Integration of Respiration and Circulation

• Respiratory Failure

  – Either hypoxemia or hypercapnia, signs and symptoms for both are vague at best

  – To really determine blood gases are used, respiratory distress defined as

    • $\text{PaO}_2 < 50 \text{ mmHg}$
    • $\text{PaCO}_2 > 50 \text{ mmHg}$
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- Case Study – Adult Respiratory Distress Syndrome (ARDS)
  - Mr. B, blood pressure low, difficulty breathing
  - Mr. B is on a rehabilitation ward, treated for alcoholism, diverticulitis, schizophrenia, and a seizure disorder
  - Appeared older than his years, 55
  - Ashen gray
  - Sweat on forehead
  - Breathing rapidly
  - Shallow breaths requiring a great deal of effort
  - Using accessory muscles for inspiration, demonstrated supraclavicular and suprasternal depressions
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- Not cyanotic

- Mr. B’s bed sheets were blood stained and dark yellow urine was in the bag by the side of his bed

- When asking about the blood and urine, nurse replied
  - Bleeding into sheets the result of diverticulitis
  - Only produced 350 ml’s over the last three shifts

- Blood pressure could not be measured using auscultation, rather palpation was used, 65

- Pulse 110 b/min, feeble but regular

- Respiration 26 b/min, shallow and labored

- Oral temp 102° F
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- Skin was dry and tented when pinched over the sternum
- Neck veins flat when lying semirecumbent
- Lung sound faint with rhonchi
- First and second heart sounds soft with no gallops or murmurs
- Initial diagnosis, septic shock with respiratory distress
- Ordered oxygen and admittance into ICU
- While in ICU and ECG was performed
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- A = prior to endotracheal tube, sinus tachycardia
- B = during insertion of trache, bradycardia
- C = following injection of atropine, sinus bradycardia, first degree AV block
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- Blood was drawn for
  - Hematologic tests
  - Chemical analysis
  - Bacterial culture
  - Typing and cross matching

- Patient infused with 0.9% saline, 200 ml/hr, with a dopamine drip to raise blood pressure to 90

- Switched from the portable oxygen tank to a face mask connected to a non-breathing oxygen bag (increased inspired oxygen from 40 to 100%, 20 minutes later blood gases were determined
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- ABG #1
  - pH 7.26
  - PaCO₂ 49 mmHg
  - PaO₂ 33 mmHg
  - SaO₂ 54%
  - [HCO₃⁻] 22 mEq/L
  - Base excess -5

- Based on the above results, patient was intubated and placed on a respirator with PEEP (positive end expiratory pressure), a Swan-Ganz catheter was used for precise cardiac monitoring

- Upon inserting the tube Mr. B’s pulse dropped from 130 to 40 b/min, administered atropine, pulse rose to 65 b/min
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- Ventilator was set to the following
  - TV 800 ml
  - Respiratory rate 16 b/min
  - FiO₂ 1

- Swan-Ganz #1
  - Central Venous Pressure, CVP 2 mmHg
  - Mean pulmonary artery pressure, Pa 6.5 mmHg
  - PAWP 4 mmHg
  - CO 2.5 L/min

- Systemic Arterial Pressure (on dopamine drip) 90/60 mmHg

- Patient given
  - Saline increased from 250 to 500 ml/hr
  - Ceftazdime (cephalosporin derivative, anti-gram-negative bacterial agent)
  - Vancomycin (anti-gram-positive bacterial agent)
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- Swan-Ganz #2 - 1 hour later
  - PAWP 10 mmHg
  - CO 4 L/min

- Swan-Ganz #3 - 1 hour later
  - PAWP 12 mmHg
  - CO 5 L/min

- Chest x-ray shows fluffy peripheral infiltrates throughout both lung fields, giving a hazy almost ground glass appearance

- Mr. B went into seizure, Dr. ask for latest blood gases

- ABG #2
  - pH 7.53
  - PaCO₂ 25 mmHg
  - PaO₂ 40 mmHg
  - SaO₂ 75%
  - [HCO₃⁻] 20 mEq/L
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- Dr. ordered the following
  - Decrease ventilation rate from 16 to 10 b/min
  - Set ventilator to 10 cmH₂O PEEP
  - Repeat blood gases in 30 minutes, including a mixed venous PO₂ from a blood sample collected with the Swan-Ganz catheter

- Seizure stopped

- ABG #3
  - pH 7.3
  - PaCO₂ 38 mmHg
  - PaO₂ 150 mmHg
  - SaO₂ 99%
  - [HCO₃⁻] 19 mEq/L
  - Base excess -5
  - PᵥO₂ 28 mmHg
  - SᵥO₂ 55%
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- Swan-Ganz #4
  - PAWP 18 mmHg
  - CO 4 L/min

- In response to the previous findings, Dr. ordered
  - Decrease PEEP from 10 to 2.5 cmH₂O
  - Decrease FiO₂ from 1 to .6
  - Infuse with 2 unit of whole blood
  - Repeat ABG’s in 30 minutes

