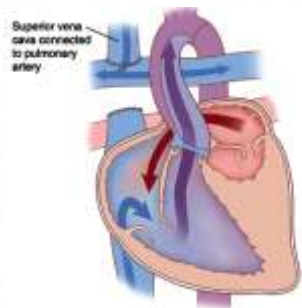
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**CLINICAL IMPLICATIONS:**  
**HLHS**

**Bidirectional Glenn: 15 patients ages 4.7 - 15.5 months (mean 8.5 mo)**

**Glenn shunt**

- post-op cyanosis
- $\uparrow PaCO_2 \Rightarrow \downarrow CVR$
- $\uparrow$  cerebral, caval, and pulmonary blood flow
- $\Rightarrow \uparrow O_2$  sats
- if  $\uparrow PVR$  suspected  $\Rightarrow$  add NO




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**CLINICAL IMPLICATIONS:**  
**HLHS**

**Extra-cardiac Fontan with Gortex lateral tunnel**

**Post-op Fontan**

- pulmonary blood flow (VR) is "passive"
- promote PBF:
  - ↓ PVR –
    - O<sub>2</sub>, ↓ CO<sub>2</sub>, NO, ↑ pH
    - normal lung volume
    - analgesia
  - ↓ ITP –
    - limit PEEP, tidal volume, I-time
    - limit pressure support
    - add spontaneous resps
- avoid atelectasis / VAP




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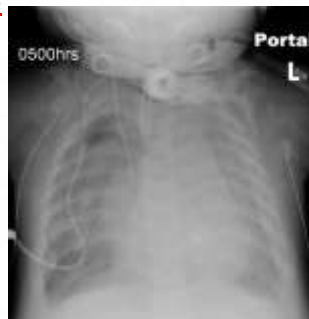
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**CLINICAL IMPLICATIONS:** **CHF**

**Chronic Heart Failure**



- abnml lung mechanics
- ↓ lung diffusion
- ↑ dead space ventilation
- ↓ lung compliance
- ↑ airway resistance




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**SUMMARY**



- The heart and lungs are intimate neighbors.
- Autonomic reflexes are linked within a central controller and coupled by ITP.
- ITP changes impact the function of both organs.
- Spontaneous breathing is driven by -ITP:
  - ↑ venous return
  - ↑ left ventricular afterload
- Mechanical ventilation is driven by +ITP:
  - ↓ venous return
  - ↓ left ventricular afterload
- PVR approximates RV afterload.
- LV ejection pressure parallels LV afterload.
- Airway obstruction *in extremis* results in biventricular failure.
- Heart failure disrupts pulmonary function.
- Heart–lung interactions are common in critical care.

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