- ABG #4
  - pH 7.4
  - PaCO₂ 40 mmHg
  - PaO₂ 60 mmHg
  - SaO₂ 89%
  - Base excess 0
  - PVO₂ 35
  - SVO₂ 63%
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- Swan-Ganz #5
  - PAWP 15 mmHg
  - CO 5 L/min

- Mr. B is now alert, can respond to questions by nodding his head

- ECG and blood enzymes show no indication of an MI

- Blood chemistry showed
  - Glucose 178 mg/dL (65 – 110)
  - BUN 27 mg/dL (10 -20)
  - Creatinine 1.2 mg/dL (0.7 – 1.4)
  - Na 158 mEq/L (135 – 145)
  - K 4.7 mEq/L (3.5 – 5.0)
  - Cl 115 mEq/L (96 – 106)
  - Ca 7 mg/dl (8.5 – 11)
  - Mg 2.5 mg/dL (1.5 – 2.5)
  - Total protein 4.5 gm/dL (6.0 – 8.6)
  - Albumin 2 gm/dL (3.2 – 6.0)
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- Hematology report showed
  
  - WBC 29,400/mm
  - Hb 8.3 g/dL
  - Hct 26%
  - Prothrombin time 22 sec (normal 11)
  - Partial thromboplastin time (PTT) 90 sec (normal 30)
  - Fibrinogen 300 mg/dL (normal)
  - Hb rose to 11 g/dL after transfusion

- Blood culture showed
  
  • *Pseudomonas aeruginosa* – sensitive to cephalosporins (ceftazidime) therefore it was continued and Vancomycin was discontinued
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• Case Discussion

  – What caused Mr. B’s low blood pressure?
    • Endotoxins from septicemia caused
      – Fever
      – Chills
      – Vasodilation
    • Decreased fluid volume, dehydration (tenting skin, small vol. concentrated urine)
      • Diminished myocardial contractility (acidemia)
  – Why was 0.9% NaCl infused?
    • To increase volume prior to whole blood becoming available
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- Why was dopamine infused?
  - Remember, acts as a vasodilator at low doses and a vasoconstrictor at high doses
  - In this case high doses were being used to increase SVR to raise BP (>90)

- Why was PEEP not used initially?
  - Problem with PEEP is that it keeps a positive pressure in the alveoli, making intrathoracic pressure positive, pressing on large blood vessels returning to the heart and diminishing venous return – reduced CO was already a problem
  - Why when PEEP at 10 cmH₂O did the CO drop to 4 L/min, while the PAWP increased to 18
    - Compression on Lft atria by increased intrathoracic pressure
  - PEEP was adjusted to 2.5 cmH₂O and 2 units of whole blood were given
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- Why was atropine administered?
  
  - Mr. B was experiencing bradycardia because of excessive stimulation of the SA node by acetylcholine (causes hyperpolarization) therefore the AV was acting as the pace maker

  - Atropine inhibits the action of acetylcholine and restored the normal pace making of the SA node with the return of P waves
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- Why did Mr. B show suprasternal and supraclavicular retractions on inspiration
  
  • Greatly reduced intrathoracic pressure during forced inspirations
  
  • Why was he forcing inspirations?
    
    - Low lung compliance for two reasons:
      
      » Deficiency of surfactant because of a decreased perfusion of upper lobes of the lung and their inability to produce surfactant
      
      » Pulmonary edema the result of inflammation of capillary endothelium because of hypoxia – inflammation products causing leaking of proteins from capillaries, reducing capillary oncontic pressure – this was even more of a problem because of Mr. B’s drinking problem (Why? Hint: liver)
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- Why was Mr. B tachpneic?
  
  • Stimulation of peripheral chemoreceptors
  
  • Because compliance was already low, stretch receptors were stimulated prematurely causing the lungs to deflate sooner, shallow breaths

- Why was Mr. B using his accessory muscles of inspiration?

- Why was Mr. B not cyanotic?
  
  • Being anemic, there was enough oxygen to saturate the hemoglobin that he did have enough so that it was not cyanotic
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- Why was the PaCO₂ value elevated in ABG’s #1?
  
  - Although physiological mechanisms were in place to try to get Mr. B to breathe more effectively the work of breathing was too great and he was experiencing exhaustion – his work of breathing was increased for two reasons
    
    - An increase in physiologic dead space the result of poor lung perfusion, especially to the upper lobes
    
    - The decrease in lung compliance

- What was the significance of the low PaO₂ value in ABG’s #1?
  
  - A PaO₂ of 33 is very low requiring immediate attention
  
  - There were two major reasons for Mr. B’s hypoxia, what were they?
    
    - Poor perfusion increasing physiologic dead space
    
    - Shunting of blood through poorly ventilated areas of the lung
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- Why was there a base deficit in Mr. B when he was being ventilated with high PEEP?

  - Increased lactic acid production because of hypoxia
  
  - Decreased capacity for Mr. B’s liver to metabolize the lactic acid